A CROSS SECTIONAL STUDY TO EVALUATE THE EFFICACY AND FEASIBILITY OF SERUM LACTATE, TOTAL LEUCOCYTE COUNT AND NEUTROPHIL TO LYMPHOCYTE RATIO AS EARLY PREDICTORS OF PUERPERAL SEPSIS

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MASTER OF SURGERY

LIST OF ABBREVATIONS

PPH	POST PARTUM HEMORRHAGE
CRP	C -REACTIVE PROTEIN
WBC	WHITE BLOOD CELL COUNT
NLR	NEUTROPHIL TO LYMPHOCYTE RATIO
CDC	CENTER FOR DISEASE CONTROL
LARC	LONG ACTING REVERSIBLE CONTRACEPTIOM
IUD	INTRA UETRINE DEVICE
MRSA	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS
HIV	HUMAN IMMUNO DEFICIENCY VIRUS
AIDS	ACQUIRED IMMUNODEFICICNECY SYNDROME
SIRS	SYSTEMIC INFLAMMATORY RESPONSE SYNDROME
SOFA	SEQUENTIAL ORGAN FAILURE ASSESMENT
WHO	WORLD HEALTH ORGANISATION
SBP	SYSTOLIC BLOOD PRESSURE
DBP	DIASTOLIC BLOOD PRESSURE
MAP	MEAN ARTERIAL PRESSURE

RCOG	ROYAL COLLEGE OF OBSTETRICS AND GYNECOLOGY
PAMP	PATHOGEN ASSOCIATED MOLECULAR PATTERN
TLR	TOLL LIKE RECEPTORS
CLR	C TYPE LEPTIN RECEPTORS
DAMPS	DAMAGE ASSCOCIATED MOLECULAR PATTERNS
ICAM	INTERCELLULAR ADHESION MOLECULE
VCAM	VASCULAR CELL ADHESION MOLECULE
PRR	PATHOGEN RECOGNITION RECEPTORS
PMN	POLYMORPHO NUCLEAR LEUCKOCYTE RECEPTORS
LPS	LIPOPOLYSACCHARIDE
IL	INTERLEUKIN
ARDS	ACUTE RESPIRATORY DISTRESS SYNDROME
AKI	ACUTE KIDNEY INJURY
ICU	INTENSIVE CARE UNIT
ROS	REACTIVE OXYGEN SPECIES
PROM	PREMATURE RUPTURE OF MEMBRANES
PIH	PREGNANCY INDUCED HYPERTENSION
GDM	GESTATIONAL DIABETES MELLITUS
PPV	POSITIVE PREDICTIVE VALUE
NPV	NEGATIVE PREDICTIVE VALUE

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INTRODUCTION

Sepsis is one of the major cause of maternal, perinatal, morbidity and mortality. Early detection of sepsis can improve maternal and perinatal outcomes (1).

Sepsis is defined as a generalized inflammatory response in the host, with systemic manifestations, caused by one or more infectious agents. Its management is particularly challenging duringpregnancy and the postpartum period. Therefore, the development of guidelines for the diagnosis which can predict early clinical deterioration and treatment of maternal sepsis (2).

Puerperal sepsis is one among the major causes of maternal mortality worldwide and the second most common cause of maternal death after PPH in Asia and Africa, accounting for as much as 15% of death. (3). At national level, one-sixth of maternal deaths is reported to be due to puerperal sepsis (3). Globally, the incidence of the maternal mortality from sepsis is increasing, which is 28.46 per 100,000 women aged 15 to 49 years. Surviving Sepsis Campaign, was developed for the early identification and treatment of sepsis (4).

Lactic acid is the direct biproduct of anaerobic metabolism. Circulatory alterations in sepsis and septic shock cause an increase in lactic acid generation and a decrease in lactic acid clearance, which results in the development of lactic acidosis. A patient with sepsis who has a serum lactic acid content of 4 mmol/L is indicative of severe sepsis in the general population. Increased morbidity and death are linked to elevated lactic acid concentrations[7].

The Surviving Sepsis Campaign recommends measuring lactic acid concentration within the first three hours of presentation with sepsis symptoms and then reevaluating after six hours if initial lactate levels were elevated (>4 mmol/L) to assess treatment response. It is

also known that pregnant women typically have lower serum bicarbonate levels than non-pregnant patients, which lowers their body's ability to act as a natural buffer. The pregnant patient is more vulnerable to metabolic acidosis as a result of this decrease. However, no research has examined lactic acid levels or changes in healthy pregnant women. (4).

Inflammatory process in labor could potentially influence concentrations of inflammatory markers like serum lactate, CRP etc. During labor, with bearing down effectsduring the second stage of labour there will be excess exertion of body muscles, especially uterine muscles and abdominal muscles due to contractions leading to decreased blood supply resulting in tissue hypoxia. This tissue hypoxia leads to the conversion of aerobic glycolysis to anaerobic glycolysis leading to increased lactate production which an endproduct of anaerobic glycolysis. This results in increased serum lactate levels (1,7).

"One of the hematologic changes is increase in number of the white blood cells mostly neutrophils. The increase in the number of the neutrophils is due to decrease the activity oftheir apoptosis mechanism during pregnancy(6). During labour, there is further delayed in the neutrophil apoptosis which lead to further increase of the white blood cell count after normal vaginal delivery Because the white blood cell and neutrophil counts are physiologically high during early puerperium, therefore the white blood cell count is not specific for detection of postpartum infection (10)". For parturient, an increased WBC count may also be associated with antenatal and puerperal infectious morbidities like chorioamnionitis and endometritis. Other factors that alter the WBC count include race, smoking and body mass index. WBC count range usually varies throughout pregnancy(11).

Although various clinical biomarkers are available, only a few have been used recently for the detection of sepsis. There is no proper data about the relationship between NLRlevels and clinical prognosis in patients with sepsis until now. One of the study showed a reversed NLR evolution according to the timing of death, but some studies suggested that NLR was not

associated with mortality in patients with sepsis[8]. Consequently, the clinical usefulness of NLR in patients with sepsis is still a matter of ongoing controversy and this question deserves further investigation. In this observational study, we sought to evaluate the potential association of NLR in women with vaginaldelivery [12].

Therefore, the objective of this study is to evaluate serum lactate levels, total leucocyte count and neutrophil to lymphocyte ratio as an early predictor of puerperal sepsis after vaginal delivery[13]. This analysis hypothesized that elevated lactic acid concentration would be associated with the prediction of sepsis in an obstetric population. Moreover, most of the studies available so far have been conducted in patients who are in established sepsis. This study is conducted to know the efficacy of these tests for prediction of puerperal sepsis even before the onset of symptoms[9].

PUERPERIUM:

It is the period following delivery during which pregnancy induced anatomical and physiological changes return to the pre pregnant state.

Its duration lasts for 4 to 6 weeks[14].

Physiological Changes that occur in the puerperium period are as follows:

BIRTH CANAL:

Soon after delivery, the birth canal's tissues start to regenerate to their pre-pregnancy form. The size of the vagina and its outflow gradually decreases, although they seldom restore their nulliparous proportions. By the third week, rugae start to return, though they are less noticeable than previously.The hymen is symbolised by a number of tiny tissue tags that develop the myrtiform caruncles when they scar[14].

After 4 to 6 weeks, the vaginal epithelium starts to grow and reflects the hypoestrogenic state. This period of time typically corresponds with the start of ovarian oestrogen production. Vaginal outlet relaxation can result from perineal lacerations or stretching during labour. It's possible that

some pelvic floor damage is unavoidable, and having a baby increases the risk of pelvic organ prolapse[14].

UTERUS:

Significant pelvic artery hypertrophy and remodelling result in the dramatically increased uterine blood flow required to sustain pregnancy. Following birth, their size gradually decreases to resemble that of the state before being pregnant. Larger blood arteries within the puerperal uterus are destroyed by hyaline alterations. Smaller ones gradually replace them as they are gradually reabsorbed. However, minute remnants of the larger vessels may last for years.

The dilated cervix's margin, which corresponds to the external os, may become lacerated during labour and vaginal delivery. For a few days right after labour, the cervical hole readily accepts two fingers as it slowly closes(22).

This opening had shrunk, the cervix had thickened, and the endocervical canal had reformed by the end of the first week. The fundus of the constricted uterus is located just below the umbilicus after delivery. Myometrium dominates, with serosa covering it and decidua lining the inside.

The lower uterine segment, which has significantly less strength than the uterine corpus, contracts and retracts less forcefully.

Both walls of the uterus,which are closely apposed, are each 4 to 5 cm thick just after delivery. The uterus weighs about 1000 g at this point[14].

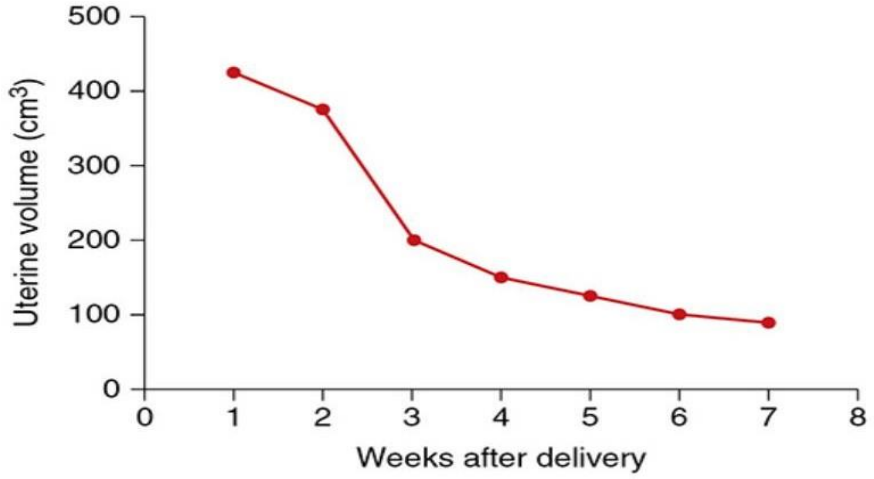
SONOGRAPHIC FINDINGS

Uterine involution and decreased uterine volume is best measured by sonography. The size of the uterus is thought to decrease by more than half in the first two weeks, from 450 to 200 g.

By eight weeks after delivery, the uterus and endometrium sonographically revert to their pre-pregnancy sizes. The multiparous uterus takes longer to fully involute than the nulliparous organ.

The uterine artery's vascular resistance is continuously rising throughout the first seven weeks after delivery, according to doppler ultrasound.

There is no difference in uterine artery flow impedance between women having vaginal birth vs caesarean birth[14].



Graph 1. Pattern of uterine involution through 7 weeks postpartum as estimated by sonography[14].

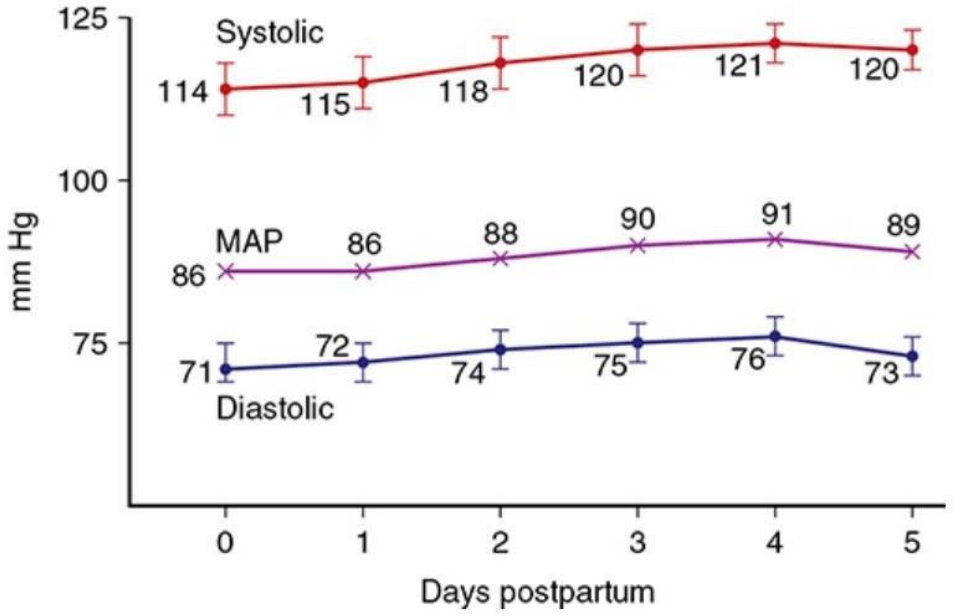
PREGNANCY INDUCED HYPERVOLEMIA:

When the postpartum haemorrhage that results from normal pregnancy hypervolemia is experienced, the lady almost soon returns to her pre-pregnancy blood volume.

Blood volume often almost completely returns to pre-pregnancy levels one week following delivery, assuming less blood was lost during delivery[14,22].

Cardiac output typically remains increased for 24 to 48 hours following delivery and then begins to fall by 10 days to non-pregnancy levels. In accordance, systemic vascular resistance stays in the lower range typical of pregnancy for two days after delivery before gradually rising to normal nonpregnant levels.

Graph 2: Blood pressure distribution in puerperium



After the delivery, the blood pressure begins to fall to reach non pregnant levels[14].

DECIDUAL AND ENDOMETRIAL REGENERATION:

The decidua basalis is not shed because the spongy layer is involved in the separation of the placenta and membranes. The in-situ decidua is infiltrated with blood, especially at the placental site, and its thickness varies noticeably. It also has an uneven, jagged border. After birth, the leftover decidua differentiates into two layers within two or three days. The top layer degenerates and sloughs off in the lochia[27].

The myometrium's surrounding basal layer is still there and serves as the origin of new endometrium. By the time of delivery, endovascular trophoblasts are reduced, and decidual vessels are almost back to normal. The spiral arteries and these vessels both experience involution. The placental location is the only region where endometrial regeneration is slow[14,27].

PLACENTAL SITE INVOLUTION

It can take up to 6 weeks for the placental location to fully extrude.

The placental location is around the size of a palm right after delivery.

It typically has a lot of thrombosed vessels first hours of delivery, which eventually organise. It has a diameter of 3–4 cm at the end of the second week. Exfoliating process of the placental site involution is largely brought on by the implantation site being undermined by fresh endometrial growth[(14)].

AFTER PAINS:

Following delivery, the uterus in primiparas usually continues to be toned down. However, in multiparas, it frequently contracts strongly at intervals and results in afterpains that are comparable to but less painful than labour contractions. These become more noticeable when parity rises and get worse when the baby suckes, probably because oxytocin is released. By the third day, afteraches typically grow milder and less intense[(26)].

LOCHIA: After delivery a variable quantity of vaginal discharge occurs which is the result of sloughing of decidual tissue

It is called as lochia which is composed of RBC's, shredded decidua ,epithelial cells and bacteria

- From day 1-4 after delivery, this discharge is called as Lochia Rubra. It contains blood, fetal membranes, lanugo and meconium.

From 5^{th}-9^{th} day this d

- ischarge is called as Lochia Serosa. It is composed of leukocytes, cervical mucous discharge, and microorganisms.
- After the 10^{th}day, it is called as Lochia Alba.It is made up of plenty of decidual cells, WBC's, epithelial cells and microorganisms.

The average duration of lochia ranges from 24 to 36 days.

SUBINVOLUTION:

Sometimes incompletely rebuilt spiral arteries, residual placental pieces, or infection impede uterine involution.

Such subinvolution is characterised by irregular or severe uterine bleeding as well as various intervals of protracted lochia.

The uterus is bigger and softer than expected during bimanual examination.

Inadequate spiral artery remodelling into uteroplacental arteries can cause subinvolution[22].

These noninvoluted arteries lack an endothelial lining and are filled with thrombi.

Additionally found in the arterial walls are perivascular trophoblasts, which points to an abnormal connection between uterine and trophoblast cells[14, 22].

Treatment

At Parkland Hospital, patients are given methylergonovine 0.2 mg orally every three to four hours for a period of 24 to 48 hours as part of conservative therapy.

An injectable injection of methylergonovine combined with a synthetic oxytocin infusion may be used to treat initially brisk bleeding (20 units in 1 L crystalloid).Antimicrobial therapy typically results in a favourable response in cases of concomitant comorbid infection. According to a study by Wager and colleagues from 1980, Chlamydia trachomatis is the root cause of one-third of late instances of postpartum metritis.Regardless of the bacterial cause, empirical therapy with azithromycin or doxycycline typically results in resolution.

Other recommended oral options include taken twice daily for 7 to 10 days include azithromycin, 500 mg OD; amoxicillin-clavulanate, 875 mg; or doxycycline, 100 mg twice daily[14].

LATE POST PARTUM HEMORRHAGE:

Secondary postpartum haemorrhage is defined by the American College of Obstetricians and Gynecologists (2017d) as bleeding that occurs between 24 hours and 12 weeks after birth.

In about 1% of women, uterine bleeding that is clinically concerning develops within 1 to 2 weeks.This bleeding typically results from improper placental site involution. On rare occasions, a uterine artery pseudoaneurysm or the retention of a placental bits causes it[22].

Rarely, retained products may undergo neovascularization, fibrin deposition, and necrosis to create a placental polypoid tumour.Only 6% of these cases result in severe bleeding from blood vessel rupture.Other hereditary coagulopathies or von Willebrand disease may also contribute to delayed postpartum bleeding[22].Few women with delayed bleeding were found to have retained placental pieces, according to some investigations. Therefore, curettage is not something they do frequently.

Another issue is that by partially avulsing the implantation site during curettage, bleeding may intensify. Thus, in a stable patient, oxytocin, methylergonovine, or a prostaglandin analogue is administered if sonographic testing reveals an empty cavity[22]. If uterine infection is thought to be present, antibiotics are added.Gentle suction curettage is an option if sonography reveals big clots in the uterine cavity. Otherwise, curettage is only done if significant bleeding continues or returns after receiving medical attention[14].

URINARY TRACT CHANGES:

During the puerperium, normal pregnancy-induced glomerular hyperfiltration continues, but it quickly recovers to its pre-pregnancy level.A brief increase in glomerular podocyte excretion is brought on by parturition.. By 2 to 8 weeks after delivery, the renal pelvis and dilated ureters are back to normal. This dilated collecting system, along with leftover urine and bacteriuria in a traumatised bladder, continue to raise concerns for symptomatic urinary tract infection in the puerperium.Bladder trauma is quite common during a typical vaginal birth because it is most closely related to the length of labour[14].

The bladder is more capable and less sensitive to intravenous pressure after giving birth. As a result, excessive residual urine, inadequate emptying, and overdistention are common.Epidural or narcotic analgesia is more likely to cause acute urine retention.5% of women may experience stress incontinence during the puerperium[22].

HEMATOLOGICAL CHANGES:

During and after labour, significant leukocytosis and thrombocytosis might happen. Rarely does the white blood cell count exceed 25,000/L, and granulocytes are primarily responsible for the increase. A significant volume of blood has been lost if they drop significantly below prelabor levels.The laboratory results that measure coagulation have changed by the end of pregnancy[(14)].

POST PARTUM DIURESIS:

Postpartum diuresis is a physiological reversal of this process. Physiological extracellular sodium and water retention are connected with normal pregnancy..

According to a study by Chesley and colleagues (1959), the sodium space decreased by around 2 litres in the first postpartum week. This is consistent with the disappearance of gestation hypervolemia. Pathological antepartum fluid retention and later postpartum fluid diuresis can both be excessive in preeclampsia[(22)].

In addition to the delivery weight loss of 5 to 6 kg and regular blood loss, postpartum diuresis causes a reasonably quick weight loss of 2 to 3 kg.

By the end of the second postpartum week, weight loss from the pregnancy itself probably reaches its peak. Any weight that is still present after pregnancy relative to prenatal values likely signifies fat reserves that will endure[(22)].

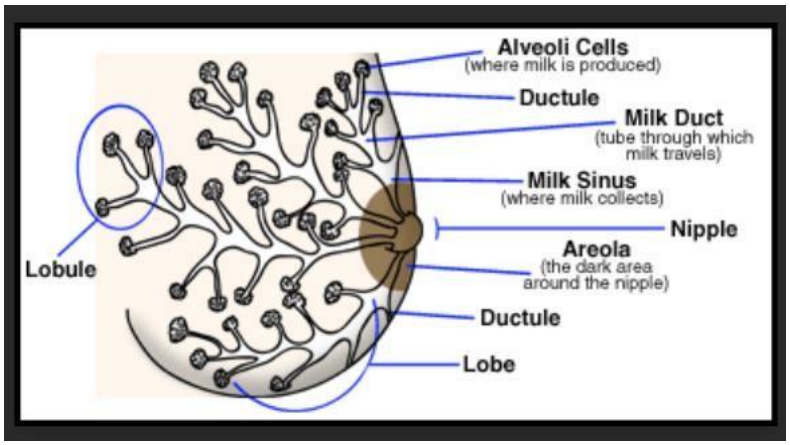
LACTATION AND BREAST FEEDING

Breast Anatomy and Secretory Products:

The number of lobes in each adult mammary gland or breast ranges from 15 to 25. They are organised radially and spaced apart by various thicknesses of fat. Each lobe is made up of a number of lobules, which are itself made up of a large number of alveoli.

Each alveolus has a tiny duct that connects to others to create a bigger duct for each lobe. These lactiferous ducts have individual openings on the nipple, where it is possible to see them as tiny yet distinct orifices[14].

Fig 1: Anatomy of breast



The alveolar secretory epithelium secretes the different components of milk. Colostrum, a dark yellow liquid, is secreted by the breasts after delivery. Usually by the second postpartum day, it can be expressed from the nipples. Colostrum includes more minerals and amino acids and is richer in immune components than mature milk. However, it contains less sugar and fat and more protein, much of which is globulin. Secretion lasts for 5–14 days before gradually transitioning to mature milk after 4–6 weeks[14].

Immunoglobulin A (IgA), which is found in colostrum, protects the baby from enteric infections. In addition to these, complement, macrophages, lymphocytes, lactoferrin, lactoperoxidase, and

lysozymes are also present in colostrum and milk.The amount and quality of the milk produced by a breastfeeding mother, which can reach 600 mL per day, is mostly unaffected by the gestational weight increase of the mother[14,22)].

A complex and dynamic biological fluid, mature milk contains many cellular components, including fat, proteins, carbohydrates, bioactive substances, minerals, vitamins, and hormones. Even during a single feeding, the composition and concentrations of human milk can alter depending on the diet of the mother and the newborn's age, health, and nutritional requirements. Milk and plasma are isotonic, and lactose is responsible for 50% of the osmotic pressure. Blood is the source of necessary amino acids, whereas the mammary gland or blood in part are used to generate non-essential amino acids. The majority of milk proteins, such as alpha-lactalbumin, beta-lactoglobulin, and casein, are distinctive. In an apocrine-like mechanism, fatty acids are produced in the alveoli from glucose and secreted. The majority of vitamins are present in varying degrees in human milk[(14)].

Table 1 depicts the composition of human breast milk[(14)].

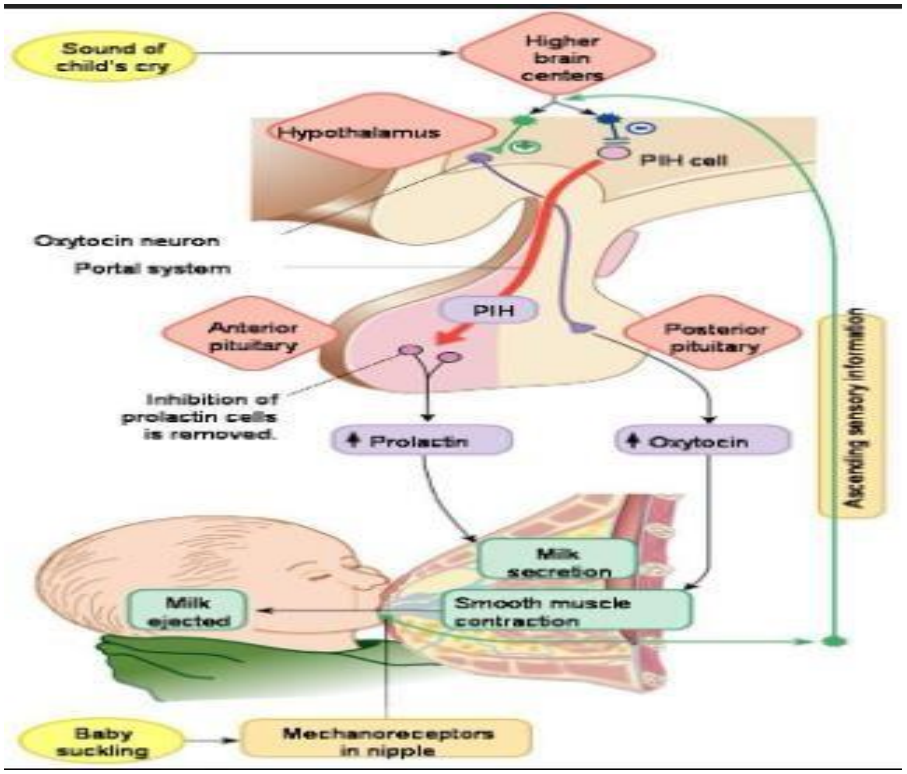
FAT	**g/100ml**
Total	4.2
Fatty acids	Trace
PUFA	0.6
Cholesterol	0.016
PROTEIN	**g/100ml**
Total	1.1
Casein	0.3
Alpha lactalbumin	0.3
Lactoferrin	0.2

CARBOHYDRATE	g/100ml
Lactose	7
Oligosaccharides	0.5

LACTATION ENDOCRINOLOGY

Prolactin, cortisol, and insulin, as well as the hormones progesterone, oestrogen, and placental lactogen, all appear to work together to promote the growth and development of the milk-secreting machinery.The maternal blood levels of progesterone and oestrogen dramatically decrease after delivery. Alpha-lactalbumin synthesis no longer experiences progesterone's inhibitory effects, and lactose synthase is stimulated, increasing milk lactose levels[22].

Fig 2: Endocrinology of lactation



Prolactin can work unimpeded in its stimulation of alpha-lactalbumin synthesis after progesterone removal. Mammary epithelial cells also create serotonin, which contributes to the maintenance of milk production. The recurrent sensation of sucking and milk ejection from the breast governs, in considerable part, the intensity and length of later lactation.

Women with Sheehan syndrome, a severe case of pituitary necrosis, are unable to breastfeed because prolactin is required for breastfeeding[22].Stimulation of breast reduces the release of dopamine from hypothalamus. As a result, there is a brief increase in prolactin secretion. This is even stimulated by infant cry and inhibited by anxiety and stress of the women[22].

LACTATION:

Within an hour of birth is the optimal time to start nursing.Human milk is the best diet for babies because it contains nutrients, immunological components, and antimicrobial compounds that are appropriate for their age.

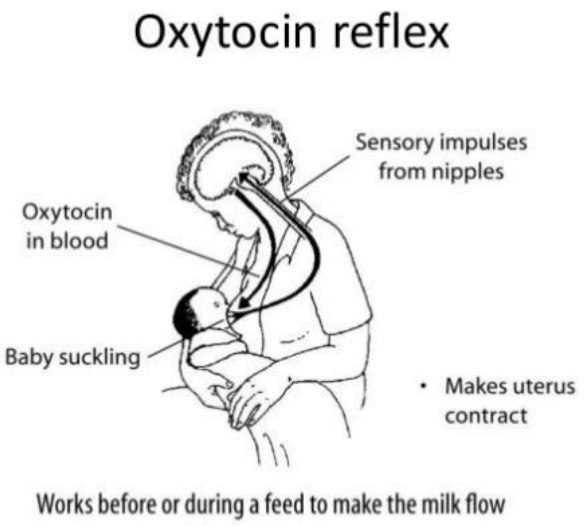


Fig 3: Oxytocin reflex

Biological cues for encouraging cellular development and differentiation are also present in milk. Both the mother and the child benefit over the long run from breastfeeding. As an illustration, women who breastfeed are less likely to get breast and reproductive cancer.

Independent of a wide range of potential confounding circumstances, children who were breastfed have higher adult intellect ratings.Lactation is linked, in the near term, to decreased postpartum weight retention[(14)].

BREAST CARE

Apart from cleaning and paying attention to skin fissures, the nipples don't need much care. Fissured nipples make nursing uncomfortable and may negatively affect milk output. Additionally, these fissures provide as a point of entry for pyogenic bacteria. Before and after nursing, cleaning the areola with water and mild soap is important because dried milk is likely to accumulate and hurt the nipples. Some people advise applying topical lanolin and wearing a nipple shield for 24 hours or more when the nipples are sore or fissured[(14)].The infant shouldn't be allowed to nurse on the side with severe fissuring.Instead, until the lesions are healed, the breast is routinely drained with a pump. Such cracks might be caused by the neonate not latching properly to the breast.To uniformly disperse suckling forces, the nipple and areola should ideally be taken in simultaneously. Additionally, the nipple is placed closer to the soft palate as a result of the hard palate's force against the lactiferous sinuses, which helps with their effective emptying[(14)].

CONTRA INDICATIONS OF BREAST FEEDING

- Drugs and uncontrolled alcohol use
- Infant with galactosemia;
- Human immunodeficiency virus (HIV) infection;
- Active, untreated tuberculosis;

- Certain medications; or are undergoing breast cancer treatment.
- Women with active herpes simplex virus may suckle their infants if there are no breast lesions and if particular care is directed to hand washing before nursing.
- From the CDC (2021a), those with COVID-19 should wear a mask and practice hand-hygiene[14,22].

BREAST ENGORGEMENT

This happens frequently to non-breastfeeding mothers. Breast soreness and milk leakage, which peak 3 to 5 days after delivery, are its hallmark symptoms. Up to 50% of affected women need analgesics for the treatment of breast discomfort, and up to 10% report severe pain lasting up to 14 days. The breasts can be supported with a well-fitting brassiere, breast binder, or sports bra, but there is not enough evidence to firmly support any particular treatment. Pain can be relieved with ice packs and 12- to 24-hour oral analgesics.

It is not advised to restrict lactation using medications or hormones in general. Before the revival of breastfeeding, fever brought on by engorged breasts was typical.

Rarely does a fever last for more than 4 to 16 hours.

If women breastfeed, the frequency, intensity, and fever associated with engorgement are significantly reduced. Fever must not have any other causes, especially ones that are infectious. Breast parenchyma infection known as mastitis is relatively common in nursing mothers[22].

POSTPARTUM HOSPITAL CARE

Blood pressure and pulse are checked every 15 minutes, at the absolute least, during the first two hours after delivery. Temperature readings are taken every four hours for the first eight hours, and then at least every eight hours after that. Vaginal bleeding is closely monitored because the risk of postpartum heamorrhage is highest immediately after delivery[14,22].To make sure it is fully contracted, the uterine fundus is palpated. If the uterus starts to relax, you should massage

it via the abdominal wall until it doesn't. Sometimes, uterotonics are also necessary.Without outward bleeding, the uterus may fill with blood.

Uterine enlargement during fundal palpation in the initial postpartum hours may help detect this early.Within a few hours of giving birth, early ambulation is suggested. Early ambulation reduces the risk of thromboembolism[22]. Following an uncomplicated vaginal delivery, women is allowed soft diet.Dramatic hypoestrogenism may cause headaches in migraine sufferers. Importantly, severe headaches should be distinguished from post dural headache or consequences from hypertension.

Care varies depending on headache severity.

- Analgesics such as ibuprofen or acetaminophen can be given for mild headcahes.
- Oral or systemic narcotics can be used for more severe headaches
- Alternatively, a triptan, such as sumatriptan (Imitrex), can relieve headaches by causing intracranial vasoconstriction, and it is breastfeeding compatible[22].

POST PARTUM PERINEAL CARE

The woman is educated about the perineal hygiene and care that is to be taken following delivery.

A cool pack if necessary to be applied for perineal lacerations and episiotomy to reduce the swelling and discomfort for the 1st 24 hours.Conscious examination and palpation are always warranted in cases of extreme perineal, vaginal, or rectal pain.Extreme discomfort typically signals a problem, such as an infection after the third or fourth day or a hematoma within the first few days.Warm sitz baths that produce moist heat can relieve local pain starting about 24

hours after delivery. Tub bathing is permitted following a straightforward delivery. By third week, the episiotomy incision is typically completely healed and almost symptom-free.

A palpable lump at or past the introitus, voiding issues, or pressure are examples of symptoms. These haemorrhoids frequently thrombose, and second-stage pushing may contribute to this. Warm soaks, topical anaesthetics, and stool softeners are all part of the treatment[22].

POST PARTUM PSYCHOLOGIC ISSUES

The mother may experience discomfort in the initial days following vaginal delivery due to afterpain, episiotomy and lacerations, breast engorgement, and occasionally postdural puncture headache.During the first few days, mild analgesics containing codeine, ibuprofen, or acetaminophen are used as frequently as every four hours, ideally in combinations.

It is important to check for depression in postpartum women. Postpartum blues, a common mood disorder that affects moms a few days after birth, are probably the result of a number of causes.

These include:

- The emotional letdown that follows the anticipation and anxieties felt throughout pregnancy and birth, as well as the early puerperium's discomfort.
- Sleep deprivation that results from fatigue
- Concerns about being able to properly care for newborns, and
- Body image issues.

These symptoms are usually mild and resolve in 2 to 3 days, but sometimes it lasts for up to 10 days.Brain function may also be effected in some women[14].

IMMUNIZATION:

300 g of anti-D immune globulin are administered shortly after delivery to the D-negative mother who is not iso vaccinated and whose newborn is D positive.

Before discharge, vaccinations against varicella and rubella are highly recommended for women who do not already have immunity.

Tetanus/diphtheria (Tdap/Td) and influenza vaccines should be administered to those who have not received them (American College of Obstetricians and Gynecologists, 2017e).

The COVID-19 vaccine is advised by the CDC (2021b), which also applies to nursing mothers[22].

CONTRACEPTION

Contraception During the hospital stay, a concerted effort is made to provide family planning education. The immediate puerperium is an ideal time for consideration of long-acting reversible contraception—LARC.Menstruation returns for women who are not nursing often take 6 to 8 weeks.However, it can occasionally be challenging clinically to put a date on the first menstrual cycle following birth. Few women experience sporadic, light to moderate bleeding that begins soon after birth. Ovulation occurs between weeks 5 and 11, with a mean of 7 weeks.The early puerperium is therefore a time when conception is possible[16].Women who become sexually active during the puerperium and who do not desire to conceive should initiate contraception.

As a result, many advise using LARC during the puerperium. Breastfeeding mothers ovulate substantially less frequently than non-breastfeeding mothers, while there is wide variance. The intensity of nursing and individual biological variance both affect when ovulation occurs. Women who are lactating may experience their first period as soon as the second month or as late as the 18th month following delivery[14,16].Progesterone-only contraceptives, such as progestin pills, depot medroxyprogesterone, progestin implants, or IUDs, have no effect on the quantity or quality of milk produced by breastfeeding women. The puerperium is the time when these can begin.Estrogen-progestin contraceptives probably diminish breast milk production, although nursing women can also use them under the right conditions[14].

POST PARTUM HOME CARE

Coitus:

Depending on comfort and desire, coitus may be resumed after two weeks. According to a study by Wall wiener and colleagues published in 2017, 60% of women resumed sexual activity by one week and 80% by four months.In addition, they reported that one-third of these women experienced sexual dysfunction. Too soon after an episiotomy or perineal laceration, sexual activity may be unpleasant, if not painful. Only 0.4% of women in a study without an episiotomy who had a first- or second-degree tear experienced dyspareunia[14,22].

By 12 weeks, only 40% of women who had an anal sphincter lesion had resumed sexual activity Finally, dyspareunia following a caesarean delivery was also prevalent . The epithelium of vulva and vagina is thin postpartum, and minimal lubrication results from sexual stimulation. This results from the postpartum hypoestrogenic condition, which persists until ovulation restarts. Breastfeeding mothers who are hypoestrogenic for a long time postpartum may experience specific difficulties. For treatment, vulvar tissues can get daily applications of topical oestrogen cream in tiny amounts for a few weeks. Vaginal lubricants may also be used during coitus. Dysuria may result from the same vulvovaginal epithelial weakening[22].

PUERPERAL COMPLICATIONS

PUERPERAL FEVER

Puerperal fever, which is defined as a temperature of 38.0°C (100.4°F) or greater, is brought on by a number of infectious and noninfectious reasons. According to this strict criteria, Filker and Monif (1979) found that only 20% of women who became feverish within the first 24 hours of vaginal delivery ultimately had pelvic infections. Those who had caesarean deliveries had a value of 70%. The majority of postpartum fevers that persist are brought on by genital tract

infections[14]. It is important to remember that fevers that spike in the first 24 hours after giving birth may be linked to a severe pelvic infection brought on by group A streptococcus.

Other sources of puerperal fever include

- Engorgement of breast
- UTI
- Skin incision or abdominal and episiotomy wounds
- perineal tears and lacerations
- respiratory complications that occur post delivery

Fever from breast engorgement affects about 15% of women who do not breastfeed. In the first few postpartum days, "breast fever" rarely rises over 39°C and typically lasts a few days[48].

UTERINE INFECTION

Endometritis, endomyometritis, and endoparametritis are the several names for postpartum uterine infection. Infection affects not just the decidua but also the myometrium and parametrial tissues after vaginal delivery. Infection following a caesarean delivery primarily affects the myometrium that has been surgically incised.

Predisposing Factors: The most important risk factor for the development of uterine infection is the method of delivery. Rehospitalization rates for metritis and wound problems are greater in cesarean-delivery mothers than they are in vaginal births. Women at Parkland Hospital who give birth vaginally had a 1- to 2-percent incidence of metritis. In cases of ruptured membranes, protracted labor, and frequent cervical exams, this rate increases to 5 to 6%.

These numbers are comparable to those from a cohort of more than 115,000 women in whom the total pelvic infection rate was around 5%, as reported by the Maternal Fetal Medicine Units Network[14,47].

Women who undergo instrumental vaginal birth are more likely to develop metritis and perineal infections than those who give birth naturally. Notably, these births are frequently carried out during protracted labour, which is known to increase the risk of metritis. Those getting a single dosage of preventive antibiotics had a decreased rate of maternal illness, according to the study conducted by Knight.The uterus, the urinary tract, and perineal wounds can all get infected.

Prolonged labour, membrane rupture, several cervical exams, and internal foetal monitoring are significant risk factors for infection after surgery. No matter the method of birth, women with lower socioeconomic status typically get pelvic infections more frequently(47).

It is uncommon for anaemia or inadequate nutrition to predispose to infection, save in rare circumstances that are often not observed in affluent nations. An increased risk of postpartum infection has been linked to bacterial colonisation of the lower genital tract by specific microorganisms, such as group B streptococcus, Chlamydia trachomatis, Mycoplasma hominis, Ureaplasma urealyticum, and Gardnerella vaginalis.

Other factors associated with a greater risk include

- General anesthesia,
- Significant hysterotomy extension,
- Age of the women
- Nulliparous women
- Prolonged induction of labor
- Chorioamnionitis,
- Obesity, and
- Meconium stained liquor(14)

Table 2: Bacteria responsible for female genital tract infections are

"Aerobic organisms
Gram positive organisms like enterococci, staphylococcus aureus, staphylococcus epidermidis**:** Group A, B and D streptococci Gram negative organisms like Proteus spp., Klebsiella and E.coli Gram variable organism like Gardnerella vaginalis
Mycoplasma , Chlamydia trachomatis, Neisseria gonorrhoeae
Anaerobic organisms
Pepto streptococcus, Pepto coccus spp **Others agents:** clostridium, Bacteroides, fusobacterium, Mobiluncus spp"[47,50].

Most of these infections are polymicrobial, which enhances bacterial synergy.

Hematomas and tissue that has lost vitality are additional elements that increase pathogenicity. The uterine cavity is typically sterile before to the rupture of the amniotic sac, despite the cervix and vagina frequently harbouring bacteria. The uterus and amniotic fluid get contaminated with anaerobic and aerobic microorganisms as a result of labour, delivery, and related procedures[22.47].

Pathogenesis and Clinical Course:

After a vaginal delivery, the placental implantation site, decidua and surrounding myometrium, or cervicovaginal lacerations are the most common sites for puerperal infection. An infected surgical incision is the pathophysiology of uterine infection after caesarean delivery. During labour, bacteria that inhabit the cervix and vagina might enter the uterus. They infiltrate postpartum uterine tissue that has lost vitality. Infection of the pelvic retroperitoneal fibro areolar connective tissue occurs after parametrial cellulitis. Early intervention can keep the infection from spreading deeper into the pelvis and into the tissue of the parametrium and paravagina.

The most crucial factor in the diagnosis of postpartum metritis is fever. It makes intuitive sense to assume that sepsis and infection severity are inversely correlated with fever severity.

Temperatures typically range from 38 to 39 degrees Celsius, and readings over 39 indicate bacteremia or endotoxemia.Women typically complain of stomach pain, and an abdominal and bimanual examination reveals parametrial tenderness.

Caesarean birth itself increases the leukocyte count, therefore leukocytosis may range from 15,000 to 30,000 cells/L.Despite the possibility of an objectionable odour, many women experience unpleasant-smelling lochia without any signs of infection, and vice versa. There may be a connection between sparse, odourless lochia and some other diseases, particularly those brought on by group A -hemolytic streptococci[(46)].

Treatment:

Treatment with an oral or injectable antibacterial antibiotic may be sufficient if metritis occurs after vaginal delivery.However, intravenous therapy with a broad-spectrum antibiotic regimen is recommended for moderate- to severe-grade infections.

Nearly 90% of women who receive one of the several regimens covered here see improvement within 48 to 72 hours. After this time, fever that doesn't go away necessitates a thorough investigation into the pelvic infection's root causes. These include septic pelvic thrombophlebitis, abdominal incisional or pelvic abscesses, or an area of severe cellulitis known as a parametrial phlegmon[(46)].

Choice of antimicrobials:

Up to 90% of women with infections after vaginal delivery respond to regimens like ampicillin plus gentamicin.In contrast, infections following caesarean delivery are covered by anaerobic coverage[(46)].

Antimicrobials of choice following caesarean delivery are as follows:

Table 3:

"DRUGS	COMMENTS
Gentamicin plus Clindamycin	It is the gold standard regimen. Its efficacy is 90-97%, gentamycin daily once dosing with ampicillin added to regimen with sepsis or suspected enterococcal infection
Clindamycin plus Aztreonam	Used patients with renal insufficiency in place of gentamycin
Extended broad spectrum pencillins	Piperacillin,tazobactum, ampicillin/clavulanate
Cephalosporins	Cefotetan, cefoxitin, cefotaxime, ceftrioxone
Vancomycin	For suspected staphylococcus aureus infections it is added to other regimens
Metronidazole plus Ampicillin plus Gentamicin	Used mainly for anaerobic coverage
Carbapenems	Imipenem, meropenem, ertapenem, cilastatin used in special conditions .

Due to the possibility for nephrotoxicity and ototoxicity with gentamicin, some prescribe a combination of clindamycin and a second-generation cephalosporin in situations with reduced glomerular filtration. Others advise combining clindamycin with aztreonam, a monobactam substance with action resembling that of aminoglycosides.The spectra of β-lactam antimicrobials include activity against many anaerobic pathogens[33].

Apart from allergic reactions, -lactam antimicrobials are naturally safe and toxic-free. To broaden the spectrum of these antibiotics, the -lactamase inhibitors clavulanic acid, sulbactam, and tazobactam have been coupled with ampicillin, amoxicillin, ticarcillin, and piperacillin.

With regard to most anaerobes, metronidazole exhibits superior in vitro action. This medication, when combined with ampicillin and an aminoglycoside, offers protection from the majority of pathogens found in serious pelvic infections.

In some cases of Clostridiodes difficle colitis, it is also utilised as a second line of treatment.

The carbapenem class of antibiotics includes imipenem and related drugs[33].

These provide comprehensive protection against the majority of pathogens linked to metritis.

The renal metabolism of the antibiotic is inhibited when imipenem and cilastatin are combined.

It is sensible from both a medical and financial perspective to reserve imipenem for severe non-obstetrical infections because of its higher price. A glycopeptide antibiotic that is effective against gram-positive bacteria is vancomycin.

When a patient has a type 1 allergic reaction, it is utilised in place of -lactam therapy. It is also administered to treat Clositridium difficle colitis and possible S aureus infection[33].

PERIOPERATIVE PROPHYLAXIS

Numerous studies show that prophylactic antibiotics at the time of cesarean delivery reduce wound and postpartum pelvic infection rates. Both elective and emergency caesarean deliveries are covered by the observed benefit.Antimicrobials may lower pelvic infection rates following surgical vaginal birth, according to early research.However, there is no evidence to support the idea that preemptive antibiotic use lowers infection rates following spontaneous vaginal delivery, episiotomy repair, or manual placenta extraction[33].

The ACOG (2018) concluded that a single antibiotic dose with third- and fourth-degree perineal laceration is reasonable and has evidenced-based support.A 2-g dose of a first-generation

cephalosporin given as a single dose for prophylaxis is appropriate.Its effectiveness is comparable to that of multiple-dose regimens or broad-spectrum medications (American College of Obstetricians and Gynecologists, 2018b).Evidence suggests that a 3-g dose of cefazolin will help obese women achieve the best tissue concentration[12].

The American College of Obstetricians and Gynecologists (2018b) came to the conclusion that the predelivery administration is supported by the evidence. The majority of women who claim to be allergic to penicillin are not at risk of experiencing anaphylaxis. Most of these ladies can safely be administered a cephalosporin if they haven't previously experienced anaphylaxis[(12,33)].

If not, gentamicin, clindamycin, and vancomycin are also administered (American College of Obstetricians and Gynecologists, 2018).Infection risk in the pelvis and on the surgical site is reduced by preoperative abdominal skin preparation.

For reducing surgical-site infections, skin preparation with chlorhexidine alcohol is preferable to iodine-alcohol.Preoperative vaginal washing with a povidone-iodine rinse or use of metronidazole gel may result in additional positive results.The risk of infection may be decreased by letting the placenta separate naturally and exteriorizing the uterus to closure the hysterotomy[(12)].

The infection rate is unaffected by the removal of the gloves following placental delivery, cleaning the intrauterine space, or dilation of the lower segment and cervix.

When single- and two-layer uterine closures were evaluated, no differences in postoperative infection rates were discovered[(12)].

PERITONITIS AND ADNEXAL ABSCESS:

Peritonitis after a caesarean delivery is uncommon.

Metritis usually always comes first, particularly in situations with uterine incisional necrosis and dehiscence.However, it could result from an unintentional intraoperative intestinal damage or a

ruptured adnexal abscess. Peritonitis can also result from perforative appendicitis. In these situations, immediate surgical intervention is typically recommended.Peritonitis following vaginal delivery is uncommon, and many of these cases are caused by virulent strains of group A -hemolytic streptococci or other related pathogens[16,33].

Importantly, due to physiological abdominal wall flexibility brought on by pregnancy, abdominal stiffness may not be noticeable with puerperal peritonitis.Despite the possibility of significant pain, adynamic ileus symptoms are typically the initial signs of peritonitis. After a vaginal birth, considerable intestinal distention may appear, which is unusual.Typically, antibiotic treatment alone is sufficient to treat infections that start in an intact uterus and spread to the peritoneum.Rarely does an ovarian abscess form during puerperium[33].These are thought to be brought on by bacterial invasion through a hole in the ovarian capsule.Women often arrive 1 to 2 weeks after giving birth, and the abscess is typically unilateral.Peritonitis can be very serious, and ruptures are frequent[33].

SEPTIC PELVIC THROMBOPHLEBITIS:

In the days before antibiotics, septic embolization and suppurative thrombophlebitis were prevalent complications.The death rate and requirement for surgical treatment for these illnesses, however, decreased with the introduction of antibiotic treatments.Septic phlebitis develops as a venous extension and has the potential to thrombose. Oftentimes, lymphangitis coexistsDue to the fact that they drain the upper uterus, which includes the placental implantation site, the ovarian veins may then get involved[14].

According to Witlin and Sibai (1995) and Brown and colleagues (1999) observations, one or both of the ovarian venous plexuses may be involved in puerperal septic thrombophlebitis.

The clot sometimes travels to the renal vein and into the inferior vena cava in one-fourth of women.Except for chills and occasional lower quadrant pain, women with septic thrombophlebitis usually lack symptoms. The diagnosis can be confirmed by pelvic CT or MR

imaging[16]. Antimicrobial therapy usually results in clinical improvement for these women, although their fever persists. Heparin therapy is a controversial[33].

PERINEAL INFECTION

Episiotomy infections are uncommon, because the operation is now performed less frequently (American College of Obstetricians and Gynecologists, 2018a; Dillon, 2019). When the anal sphincter is disrupted at delivery, the subsequent infection rate is higher and is likely influenced by intrapartum antimicrobial treatment[14].

Pathogenesis and Clinical Course:

As is clear, dehiscence may make perineal laceration infection more difficult.

Smoking, coagulation issues, and human papillomavirus infection are additional causes of separation.Localized pain and frequent urination, either with or without urine retention, are common indications of infection. In severe cases, the entire vulva may swell, ulcerate, and become exudate-covered. Although life-threatening, septic shock or necrotizing fasciitis is rare. Cervical wounds are rarely obviously infected, however they can develop metritis. Deep wounds that penetrate the broad ligament's base directly may get infected and result in lymphangitis, parametritis, and bacteremia[14,22].

Treatment:

The treatment of infected surgical wounds is the same for infected episiotomies.

Close monitoring and broad-spectrum antibiotic therapy alone may be suitable in cases of evident cellulitis in female patients without purulence.Purulence causes drainage to be established, and the infected wound is typically debrided and its sutures removed.

Local wound treatment is combined with intravenous antibiotics when there is dehiscence.

Prior to making an early repair, careful planning is crucial. The surgical incision needs to be thoroughly cleaned and infected removed[14].

Secondary repair can be carried out if the wound's surface is free of exudate and coated in pink granulation tissue.It is necessary to sufficiently mobilise the tissue, paying close attention to locate and activate the anal sphincter muscle.To prevent recurrent dehiscence, a tension-free suture line is necessary.Similar to how the first episiotomy wound is closed, the secondary wound is closed in layers. Postoperative care includes local wound care, stool softeners, and nothing per vagina or rectum until healed. Hard stools risk wound disruption, but liquid stool can seep between sutures to reincite infection[(14,22)].

Preoperative Protocol for Early repair of Episiotomy Dehiscence:

1.Open wound, remove sutures, begin intravenous antibiotics.

2.Initiate wound care:

- Institute sitz bath several times daily or hydrotherapy
- Provide adequate analgesia or anesthesia (regional or general) for initial debridement.
- Scrub wounds daily with twice with a povidine iodine solution.
- Debride necrotic tissue.
- Close wounds when afebrile and pink, healthy granulation tissue is present.
- Provide enemas prior to fourth degree repair.

Institute postoperative stool softeners, normal diet, nothing per vaginum or per rectum[(14,22)].

BREAST INFECTIONS

The antepartum complication of parenchymal infection of the mammary glands is uncommon, while the postpartum incidence of mastitis is about 3%.The use of prophylactic measures to avoid breast infection is not supported by research.Nursing challenges, damaged nipples, and

oral antibiotic medication are risk factors. Suppurative mastitis seldom develops before the end of the first postpartum week, and symptoms typically don't show up until the third or fourth week.

Significant engorgement typically occurs before inflammation, and infection is nearly always unilateral. Chills or rigors are one of the symptoms, which are shortly followed by fever and tachycardia. The intensivity of pain is more, and the breast(s) becomes hard and congested[14].

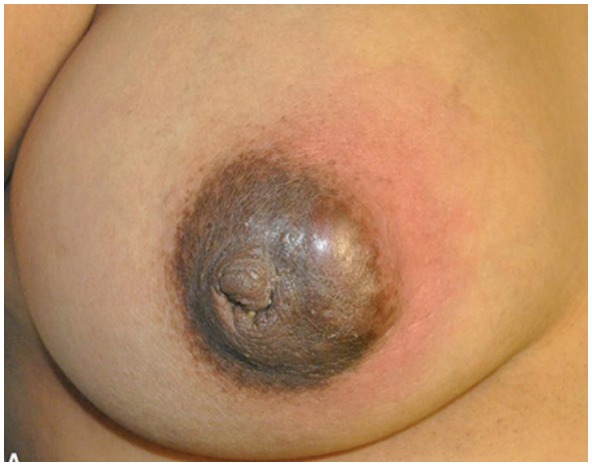


Fig 4: Puerperal mastitis with breast abscess: Indurated, erythematous skin overlies the area of right-sided breast infection.

Etiology:

The breast(s) become red and hard and there is excruciating pain.

Abscess is seen in 10% of women with mastitis. Toxic shock syndrome caused by staphylococcus aureus due to mastitis although rare has been reported in some cases. The immediate source of mastitis-causing organisms is almost always the newborn's nose and throat. These fissures or small abrasions are the entry site for bacteria.The infecting organism can usually be cultured from milk.Suppurative mastitis among lactating women occasionally reaches pandemic levels.These outbreaks frequently follow the emergence of a fresh strain of staphylococcus that is resistant to antibiotics[14].

Management:

The infection often goes away in 48 hours, provided that the proper mastitis treatment is performed before suppuration develops.Before starting treatment, many advise expressing milk from the afflicted breast onto a swab and culturing it.A successful nosocomial infection surveillance programme can use information from bacterial identification and antibiotic sensitivity.Therefore, the initial antibiotic selection is impacted by the institution's past experiences with staphylococcal infections[14,33].Dicloxacillin, 500 mg orally four times daily, may be started empirically.

Women who are sensitive to penicillin are prescribed erythromycin.Vancomycin, clindamycin, or trimethoprim-sulfamethoxazole are prescribed if resistant, penicillinase-producing staphylococcus species are the source of the infection or if resistant organisms are suspected while waiting for the results of the culture.Strong milk expression may be the only form of treatment necessary (Thomsen, 1984). The infant may occasionally refuse to suckle from the irritated breast[14].

This is most likely unrelated to any changes in the flavour of the milk and is instead a result of swelling and engorgement, which can make it difficult to hold the areola.

This can be mitigated by pumping. It is advisable to start sucking on the breast that is not involved when nursing bilaterally. As a result, let-down can start before moving to the delicate breast.Breastfeeding is permitted among mothers with human immunodeficiency virus (HIV) in regions with limited resources[(14,22)].

BREAST ABCESS

When defervescence does not occur 48 to 72 hours after therapy or when a palpable lump is present, an abscess should be suspected. Sonographic imaging is useful once more.

Large breast abscesses have been reported, and in one case, 2 L of pus were discharged (Martic, 2012). Surgical drainage has been the standard treatment, which typically necessitates general anaesthesia.Ideal incision placement for an aesthetic outcome is along Langer skin lines[!4].

Early cases typically only require a single incision across the most reliant part of the fluctuation. Multiple abscesses necessitate numerous incisions and loculation disruption.Gauze is used to fill the resulting cavity loosely, and a new, smaller pack should be added after 24 hours.Sonographically guided needle aspiration with local analgesia has gained popularity more recently (Patani, 2018). This has a success rate of between 80% and 90%. (Geiss, 2014).

Naeem and associates (2012) conducted a randomised experiment to contrast surgical drainage and aspiration.At 8 weeks, they discovered that 93 percent of patients who had aspiration had healed compared to 77 percent of patients who had surgical drainage[22].

Findings on the sonogram, the first antimicrobial choice, and the infecting organism do not indicate aspiration failure.In the case of an abscess that is not healing, other aetiologies should be taken into account. Granulomatous mastitis can occasionally manifest as puerperal mastitis (Ding, 2021 Freeman, 2017).Other factors include the risk of cancer or TB[22].

PUERPERAL SEPSIS:

Sepsis is a fatal condition. “Hippocrates quoted that sepsis is characterized by festering wounds with rotting flesh. Semmelweis, Pasteur, and others in the nineteenth century stated sepsis as a systemic infection referred to as “blood poisoning” that occurred due to pathogen invasion and spread in the bloodstream of the host”[(15)].

In 1992, Bone and colleagues made the claim that the host, and not the bacterium, was to blame for sepsis' pathophysiology. They outlined sepsis specifically as a systemic inflammatory reaction to infection. However, septicemia was not a required condition nor a helpful phrase, because sepsis developed in response to numerous different diseases. Inspite of using the term septic shock for a subset of sepsis cases characterised by hypotension despite sufficient fluid

resuscitation and perfusion anomalies, these researchers advocated the terms severe sepsis and septic shock[40,43].

Twenty years of research have shown that many patients experience immediate organ dysfunction as a result of infection, although there is no detectable inflammatory excess (i.e., without the systemic inflammatory response syndrome [SIRS]). The Third International Consensus Definitions further stipulated that septic shock be defined as a subset of sepsis cases in which underlying circulatory and cellular/metabolic abnormalities are severe enough to significantly increase mortality risk. This is due to the wide variation in how septic shock is identified in research, clinical, or surveillance settings[12].

"Sepsis-3 clinical criteria for sepsis include the following to help clinicians recognise sepsis and septic shock at the bedside.

(1) A suspected infection and

(2) Acute organ dysfunction, defined as an increase by two or more points from baseline (if known) on the sequential (or sepsis-related) organ failure assessment (SOFA) score".

Table 4. Definitions and Criteria for Sepsis and Septic Shock.

CONDITION	DEFINITION	COMMON CLINICAL FEATURES	CRITERIA IN 1991/2003 ("SEPSIS-1"/ "SEPSIS-2")	CRITERIA IN 2016 ("SEPSIS-3")
Sepsis	"A life-threatening organ dysfunction caused by a dysregulated host response to infection"	Include signs of infection, with organ dysfunction, plus altered mentation; tachypnea; hypotension; hepatic, renal, or hematologic dysfunction	Suspected (or documented) infection plus ≥2 systemic inflammatory response syndrome (SIRS) criteria	Suspected (or documented) infection and an acute increase in ≥2 sepsis related organ failure assessment (SOFA) points
Septic shock	"A subset of sepsis in which underlying circulatory and	Signs of infection, plus altered mentation,	Suspected (or documented) infection plus	Suspected (or documented) infection

	cellular/metabolic abnormalities lead to substantially increased mortality risk".	oliguria, cool peripheries, hyperlactemia	persistent arterial hypotension (systolic arterial pressure, <90 mmHg; mean arterial pressure, <60 mmHg; or change in systolic by >40 mmHg from baseline	plus, vasopressor therapy needed to maintain mean arterial pressure at ≥65 mmHg and serum lactate >2.0 mmol/L despite adequate fluid resuscitation

SIRS criteria include 1 point for each of the following (score range, 0–4): fever >38°C (>100.4°F) or <36°C (<96.8°F); tachypnea with >20 breaths per min; tachycardia with heart rate >90 beats/min; leukocytosis with white blood cell count >12,000/μL; leukopenia (<4000/μL) or >10% bands.

qSOFA score is a 24-point measure of organ dysfunction that uses six organ systems (renal, cardiovascular, pulmonary, hepatic, neurologic, hematologic), where 0–4 points are assigned per organ system.Criteria for septic shock include sepsis plus the need for vasopressor therapy to elevate mean arterial pressure to ≥65 mmHg with a serum lactate concentration >2.0 mmol/L despite adequate fluid resuscitation.

Worldwide, sepsis is a significant factor in maternal morbidity and mortality. A positive outcome can be ensured with early detection and prompt treatment. eighth article A generalised inflammatory response in the host with systemic symptoms known as sepsis may be induced by one or more infectious pathogens[8]. An abrupt shift in the overall Sequential Organ Failure Assessment Score (SOFA) is used to identify end organ damage[1]. Sepsis, along with

haemorrhage, hypertension, and abortion, is one of the four leading causes of maternal mortality, according to WHO.[1,40,41] Any bacterial infection of the vaginal tract that develops after a baby is born is referred to as puerperal sepsis.

In most cases, the symptoms and indicators don't start to show up for at least 24 hours after birth. However, if the lady experienced a lengthy membrane rupture or labour without preventive antibiotics, the sickness can show itself sooner . According to the most recent research, sepsis is defined as an infection that has systemic symptoms and is present (either suspected or documented). When sepsis-induced organ failure or tissue hypoperfusion occur, there is severe sepsis present.Sepsis-induced hypotension that lasts despite adequate fluid resuscitation is referred to as septic shock[43].

In pregnancy it is more challenging. There are currently no validated sepsis definitions for pregnant women. RCOG with green top guideline shave been used recently for diagnosing and management of sepsis in pregnant women and post delivery. In cases of pre-labor membrane rupture, during labour, or in the early postpartum period before healing of lacerations in the vaginal tract and the placental site have taken place, the uterine infection may begin before the initiation of labour[33].

Antibiotics should be used in situations of pre-labor membrane rupture either to treat amnionitis if the woman has a fever and foul-smelling vaginal discharge, or as a preventative step to lower the chance of infection[13.]Beyond the uterus, it may also affect the ovaries and fallopian tubes. Additionally, it may affect the pelvic cellular tissue, resulting in parametritis, the pelvic peritoneum, resulting in peritonitis, and the bloodstream, resulting in septicemia[14].

“The following reasons make women more susceptible to infections during the postpartum period::

1. large, warm, dark and moist placental implantation site, which allows the bacteria to grow very quickly.

2. Rich blood supply of the placental implantation site, which is invaded by large blood vessels leading directly into the main venous circulation. This cause bacteria in the placental site to move enter more easily into the bloodstream.

3. The placental site is accessible via the genital tract to both endogenous and exogenous microorganisms.

4) At the time of delivery,there may be tears in the cervix, vagina or perineal area or have had an episiotomy".

These injured areas are prone to infection, particularly if delivery is conducted in aseptic precautions[8].

EPIDEMIOLOGY OF SEPSIS

Overall, sepsis frequently contributes to morbidity and mortality. In the US, it is the main reason why individuals who are critically ill pass away. Sepsis in mothers is a rare but persistent disorder.The incidence and prevalence of sepsis reported by various studies worldwide are probably unreliable because the term "sepsis" and its associated disorders are not universally used, particularly in obstetrics[8].

According to an old study, 7.5 per 1000 obstetric admissions had bacteremia, and 8–10% of those patients had sepsis. Due to the younger age, less comorbidities, and typically confined source of infection, it appears that sepsis during pregnancy may have a better prognosis than in the non-pregnant population[46,48]. Sepsis is thought to be responsible for 15% of maternal fatalities globally today. Over the past ten years, sepsis-related mortality have nearly doubled in the United Kingdom. In actuality, genital tract sepsis associated with community-acquired Group A streptococcal illness was the most frequent cause of direct maternal death[47,43].

CAUSATIVE ORGANISMS OF PUERPERAL SEPSIS

Some of the most common bacteria causing puerperal sepsis are

- Streptococcus bacteria
- Staphylococcus aureus
- E. coli
- Clostridium species
- Chlamydia trachomatis[12,33]

ENDOGENOUS BACTERIA:

"These are bacteria that typically exist in the rectum and vagina without being harmful (e.g. some types of streptococci and staphylococci, E. coli, clostridium welchii).

"Endogenous bacteria can still cause infection even when a clean delivery strategy is used[16]. Endogenous bacteria can become harmful and cause infection if:

a) They are carried into the uterus, usually from the vagina, by the examining finger or by instruments during pelvic examinations there is tissue damage, i.e. bruised, lacerated or dead tissue (e.g. after a traumatic delivery or following obstructed labour).

b) There is prolonged rupture of membranes because microorganisms can then enter the uterus"[33,16].

EXOGENOUS BACTERIA

"These are bacteria which are introduced into the vagina from the outside (streptococci, staphylococci, clostridium tetani, etc.).

Exogenous bacteria can be introduced into the vagina:

A) By unclean hands and unsterile instruments by droplet infection (e.g. a health provider sneezing, coughing onto own hands immediately prior to examination)

B) By foreign substances that are inserted into the vagina (e.g. herbs, oil, cloth)

C)By sexual activity"[33.16].

POSTPARTUM TETANUS

Infection of the mother or child brought on by Clostridium tetani is known as postpartum tetanus.

Tetanus bacilli, which grow in the intestines of animals and humans, are particularly prevalent in rural areas[16].

They are spread by the excrement of humans and animals and are found in soil and dust.

Through a skin break or laceration, the organisms enter the body. (WHO)In the case of puerperal sepsis, they may enter via lacerations of the genital tract or through the unhealed placental site.

In some nations, it is common practise to inject potentially infectious herbs or other medicines into the vagina during or after labor in the false notion that it will be beneficial.

The umbilical cord of newborns is frequently the point of entry, especially if it is severed with a dirty tool. In some cultures, the cord is also dressed with herbs or cow dung"[16].

"RISK FACTORS FOR SEPSIS IN OBSTETRICS

- Abnormal vaginal discharge
- PID
- Infection with group B streptococci
- Twin pregnancy
- Infertility treatment history
- Invasive procedures like amniocentesis
- Cervical cerclage
- PROM
- Lower segment cesarean section
- Trauma to vagina
- RPOC"[16,33].

"**AETIOLOGY OF SEPSIS IN OBSTETRICS**

1)OBSTETRIC CAUSES

A) Genital tract causes:

- Septic abortion
- Chorioamnionitis
- Endometritis
- Wound infection following caesarean section or episiotomy or vaginal and perineal lacerations.
- Infection following invasive procedures (infected cerclage, necrotising fasciitis)

B) Non-genital tract causes:

- Pyelonephritis
- Lower urinary tract infection
- Breast infection
- Septic pelvic thrombophlebitis

2) NON-OBSTETRIC CAUSES:

- Appendicitis
- Cholecystitis
- Pancreatitis
- Gastroenteritis
- Pharyngitis
- Tuberculosis
- Malaria
- Pneumonia

HIV Influenza A and B (secondary infection)"[16,15].

PATHOPHYSIOLOGY OF SEPSIS

PATHOGENESIS

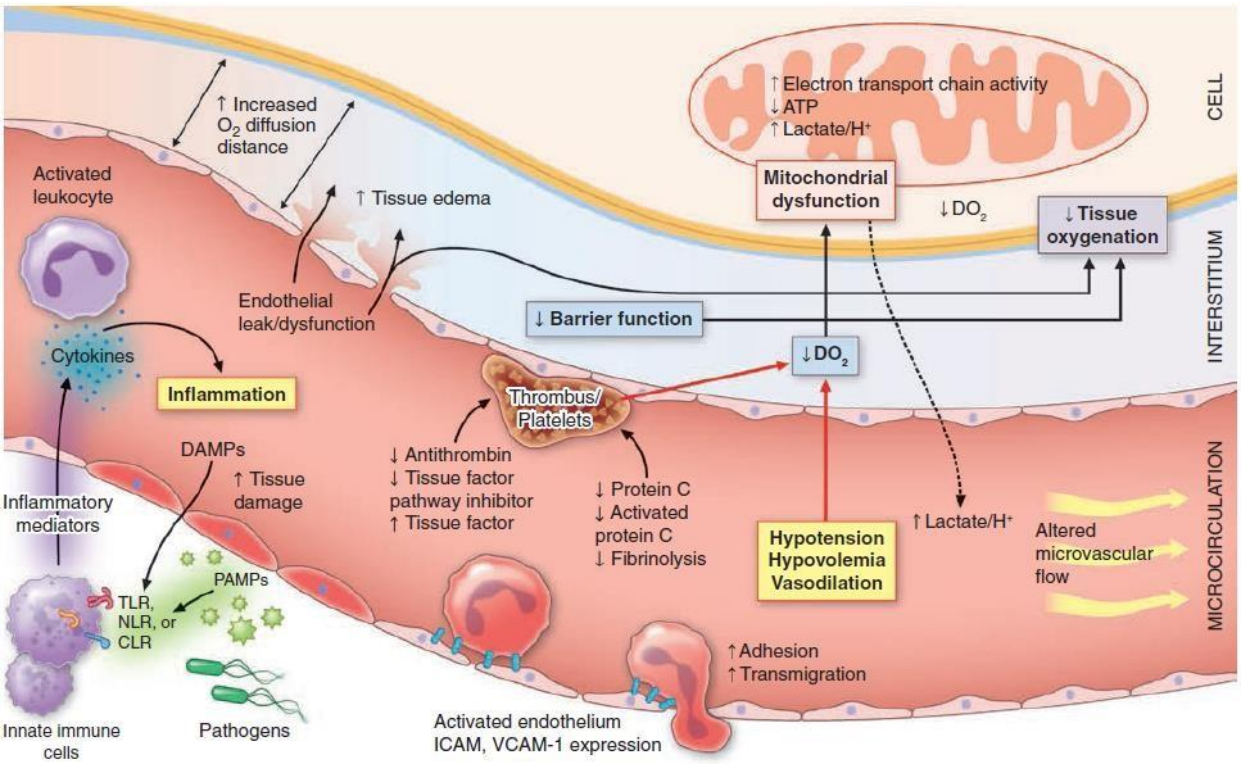
The clinical signs of sepsis were once thought to be caused by an overly inflammatory host response (SIRS). Infection results in a far more complex, variable, and protracted host response than was previously believed, it has lately been clear[15].

The patient's clinical course and the host reaction change over time. In sepsis, "collateral" tissue damage is typically caused by proinflammatory responses (aimed at removing pathogens), whereas anti-inflammatory responses are thought to increase susceptibility to secondary infections that develop later in the course. These mechanisms can be understood as the interaction of two "fitness costs": organ damage caused directly by the virus and organ damage caused by the host's immune response. Whether a simple infection develops into sepsis will depend on the host's capacity to withstand as well as endure both direct and immunopathologic harm[12,15].

Initiation of Inflammation

Our understanding of pathogen identification has greatly expanded during the last ten years. Innate immune cells, in particular macrophages, identify and bind pathogens to begin the host response to infection. Immune cells have pathogen-associated molecular patterns (PAMPs), which are conserved across microbial species, that pathogen recognition receptors (PRRs) bind to. Innate immunity is activated and inflammatory gene transcription is upregulated as a result of PRRs' interaction with PAMPs.

Fig 5: Pathogenesis of sepsis-induced organ and cellular dysfunction.



Through this contact, surface adhesion molecules are expressed, causing PMN to assemble and adhere to the vascular endothelium[15].

PMNs travel to the infection site through a multi-step process of rolling, adhesion, diapedesis, and chemotaxis, releasing inflammatory mediators that cause local vasodilation, hyperemia, and enhanced microvascular permeability. Sepsis happens when these local proinflammatory immunological responses intensify, triggering a broader immune reaction. Although the exact cause of this malignant transformation is yet unknown, the invasion-related consequences, excessive synthesis of proinflammatory mediators, and complement system activation have all been suggested as possible causes[15].

Organ Dysfunction

Cellular and hemodynamic changes are crucial, even if the processes behind organ failure in sepsis are still poorly understood. Altered inflammatory response, cellular changes, endothelial dysfunction, and circulatory abnormalities are important contributing causes. Cellular damage brought on by abnormal inflammation raises the possibility of organ malfunction. A significant

part is played by cellular changes, such as defective cell death pathways, mitochondrial malfunction, and intracellular management of reactive oxygen species[2].

When an insult is severe or lasts for a long time, the body experiences bioenergetic failure, harmful reactive oxygen species are generated, and apoptosis, which results in irreversible cell death and organ failure. Endothelial dysfunction is also crucial to the pathophysiology of multiple organ failure common to sepsis[2] . In sepsis, the vascular endothelium's cell-cell interactions are broken down for a variety of reasons, leading to a loss of barrier integrity and subcutaneous and body cavity oedema. Disruption of the endothelium glycocalyx also increases endothelial permeability and causes the development of oedema. The transport of oxygen to tissue is further hampered by microcirculatory problems, such as microthrombosis and decreased capillary density, which leads to the emergence of organ failure. New research indicates that the stomach may independently contribute to the malfunctioning of organs linked to sepsis[12,15].

Anti-inflammatory Mechanisms

The immune system has humoral, cellular, and neurological pathways that could amplify the proinflammatory response's potentially negative effects. Phagocytes have the ability to transition to a pro-tissue repair anti-inflammatory phenotype, and regulatory T cells and myeloid-derived suppressor cells further lower inflammation. The so-called neuroinflammatory reflex may also be at play; sensory information is transmitted to the brainstem via the afferent vagus nerve, from which the efferent vagus nerve activates the splenic nerve in the celiac plexus, causing norepinephrine to be released in the spleen and a subset of CD4+ T cells to secrete acetylcholine[2].

The release of acetylcholine inhibits the release of proinflammatory cytokines via binding to seven cholinergic receptors on macrophages. Animals are more susceptible to endotoxin shock

when this neural-based mechanism is disrupted by vagotomy, whereas systemic inflammation is reduced in experimental sepsis when the efferent vagus nerve or 7 cholinergic receptors are stimulated[2].

Immune Suppression

Occasionally, patients who recover from early sepsis but need ongoing intensive care show signs of a weakened immune system. Despite receiving antibiotic therapy, these patients may continue to have infectious foci or may experience the reactivation of latent infections. Numerous research have shown that individuals with sepsis have decreased blood leukocyte reactivity to pathogens; more recently, postmortem studies have shown that splenocytes taken from ICU patients who died of sepsis showed severe functional impairments. Both the lungs and the spleen displayed immunosuppression; parenchymal cells in both tissues expressed more T cell-inhibitory receptor ligands. Sepsis has been linked to increased apoptotic cell death, particularly in B cells, CD4+ T cells, and follicular dendritic cells. linked death and immune suppression[2,15].

CLINICAL MANIFESTATIONS

According to the initial site of infection, the causative pathogen, the pattern of acute organ failure, the patient's underlying health, and the length of time before therapy is started, the specific clinical manifestations of sepsis can vary quite a little. Both infection and organ failure might have mild symptoms. A lengthy list of potential early sepsis warning symptoms is provided by guidelines. The temperature and white blood cell (WBC) count frequently return to normal once sepsis has been confirmed and the illness that caused it is thought to be under control. Organ dysfunction, however, usually continues[33].

Cardiorespiratory Failure

The classic indicator of respiratory compromise is acute respiratory distress syndrome (ARDS), which is characterised by hypoxemia and bilateral infiltrates of noncardiac origin that occur within 7 days after the suspected infection. According to the Berlin criteria, ARDS can be classed as mild (Pao2/Fio2, 201-300 mmHg), moderate (101-200 mmHg), or severe (edema below 100 mmHg due to cardiac failure or volume overload). The most common symptom of cardiovascular impairment is hypotension. Frank hypovolemia, diffuse capillary leakage that causes an imbalance in blood flow and intravascular volume, decreased systemic vascular resistance, or impaired myocardial function are possible causes.

Hypotension frequently continues even after appropriate volume expansion, necessitating the administration of vasopressors. Systemic vascular resistance may be fairly high in the early stages of shock when volume status is lowered, and cardiac output may be low[(2)].

Kidney Injury

Acute kidney damage (AKI) is observed in more than 50% of septic patients, which more than doubles the chance of dying in the hospital. Oliguria, azotemia, and rising serum creatinine levels are signs of AKI, which commonly calls for dialysis. AKI caused by sepsis is accompanied by poorly known processes. Without overt hypotension, AKI can happen in up to 25% of patients. Current mechanistic research reveals that sepsis-induced AKI is caused by a variety of factors beyond organ ischemia, including inflammation, diffuse microcirculatory blood-flow irregularities, and cellular bioenergetic responses to injury[(2,15)].

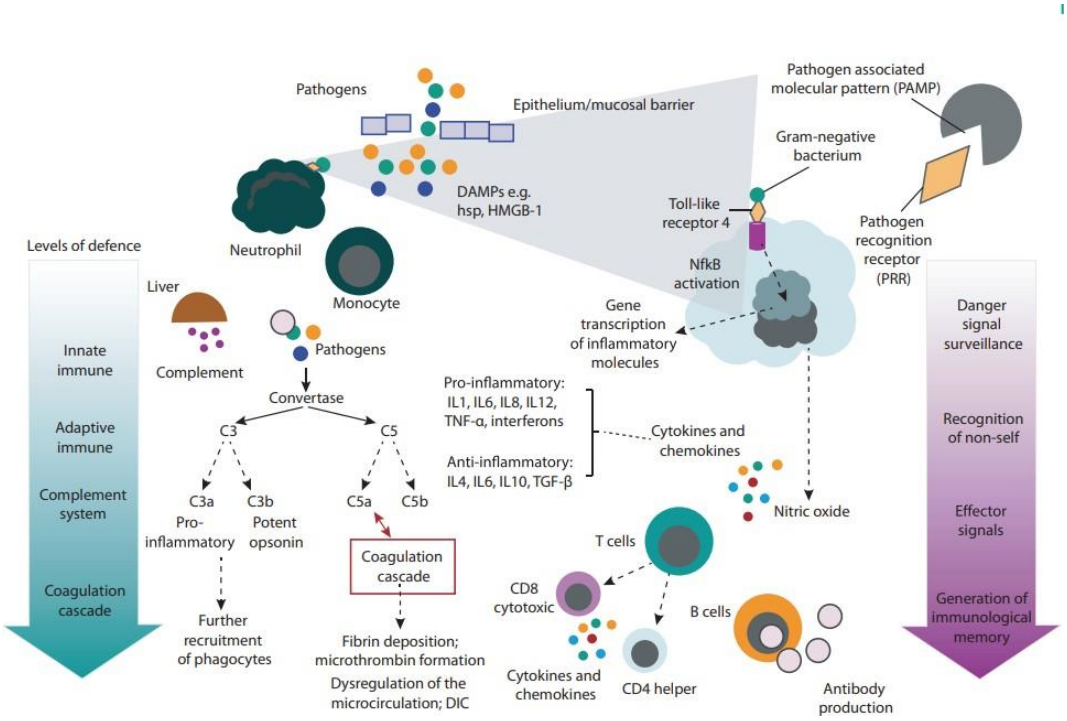
Neurologic Complications

Coma or delirium are common symptoms of central nervous system disorders. Electroencephalographic findings are often consistent with non-focal encephalopathy, and imaging examinations normally indicate no focal abnormalities. Without signs of an underlying

main central nervous system infection, sepsis-associated delirium is thought to be a diffuse cerebral dysfunction brought on by the inflammatory response to infection[15]. Consensus recommendations urge delirium screening using effective and trustworthy instruments like the Intensive Care Delirium Screening Checklist and the Confusion Assessment Method for the Intensive Care Unit (CAM-ICU) (ICDSC).

Myopathy and polyneuropathy associated with critical illness are very frequent, particularly in individuals with a protracted course. Neurologic consequences might be severe for sepsis survivors. 25–50% of sepsis survivors experience post sepsis syndrome, a developing pathologic condition marked by persistent cognitive impairment and functional dysfunction. However, a combination of cerebrovascular injury, metabolic derangements, and neuroinflammation is offered as one possible explanation for the neurocognitive derangements in post-sepsis syndrome[15].

Fig 6:Pathophysiology of sepsis



Sepsis is a complicated pathophysiology that involves the interplay of numerous cellular pathways through positive and negative feedback loops. Uncontrolled communication between these routes may result in sepsis and broad tissue harm when it causes organ malfunction. When the tissue epithelium/mucosal barrier is breached, monocytes and neutrophils of the innate immune system are the first to identify pathogens for removal[(2)].

Pathogen associated molecular patterns [PAMPs] are conserved surface molecular regions of invasive pathogens that serve as danger signals and are recognised by monocyte pathogen recognition receptors (PRRs), such as the toll-like receptor 4. (TLR-4). In a nutshell, the interaction between the two activates inducible nitric oxide synthase (iNOS), which results in excessive nitric oxide production that aids in pathogen clearance but also acts as a potent vasodilator that attenuates cardiac function and triggers monocyte intracellular gene

transcription of pro-inflammatory intracellular cytokines and chemokines via NFKB. This attracts more immune cells to the site of infection[2,15].

By phagocytosis or enzymatic breakdown, neutrophils eliminate bacteria, although this process could harm nearby tissue. As a result, molecules known as danger-associated molecular patterns (DAMPs) are released, including heat shock protein (HSP) and high-mobility group box-1 (HMGB-1), which can cause tissue damage, cell recruitment, and cytokine production.

T and B lymphocytes, which are part of the adaptive immune system, are able to recognise particular bacterial related antigens. T cells attack the pathogen either by producing cytokines or engaging in cytotoxic activities (CD8) (CD4). During the contraction phase of the immune response, adaptive responses from T regulatory cells and other immune suppressive leukocytes release immuno-modulatory molecules (IL-10, TGF-b), coupled with T-helper CD4 synthesis of "anti-inflammatory" cytokines (IL-4)[2,15].

In order to help neutrophil phagocytosis, B cells create antigen-specific antibodies that opsonize to pathogens together with complement.

The complement cascade, which interacts with the coagulation system, can be started by pathogens. Disseminated intravascular coagulation (DIC), which presents as a mixed clinical picture of microthrombus development and haemorrhage, is linked to severe sepsis[15].

"DIAGNOSTIC CRITERIA FOR SEPSIS IN PREGNANT WOMEN.

1)"General variables:

- Pyrexia (38.3 C)
- Hypothermia
- Tachycardia(HR>100 bpm)
- Tachypnoea(RR>20cpm)
- Altered sensorium

- Generalised edema
- Raised blood sugars (plasma glucose>7.7 mmol/l) in patients without diabetes history[2].

2) Inflammatory variables:

- Increase in white blood cell count >12x10^9 /l
- Decrease in white blood cell count <4 x10^9 /l
- >10% immature band forms and Plasma C-reactive protein>7 mg/l with normal leucocyte counts[2].

3) Hemodynamic variables:

SBP<90 mm Hg, MAP<70 mm Hg, or an SBP decrease>40 mm Hg depicting areterial hypotension[2].

4) Organ dysfunction variables:

- Arterial hypoxemia.
- Decreased urine output<0.5 mL/kg/hr for at least 2 hours inspite of adequate fluid resuscitation.
- Increased creatinine of >4.2 mmol/l.If creatinine is >176 mmol/l it indicates severe sepsis.
- Abnormalities of coagulatory function with raised INR>1.5 or aPTT>60 s)
- Decreased platelet count of <100 x 10^9 /L
- Increased bilirubin levels of >70 mmol/l)[2,15].

5) Tissue perfusion variables:

- Increased serum lactate of >4 mmol/l.

  Mottling or decreased capillary filling"[15]

Hemodynamic response:

Early on in the course of sepsis, the host experiences a hemodynamic reaction. The release of nitric oxide causes vasodilatation, which leads to "relative" hypovolemia and the sympathetic nervous system's activation, which causes tachycardia. Pregnant patients experience intricate physiologic changes that must be taken into account[33].

Cardiovascular system:

Because sepsis and normal pregnant cardiovascular changes are similar, they may disguise the condition's early symptoms and exacerbate poor organ perfusion. As the symptoms of septic shock worsen, indicators of hypoperfusion appear, and the tissues' diminished ability to receive oxygen results in anaerobic metabolism, lactate buildup, decreased uterine perfusion, foetal acidosis, and end-organ failure[15,33].

Haematologic system:

Pregnancy is characterised by an increase in leukocyte count, a decrease in platelet count, an increase in clotting factors, and a decrease in fibrinolysis. During advanced sepsis, these modifications may favour intravascular fibrin production. During pregnancy, both plasma volume and red cell mass increase, although the former does so more. Colloid osmotic pressure is reduced as proteins decline. Pregnant patients are therefore more vulnerable to pulmonary oedema during sepsis[2,15].

Renal system:

Pregnancy causes an increase in renal plasma flow and glomerular filtration rate, which lowers creatinine levels. Therefore, even normal levels of this metabolite in non-pregnant women's serum can indicate mild renal impairment[2,15].

Gastrointestinal system:

Pregnancy lowers the smooth muscle tone of the gastrointestinal system, increasing the risk of aspiration pneumonia (Guinn et al. 2007). Sepsis causes the gastric mucosa to become hypoperfused, which causes mucosal atrophy. This, in turn, causes bacterial translocation and an aggravation of the illness[14].

Respiratory system:

In the upper airways during pregnancy, there is mucosal oedema, hyperaemia, and capillary congestion. Tidal volume is increasing, residual volume and functional reserve capacity are decreasing, and total lung capacity is somewhat declining, but vital capacity is unaffected, all of which are related to pulmonary function[58]. According to Elkus and Popovich (1992) and Pereira and Krieger (2004), there is a considerable rise in minute ventilation that results in a drop in PaCO2 and compensatory respiratory alkalosis. This is advantageous in a healthy pregnancy, but it is harmful in sepsis because it increases the risk of a metabolic acidosis and a quick reduction in oxygenation[2,15].

Immune system:

The outdated idea that pregnancy is an immunocompromised state produced in order not to reject the developing foetus, which predisposes the mother to infectious diseases, is one of the gaps in our understanding of infectious disorders during pregnancy[64]. Pregnancy is now thought of as a state of immunomodulation, where a capable immune response is essential to protect both the mother and the foetus (Mor and Cardenas, 2010; Mor et al.Pregnancy is divided into three distinct immunological stages that largely correlate to the first, second, and third trimester[2].

The first stage begins with implantation and placentation being a phase of strong inflammatory response, which affects the mother's well-being.

The second stage is an anti-inflammatory response, which is necessary for rapid foetal growth and development.

The final stage is characterised by increased inflammation, in order to prepare parturition and delivery of the baby[33].

In addition, because the placenta and fetus also react to microbial infections, they serve as an extra immune organ. The embryo: Most information on maternal sepsis has focused on mother outcomes, with scant reporting on fetal outcomes[59]. The inflammatory process that occurs during sepsis can harm or destroy the fetus. This is due to the disruption of the maternal-fetal barrier. Though it is thought that the fetus is more resistant to the inflammatory process than its mother, perhaps because its immune system is not yet fully established in gestation[2,33].

CLINICAL PRESENTATION

The following symptoms and signs occur in puerperal sepsis:

- Pyrexia (temp> 38°C or high)
- Malaise or chills
- Pain in the lower abdomen
- On palpation of uterus, it will be tender
- Uterus fundal height is plapated above the umbilicus(subinvolution)
- Abnormal and foul smelling vaginal discharge
- Minimal vaginal bleeding
- Shock[15].

INVESTIGATIONS

DIAGNOSIS

Laboratory and Physiologic Findings

A variety of laboratory and physiologic changes are found in patients with suspected infection who are at risk for sepsis. In a 12-hospital cohort of electronic health records related to >70,000 encounters **(Fig. 9).**

FIGURE 7 Distribution of systemic inflammatory response syndrome (SIRS) and sequential (or sepsis related)organ failure assessment (SOFA) variables among infected patients at risk for sepsis.

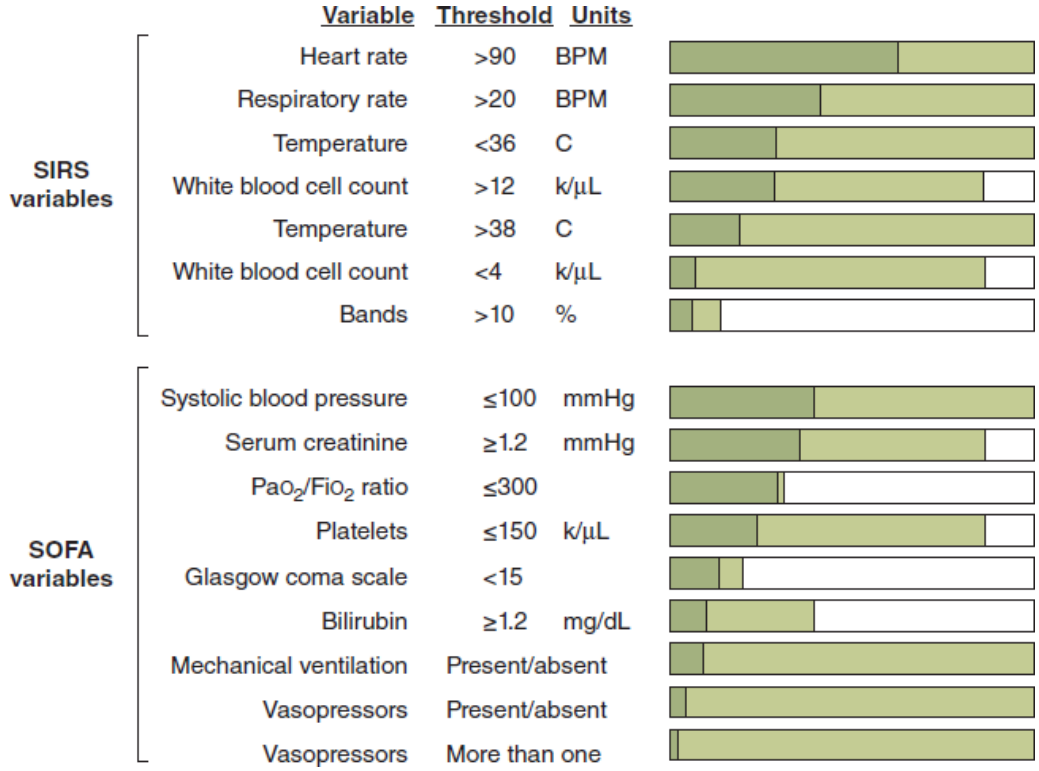
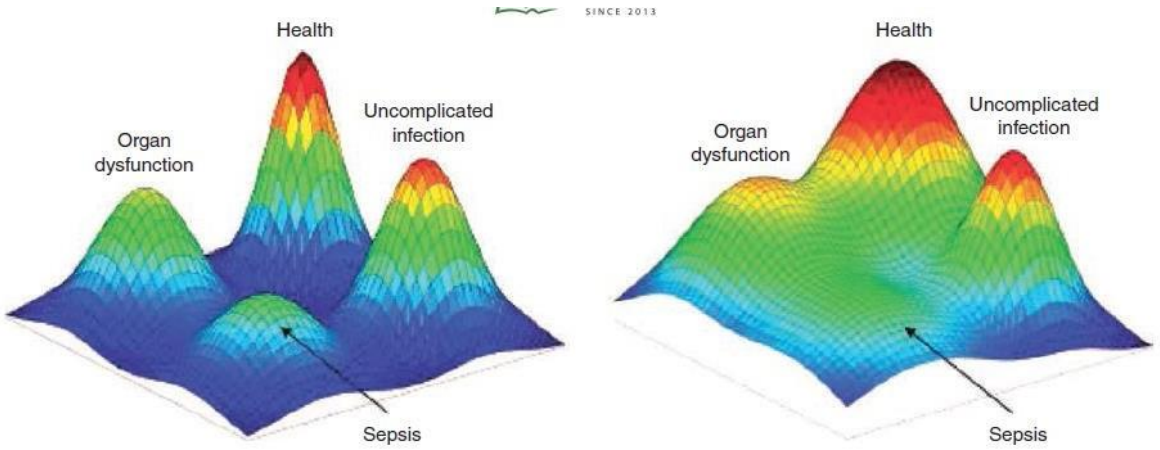


FIGURE 8: Schematic of the importance of accurate, easy-to-use criteria for sepsis and its components, infection and organ dysfunction.



In the ideal case (*left*), criteria clearly distinguish sepsis patients from other patients with uncomplicated infection or organ dysfunction. The reality (*right*), however, is that existing criteria fail to make clear distinctions, leaving a significant proportion of patients in areas of uncertainty[15].

The Sepsis Definitions Task Force, with the introduction of Sepsis-3, has recommended that, once infection is suspected, clinicians consider whether it has caused organ dysfunction by determining a SOFA score. The SOFA score ranges from 0 to 24 points, with up to 4 points accrued across six organ systems. The SOFA score is widely studied in the ICU among patients with infection, sepsis, and shock[2,15].

With ≥2 new SOFA points, the infected patient is considered septic and may be at ≥10% risk of in-hospital death. To aid in early identification of infected patients, the quick SOFA (qSOFA) and the National Early Warning Score (NEWS) scores are proposed as clinical prompts to identify patients at high risk of sepsis outside the ICU, whether on the medical ward or in the emergency department. The qSOFA score ranges from 0 to 3 points, with 1 point each for systolic hypotension (≤100 mmHg), tachypnea (≥22 breaths/min), or altered mentation. A

qSOFA score of ≥2 points have a predictive value for sepsis similar to that of more complicated measures of organ dysfunction[2]. The National Early Warning Score (NEWS) is an aggregate scoring system derived from six physiologic parameters, including respiratory rate, oxygen saturation, systolic blood pressure, heart rate, altered mentation, and temperature. Recent work has also shown that, although SIRS criteria may be fulfilled in sepsis, they sometimes are not and do not meaningfully contribute to the identification of patients with suspected infection who are at greater risk of a poor course, ICU admission, or death—outcomes more common among patients with sepsis than among those without[2,33].

Although the use of this definition as a criterion for patient enrollment in clinical trials, observational studies, and quality improvement work varies significantly, septic shock is a subset of sepsis in which circulatory and cellular/metabolic abnormalities are severe enough to significantly increase mortality risk.

Septic shock criteria

(1) sepsis plus

(2) Need for vasopressor therapy to elevate mean arterial pressure to ≥65 mmHg, with

(3) Serum lactate concentration >2.0 mmol/L after adequate fluid resuscitation[2].

A long-studied indicator of tissue hypoperfusion is arterial lactate, and sepsis is associated with higher rates of organ failure and mortality due to hyperlactemia and delayed lactate clearance. Additionally, in sepsis, a high lactate concentration may simply be a sign of poor clearance. Because of these characteristics, lactate should be utilised in conjunction with other indicators of infection and organ dysfunction rather than being employed as a sole biomarker for the diagnosis of sepsis[2,15].

SERUM LACTATE

Increased lactic acid indicates tissue hypoxia; serum lactate is a substitute marker for tissue perfusion. Accelerated aerobic glycolysis fueled by excessive beta-adrenergic stimulation results in the production of serum lactate[62]. The SSC 2016 guidelines recommend using lactic acid as a prognostic marker in sepsis, and serial lactic acid monitoring and lactic acid clearance can both forecast mortality[(1)]. There aren't any trustworthy clinical or microbiological indicators that could be utilised to identify obstetric sepsis. Microbiological cultures result in subpar sensitivity and specificity, as well as a delay in diagnosis[(8,25)].

Since it is a quick test, an immediate response can be performed to lower sepsis-related morbidity and death, aiding in the improvement of maternal health standards. Organ dysfunction occurs as septic shock progresses, and lactic acid clearance decreases and serum lactic acid levels rise as a result[17]. An increase in lactate levels indicates tissue hypoxia, which prompts aggressive fluid delivery and more targeted resuscitation to reverse tissue hypoxia and, as a result, organ failure at the pulmonary level[(7,18)].

Additionally, enhanced organ function and a decline in inflammatory biomarkers were linked to lactate clearance[(1,5)]. (1) Higher levels of lactate were consistent with a more impaired circulatory system, and lactic acid was a sign of poor perfusion. (2) Lactic acidosis develops as a result of increased generation and slower clearance of lactic acid in sepsis and septic shock, arterial hypotension, microcirculatory dysfunction, and decreased oxygen extraction from peripheral tissues[(6)].

Lactic acid measurement should be normal procedure for assessing the potentially septic obstetric patient, in addition to the other recommendations made by the Surviving Sepsis Campaign. C-reactive protein and erythrocyte sedimentation rate are two basic standard tests that are not frequently utilised during pregnancy since they can be affected by the condition. These tests are used to assess infection and inflammation outside of pregnancy[(4)].

TLC-TOTAL LEUCOCYTE COUNT

Systemic inflammation is related to alterations in circulating blood cell composition as a response of the immune system to stress or sepsis[32,36]. White blood cell (WBC) count has been identified as an important systemic inflammation marker[9].Recent reports demonstrated that the WBC count had an independent ability to predict all-cause mortality[10].

N/L RATIO- NEUTROPHIL TO LYMPHOCYTE RATIO

Relative lymphopenia with neutrophilia is the most well-known WBC condition[50]. The neutrophil-to-lymphocyte ratio (NLR) measures systemic inflammation and stress quickly and simply and expresses the severity of the illness in critically ill individuals[23]. The neutrophil-to-lymphocyte ratio (NLR), which is determined as a ratio between the counts of neutrophils and lymphocytes in peripheral blood, is a biomarker that unites the innate immune response, which is primarily supported by neutrophils, and adaptive immunity, which is supported by lymphocytes[11,5]. Invading pathogens are dealt with by neutrophils as the first line of defence using a variety of methods, such as chemotaxis, phagocytosis, release of reactive oxygen species (ROS), granular proteins, and the creation and release of cytokines[41,50].

Numerous research have assessed the relevance of inflammatory tests, such as the neutrophil-to-lymphocyte ratio (N/L ratio), in the prediction of infections and malignancies in different regions of the body, and these investigations have produced conflicting results[20].

As a result, the N/L ratio is employed as a marker of tumours and subclinical inflammation. Based on a complete blood count, the neutrophil-to-lymphocyte ratio (NLR), a commonly available biomarker, can be computed[24]. In a recent study, researchers found that the evolution of NLR in the context of infection was reversed depending on when a patient passed away. However, other studies found no correlation between NLR and mortality in sepsis patients[15,21].

The results for patients with sepsis are significantly influenced by a number of essential criteria, including the diagnostic, prognostic, and therapeutic utility of biomarkers[3].

Additionally, the relationship between elevated NLR and the need for intensive care as well as death in patients with community-acquired pneumonia has been shown to be helpful[34.35]. Neutrophils were linked to reperfusion injury and performed a significant role in the acute inflammatory response to tissue injury. Lymphocytopenias have been linked to neuroendocrine stress and cortisol production[11,20].

These two immune pathways, one signifying uncontrolled inflammation and the other a latent immunological route, were shown by NLR to be inversely connected[20].
It had been demonstrated that NLR might actively contribute to risk stratification, therapeutic optimization, and patient management[3]. NLR and lactate may be important prognostic biomarkers to optimize treatment and manage patients[21,23].

In the early stages of sepsis, elevated levels of NLR are found and are therefore helpful in making a diagnosis, especially when getting a microbiological culture is time-consuming and has a low positive rate[4]. The prognosis can also be determined by the inflammatory biomarker's late phase value[38,42]. This marker has predictive value, but it can also be used to determine when to stop giving antibiotics as the patient gets better[24]. To reinforce the function of this possible marker in sepsis, additional study with a large sample size and in individuals with noninfectious inflammatory diseases is required[36].

Even if there are still no available set cut-off levels, fluctuations in NLR over time are a marker of immune system dysfunction. Additional case-control studies that are adjusted for age groups may also be able to help with the precise identification of this range of normalcy[28]. Finally,

rather than using cut-off values, tertiles of range values based on the severity of the disease could improve the efficacy of it.[5].

MANAGING PUERPERAL SEPSIS

GENERAL MANAGEMENT OF PUERPERAL SEPSIS:

1) Isolation and barrier midwifery care of the woman:

- The main moto is to prevent the spread of infection to other women.
- If possible, the health care providers should care for the woman in a isolated room from the other women; if not, she should be placed in a corner of the ward. When caring for the woman, put on a robe and gloves, then take them off once finished.
- Hands should be sanitized properly before and after attending to these woman. A set of instruments and equipments should be kept separately for these women[16].
- Ensure that soiled dressings are properly disposed of, for as by placing them in a separate container that is periodically emptied and burning the dressings. Make sure soiled linen is put in a bag that is clearly tagged for transportation to the laundry so that it can be given special treatment.
- If at all feasible, a health care provider should be assigned specifically to care for this woman and her child.
- Visitors should be limited[16].

2) Administration of Antibiotics:

- Puerperal sepsis will kill women if therapy is ineffective or significantly delayed.

- Metritis can result in pelvic abscess, peritonitis, septic shock, deep vein thrombosis, pulmonary embolism, persistent pelvic infection with recurrent dyspareunia, tubal blockage, and infertility (uterine infection after birth).
- Timely and effective treatment will prevent all the complications[33].

Choice of antibiotic:

- "A combination of antibiotics is given until the woman is fever-free for 48 hours, and the following regime is recommended.
- Ampicillin 2 g IV every 6 hours, and gentamicin 5 mg/kg body weight IV every 24 hours, and metronidazole 500 mg IV every 8 hours.
- If fever is still present 72 hours after starting the antibiotic regime outlined above, the doctor will re-evaluate the woman and her treatment.
- Referral to a higher-level health facility may be necessary. Oral antibiotics are not necessary after stopping IV antibiotics"[33].

Tetanus toxoid:

Tetanus toxoid should be administered if there is a chance that she was exposed to tetanus (for instance, if cow dung, mud, or herbs were introduced into the vagina) and if there is any doubt regarding her immunisation history[16,12].

Giving plenty of fluids:

Women should be encouraged to take plenty of oral fluids as it prevent dehydration, help to lower the fever and, improves the circulatory volume there by improves shock. In severe cases, intravenous fluids are to be administered first[16].

4)Ruling out retained placental fragments:

Retained bits of placenta can be a cause of puerperal sepsis. Suspicion of retained placental bits is to be done if the uterus is soft and bulky, presence of excessive lochia, foul-smelling lochia with blood clots. Exploration of the uterus may be needed sometimes to remove the placental bits. Ovum forceps or a large curette can also be used when indicated[16].

EARLY TREATMENT OF SEPSIS AND SEPTIC SHOCK

Treatment of sepsis begins with prompt diagnosis. Early diagnosis of septic shock by a clinician should be made in order to give proper and timely intervention. Treatment of sepsis and septic shock are based on the guidelines provided by the Surviving Sepsis Campaign[12].

"Table 5:Guide lines for Sepsis and Septic Shock

RESUSCITATION
• Treatment should be started immediately as sepsis and septic shock are emergency conditions. • Within 3hours of onset of sepsis iv administration of fluids should be done • Normal saline or crystalloids can be used for initial resuscitation Resuscitation should be aimed towards normalizing the serum lactate levels in patients with raised serum lactate. • When commencing vasopressors in septic shock, a target mean arterial pressure of 65 mmHg is advised. • Hydroxyethyl starches and gelatins should not be used. • The first-choice vasopressor of choice is norepinephrine. • The goal of using vasopressin should be to lower the dosage of norepinephrine. • Dopamine is indicated in patients who are risk of tachyarrhythmias or relative bradycardia. • When there is persistent evidence of hypoperfusion despite adequate fluid loading and use of vasopressors dobutamine is suggested. • When the hemoglobin concentration decreases to <7.0 g/dL in the absence of acute myocardial infarction, severe hypoxemia, or acute hemorrhage then only Red blood cell transfusion is recommended.
INFECTION CONTROL
• Before starting on antimicrobial therapy, appropriate samples for microbiological cultures should be taken • As soon as feasible (within one hour), IV antibiotics should be started; more specifically, empirical broad-spectrum treatment should be utilised to treat all potential infections. • Once the sensitivity of pathogen is determined or if there is clinical improvement antibiotic therapy should be narrowed. • De- escalation of the antimicrobial therapy should be done based on the daily assessment[12].
RESPIRATORY SUPPORT
• • Compared to adult patients, goal tidal volumes for sepsis-induced ARDS should be 6 mL/kg of anticipated body weight.

• In moderate to severe sepsis-induced ARDS, rather than a lower PEEP high PEEP is used. • • In cases of severe ARDS, prone positioning is advised, and recruitment procedures and/or neuromuscular blocking medications for up to 48 hours are proposed. • If there is no evidence of tissue hypoperfusion , conservative fluid strategy should be used in sepsis-induced ARDS. • It's not advised to utilise a pulmonary artery catheter on a regular basis. • Trials of spontaneous breathing should be used in patients who are ready to wean off mechanical ventilatio12n.
GENERAL SUPPORTIVE CARE
• Arterial catheter should be placed as soon as possible in patients requiring vasopressor support. • In septic shock if adequate fluids and vasopressor therapy can restore hemodynamic stability hydrocortisone is not suggested. • When two consecutive blood glucose levels are >180 mg/Dl insulin dosing should be initiated as per a protocol approach • In patients with sepsis and acute kidney injury continuous or intermittent renal replacement therapy should be used[12]. • If there is no contraindication, pharmacological prophylaxis against venous thromboembolism should be initiated'.

Source: Surviving Sepsis Campaign: International guidelines for management of sepsis and septic shock: 2016. Crit Care Med 45:486, 2017.

Forming a probable diagnosis, acquiring samples for culture, starting empirical antibiotic medication, and attaining source control are the first steps in managing an infection[43,61]. Source control is necessary for more than 30% of sepsis patients, primarily for infections of the abdomen, urinary tract, and soft tissues[33]. Although there is disagreement over the best time to intervene, patients with source control have a lower mortality rate than those without delaying antibiotics could be fatal[41,44]. The likelihood of an in-hospital death among septic patients increase by 37% for every hour of delay[37]. Therefore, within 1 hour after the diagnosis of sepsis or septic shock, adequate broad-spectrum antibiotics should be administered, according to worldwide clinical practise recommendations[15]. The best option for empirical therapy relies on the suspected infection site, where the illness first appeared (for example, in the community, a nursing home, or a hospital), the patient's medical history, and regional patterns of microbial susceptibility[33].

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Table6: Initial Antimicrobial Therapy for Severe Sepsis Obvious Source in Adults with Normal Renal Function

CLINICAL CONDITION	ANTIMICROBIAL REGIMENS
Septic shock (Immunocompetent adult)	The many acceptable regimens include (1) piperacillin,tazobactam combination of 4.5g every 6th hourly ((2) cefepime 2g every 8th houry or (3)meropenem 1g every 8th hourly or imipenem-cilastatin -0.5 g every 6th hourly. If the patient is allergic to β-lactam antibiotics, (1) Aztreonam 2g -6th hourly or (2) Ciprofloxacin -400 mg ,12th hourly or Levofloxacin 750mg for every 24hours can be used. Vancomycin (loading dose of 25–30 mg/kg, then 15–20 mg/kg every 12th hourly can be added to each of the above regimens[2].
Neutropenia (<500 neutrophils/μL)	Regimens include (1) cefepime every 8th hourly, (2) meropenem-1gm for every 8hours or imipenem-cilastatin -0.5 g 6th hourly) or doripenem-500 mg 8th hourly), or (3) piperacillin-tazobactam -3.375 g for every for hours can be used. If the patient has a suspected central line–associated bloodstream infection, severe mucositis, skin/soft tissue infection, or hypotension , vancomycin to be added as above[2,12].
Splenectomy	Ceftriaxone -2 g for every 24h, or—in meningitis—2 g for every 12hours. Vancomycin (as above) to be added if the local prevalence of cephalosporin resistant pneumococci are high. Levofloxacin-750mg every 24hourly or moxifloxacin (400 mg for every 24hours plus vancomycin (as above) to be used if the patient is allergic to β-lactam antibiotics[2,12].

If the initial level is less than 2 mmol/L, serum lactate levels should be reassessed. Cardiorespiratory resuscitation and the reduction of the immediate risks from uncontrolled infection are further components of the management bundle[33]. An organised strategy is needed for early resuscitation, including the provision of IV fluids, the use of vasopressors, oxygen therapy, and mechanical breathing to support damaged organs[63]. The precise elements necessary to improve resuscitation, such as the choice and quantity of fluid, the proper kind and level of hemodynamic monitoring, and the function of adjunctive vasoactive drugs, are all still up for

debate[39].Evidence suggests that clinical assessments of organ perfusion and management without a protocol are less likely to improve survival than protocolized therapy bundles[12,33].

"Arguably the first protocol-based sepsis treatment strategy—early, goal-directed therapy (EGDT)—included an aggressive resuscitation protocol with specific hemodynamic thresholds for fluid administration, blood transfusion, and use of inotropes[45,51]. Given the many controversial features of this older single-center trial, subsequent trials, including ProCESS, ARISE, and ProMISe, compared protocol-based standard care with protocol- based EGDT and usual care. Each found that EGDT offered no mortality benefit in early septic shock but did increase treatment intensity and cost[33]. Multiple subsequent meta-analyses of these trials confirmed that EGDT offers no mortality benefit while increasing health care utilization and ICU admission in well-resourced countries. Modified versions of EGDT were also tested in lower resourced settings, with no change in outcome[53]."

Thus, EGDT is no longer recommended as the primary strategy for early resuscitation in septic shock[12].

SUBSEQUENT TREATMENT OF SEPSIS AND SEPTIC SHOCK

After the first resuscitation, the emphasis is on monitoring and supporting organ function, preventing complications, and, when practical, de-escalating the level of care[52].

Monitoring

The primary physiologic symptoms of sepsis and septic shock may be clarified by hemodynamic monitoring devices. These monitoring devices' clinical use may be attributed to the device itself, the algorithm connected to the device, or the algorithm's static or dynamic aim[55,51]. Invasive devices like the pulmonary artery catheter (PAC), also known as the continuous ScvO2 catheter, were once a common part of treating shock victims[15,54]. Among other measures, the PAC can

calculate cardiac output, monitor mixed venous oxygen saturation, and calculate shock aetiology to help determine how patients would fare[15].

The PAC is no longer advised for routine usage because a recent Cochrane evaluation of 2923 general-ICU patients (among whom the percentage of patients in shock was not reported) found no difference in mortality with or without PAC administration[57]. Instead, a number of noninvasive monitoring techniques, such as focused echocardiography and arterial pulse contour analysis (PCA), can offer continuous estimates of variables like cardiac output, beat-to-beat stroke volume, and pulse pressure change[15]. These tools can help determine a patient's volume responsiveness, along with passive leg-raise manoeuvres and inferior vena cava collapsibility on ultrasound, but they need a number of different clinical conditions to be met (such as the patient being on mechanical ventilation or having sinus rhythm), and more proof from larger randomised trials on their effectiveness in day-to-day management is required[15,56].

PREVENTION

Given the continually high mortality risk associated with sepsis and septic shock, prophylaxis may be the most effective strategy for lowering unnecessary fatalities, yet sepsis prevention is difficult[60]. Sepsis occurrences continue to increase as a result of the ageing population, excessive use of unsuitable antibiotics, an increase in the occurrence of resistant microorganisms, and the use of indwelling devices and catheters[33]. Avoiding the overuse of antibiotics, restricting the use of catheters and indwelling devices, eliminating needless immune suppression, and improving adherence to infection control protocols at hospitals and clinics can all help to lower the number of infections[15,12].

Even when sepsis symptoms are incipient, such pragmatic work could be supplemented with study into the initial pathophysiology of infection to enable earlier treatment. In addition, the discipline of implementation science can help determine how to improve the uptake of infection control in high-risk environments and might direct appropriate care[12].

AIMS AND OBJECTIVES OF STUDY

To evaluate serum lactate levels, total leucocyte count and neutrophil to lymphocyte ratioas a predictor of puerperal sepsis after vaginal delivery.

REVIEW OF LITERATURE

1) **Alveera et al (2014)** [5] studied individuals with obstetric-related sepsis to see if serum lactate could be utilised as a reliable and distinct indicator of severe sepsis. The study comprised 40 patients who had two or more signs and symptoms of infection and a suspected infection. These individuals got a baseline serum lactate test, followed by additional testing over time, after sepsis was diagnosed. Of the 40 sepsis patients they examined, they found that not all had high serum lactate levels. Throughout the trial, it became clear that patients with an initial lactate level greater than 2.2 mmol/l needed more acute care and close monitoring while they were in the hospital.

2) **Albright et al (2015)** [18] It is a retrospective cohort study in which pregnant and postpartum women with signs of sepsis is the subject of this one. According to the findings of this study, pregnant women with elevated lactic acid are more likely to have adverse maternal outcomes from suspected sepsis.

3) **Nissim Arbib et al (2015)** [10] conducted a retrospective cohort study on 12 079 healthy women who gave birth to a singleton term foetus and had an easy labor, delivery, and puerperium. Every woman gave birth at the same tertiary institution. There is an obvious expansion in the WBC after conveyance with tremendous contrasts as per method of conveyance. Leucocytosis after childbirth can be influenced by a number of factors, including: age, parity, gestational age, delivery method, type of anesthesia, caesarean delivery timing in relation to labor onset, and extent of perineal trauma

4) Orr, K et al (2016) [1] study was done to figure out what normal PCT, lactate, and CRP levels are for normal parturition in low-risk women. Prospective data were gathered from a group of low-risk women who gave birth naturally at term. The study came to the conclusion that an increase in maternal lactate and CRP is linked to normal parturition. PCT is a better

marker for excluding infection during pregnancy, so it should be incorporated into obstetric practice so that intrapartum sepsis can be detected quickly.

5) **Vaidyanathan Gowri et al (2016)** [9] studied a total of 250 women, 150 of whom had caesarean births and 100 of whom had vaginal births. Women who were febrile and women who were afebrile were compared based on their white cell count and neutrophil count. Disease was affirmed by assessing the lab results. Women with and without an infection were compared for their WBC. Puerperal fever affected 4% of the caesarean delivery group and 6% of the vaginal delivery group. In both the caesarean and vaginal delivery groups, only two women tested positive for infection on culture. The white cell count and neutrophil were essentially more in febrile ladies following vaginal conveyance yet NOT in the cesarean conveyance bunch. The white cell count and neutrophil count of the women with fever in the culture-positive group and the culture-negative group were not significantly different.

6) Xuan Liu et al (2016) [11]**.** In this prospective, observational study cohort, eligibility was checked on 333 adult sepsis patients. On entry into the intensive care unit, severity scores and leukocyte counts were recorded. They discovered that patients who passed away had significantly higher median NLR levels than those who survived. Increased NLR levels were associated with adverse outcomes, regardless of the influence of potential confounders, according to the modest predictive power of NLR. In sepsis patients, a negative clinical prognosis was independently linked to elevated NLR levels.

7) **Melissa E et al (2019)** [7] The purpose of this article is to conduct a systematic literature review to determine the normal range of lactic acid in pregnant women in good health. The purpose of the study was to locate studies that reported maternal lactic acid in pregnant women in good health. Even though the pooled ranges were less than 4 mmol/L, many individual studies reported ranges greater than 4 mmol/L during labor, according to this study.

8) **Agarwal R et al (2019)** [8]. done a study on the level of lactic acid in one index, which is

being looked into for its role in pregnancy-associated sepsis (PAS) right now. They wanted to use lactic acid levels to figure out how bad PAS was. They estimated the admission levels of lactic acid in all pregnant, post-abortive, and postpartum women with clinical sepsis according to systemic inflammatory response syndrome criteria (up to 6 weeks). They came to the conclusion that a lactic acid level greater than 4 mmol/lit was sufficiently specific for PAS severity and culture positivity. A greater number of organ failures were correlated with serum lactic acid levels greater than 3 mmol/lit.

9) **Penzy Goyal et al (2020)** [(6)] studied to find a link between the prognosis of the mother and the serial monitoring of lactic acid in patients with pregnancy-associated sepsis (PAS). After admission, lactic acid levels were measured at 0, 24, and 48 hours, and lactate clearance was calculated. They found the mean worth of lactic corrosive was altogether higher in the Emergency unit bunch than the Non-ICU bunch at 0, 24, and 48 h.

The lactic corrosive in the survivor bunch was altogether lower when contrasted with the mortality bunch. For predicting admission to the ICU, a cutoff of 3.8 mmol/l has a sensitivity of 84% and a specificity of 68%. In patients who survived, the mean lactate clearance was 46%, and in patients who died, it was 22.5 percent. There was no mortality when lactate clearance was 60%, but when lactic acid levels rose by 100%, all of them died.

10) **F.0.DareMD et al(1998)[(16)],** carried out research on Puerperal Sepsis: a postpartum problem that can be avoided. The case notes of the patients as well as information regarding: age and parity The booking status, the pregnancy complications, the hemoglobin level at admission, the history of labor and delivery, the organisms found in the genital tract, and the outcome for the mother were all extracted and analyzed. That's what they reasoned, puerperal sepsis was viewed as related with recently detailed clinical gamble factors like primiparity, pallor, PROM, delayed impeded work, different vaginal assessments and conveyance outside wellbeing offices.

11) **CD Acosta et al(2012)**[29], led a concentrate on Maternal sepsis: a population-based case–control study in Scotland. Their study's objective was to describe the risk of maternal sepsis that is connected to obesity and other risk factors that have not been extensively studied, such as operative vaginal delivery. Obese women had twice the risk of uncomplicated sepsis (OR 2.12; control for delivery method, demographic, and clinical factors). 95% CI, 1.14–3.89) when compared to women whose weight is normal. Their study came to the conclusion that obesity, having an operative vaginal delivery, and being younger than 25 years old are significant risk factors for sepsis.

12) **Meharun-Nissa Khaskheli et al(2015)**[49], carried out research on: At a tertiary healthcare facility, puerperal sepsis risk factors and complications. The majority of the women in their study, 84 (65.11 percent), were over the age of 31, multiparous 101 (78.29 percent), and unbooked 98 (75.96 percent). The absence of membranes in 108 (83.72 percent) of the women, their mismanagement, and the fact that 95 (73.64 percent) of the referred cases are being delivered in this hospital are common risk factors. Septicemia was seen in 35 cases (27.13 percent), disseminated intravascular coagulation was seen in 23 cases (17.82 percent), and 11 women (8.52%) died. Their study came to the conclusion that anemia was a common risk factor. Poor sanitation and sterilization caused serious health problems like septicemia and disseminated intravascular coagulation, which ultimately led to death.

13) **Willa Antoniette B et al(2005)**[19], conducted research into the clinical correlation of hematological parameters of the newborn and the mother as predictors of neonatal sepsis. Their study had as its goal the development of a scoring system for the diagnosis of neonatal sepsis based on the clinical manifestations and hematological parameters of the mother and child, both individually and collectively. Their study came to the conclusion that a scoring system for predicting neonatal sepsis could be created by combining the hematological parameters of the mother and the neonate's clinical signs.

14) **Naser Gharebaghi et al(2019)[20],** conducted research on the neutrophil-to-lymphocyte ratio in Intensive Care Unit patients with gram-negative sepsis. Patients admitted to the general intensive care unit with gram-negative sepsis were included in their study. characteristics of the population; Score in APACHE II; duration of mechanical ventilation and stay in the intensive care unit; On the first, second, and third days of hospitalization, the mortality rate and the average neutrophil and lymphocyte count were examined. Their study came to the conclusion that in addition to other diagnostic procedures, blood cell analysis and the N/L ratio can be used as a predictor for the severity of gram-negative sepsis.

15) **Yunlong Liu et al(2019)[21],** directed a concentrate on Neutrophil-lymphocyte proportion and plasma lactate foresee 28-day mortality in patients with sepsis. The neutrophil-to-lymphocyte ratio (NLR) and plasma lactate as prognostic indicators for sepsis patients were the focus of their investigation. Their study came to the conclusion that plasma lactate and the neutrophil-to-lymphocyte ratio were linked to poor outcomes and predicted mortality in sepsis patients (N/L Ratio article).

16) **Agata Buonacera et al(2022[23]),** conducted research on the ratio of neutrophils to lymphocytes: An Arising Marker of the Connections between the Insusceptible Framework and Sicknesses. Their research aims to provide an overview of this biomarker's main uses, focusing on its pathophysiology and molecular basis as a reliable indicator of inflammatory status and adaptive immunity. According to their findings, NLR is a low-cost and simple-to-obtain biomarker that reflects the equilibrium between two aspects of the immune system: 1) inflammation, both acute and chronic, and 2) adaptive immunity. Changes in NLR over time are a sign of a dysfunctional immune system, even though there are no established cut-off values.

17) **Are Naess et al(2016)[24],** conducted research on the role that ratios of neutrophils to lymphocytes and monocytes to lymphocytes play in the diagnosis of bacterial infection in fevered patients. Their study came to the conclusion that NLR is a better diagnostic tool for septicemia detection than other more common diagnostic blood tests. NLR and MLR may be

useful for diagnosing bacterial infections in fever-stricken patients admitted to hospitals (n/l ratio articles).

18) **Elizabeth Morris et al(2016)**[25] conducted a study on lactate testing for sepsis at the point of care when a patient presents to health care: a thorough analysis of the outcomes for patients. In patients who presented with acute infections, the purpose of their study was to investigate the effect of using point-of-care lactate on mortality and other clinical outcomes. It was deduced in their survey that there is no great proof to help the utilization of point-of-care lactate in local area settings. However, the evidence suggests that point-of-care lactate testing is associated with a decrease in in-hospital mortality. To evaluate this potential diagnostic technology, RCT evidence from community settings is required.

19) **Bigna S Buddeberg et al(2015)**[26] conducted a research project on Puerperal sepsis in the modern era: advancement, new obstacles, and the global situation. Their study came to the conclusion that caesarean sections come with an increased risk of infection, particularly given that caesarean section rates are rising worldwide. The HIV pandemic poses a threat to the developing world because of its connection to high maternal mortality in South-East Asia and sub-Saharan Africa. Finally, just like with any other patient population, obstetric patients will be at risk from newly emerging antibiotic-resistant superbugs. It is vital to recall that the changed physiology of pregnancy and the puerperium can cloud the signs and side effects of sepsis. Current guidelines recommend starting empiric antibiotic therapy within one hour if sepsis is suspected.

20. **Joseph Ngonzi et al(2016)**[29] at a Tertiary University Teaching Hospital in Uganda, conducted a study titled Puerperal sepsis, the leading cause of maternal death. Their research revealed that while indirect causes accounted for 22.3 percent of deaths, direct causes accounted for 77.7 percent. Puerperal sepsis was the leading cause of maternal death (30.9 percent),

followed by obstetric hemorrhage (21.6 percent), hypertensive disorders during pregnancy (14.4 percent), and abortion complications (10.8 percent). Jungle fever was the commonest aberrant reason for mortality representing 8.92 %.It was deduced in their review that,most maternal passings happen among moms from country regions, uninformed, HIV positive, unbooked moms (absence of antenatal consideration), alluded moms in basic circumstances and moms postponing to look for medical care.

21. **Sheeba Marwah et al (2017)[30]** a study with the title: A pervasive threat is severe puerperal sepsis. Their research sought to ascertain the prevalence, risk factors, and mortality rates of women with puerperal sepsis who presented to a tertiary care health facility in India. Anaemia, prolonged labor, untrained delivery, and unsafe abortion were identified as the primary identifiable risk factors in their study. Seventy percent of women succumbed to their illness, mostly due to multiorgan failure, requiring surgical treatment. Their study came to the conclusion that a significant contributing factor is a lack of safe and sanitary methods for carrying out abortion and delivery.

22. **Wafaa Mostafa Ahmed Gamel et al(2020)[31]** a study titled "The Impact of the Puerperal Sepsis Self-Care Nursing Guideline on Women's Knowledge and Practices" was carried out. Their study concluded that primiparous postpartum mothers who received and adhered to the guidelines regarding puerperal sepsis and its prevention had a higher knowledge and practices score in the post-test of the intervention program than in the pre-test (P0.01), demonstrating the effectiveness of puerperal sepsis self-care nursing guidelines in increasing the knowledge score of primiparous postpartum mothers regarding the prevention of puerperal sepsis.

23. **Shamshad et al(2010)[32]** conducted a research project titled “Puerperal sepsis — Still a major parturient risk”. Their research revealed that 34.4% of postnatal complications and 1.7% of all obstetrical admissions were caused by puerperal sepsis. It was observed frequently in patients between the ages of 15 and 25; they were of lower parity; had low socioeconomic status;

were uneducated; delivered at home; prolonged labor; prolonged membrane rupture for 48 to 72 hours; and were delivered by untrained birth attendants. Endotoxic shock, disseminated intravascular coagulation, foul-smelling discharge, retained product of conception, peritonitis, septicemia, pelvic abscess, and puerperal sepsis morbidity were the most common complications.

MATERIALS AND METHODS

SOURCE OF DATA

Patients delivered at B.L.D.E (DEEMED TO BE UNIVERSITY) SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE will be taken into study. The patients will be informed about the study in all aspects and informed consent will be obtained in accordance with declaration of Helsinki.

Health care setup - Tertiary care hospital

Sample size – 210+40 more cases were studied with a 20% drop out rate with 125 in each group.

Type of study – Observational study

Study Period: JANUARY 2021 TO APRIL 2022

METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA

All the patients with gestational age >24weeks who delivered vaginally were studied.They are divided in to two groups, low risk group and high-risk group**.**

- **LOW RISK GROUP**

1) Spontaneous onset of labour
2) Delivery within 24 hrs of onset of labour
3) Cephalic presentation
4) 1^{st} and 2^{nd} degree perineal tear

- **HIGH RISK GROUP**
  - **1)** Induced labour
  - **2)** Prolonged labour more than 24hrs
  - **3)** PROM
  - **4)** Preterm labour
  - **5)** Instrumental delivery

EXCLUSION CRITERIA

- Pregnant women with PIH and GDM.
- Pregnant women with already established sepsis.
- The patient undergoing caesarean section.
- Pregnant women other than cephalic presentation
- Pregnant women with pre-existing medical disorders
- Pregnant women on drugs like metformin, acetaminophen, linezolid, beta two agonists, and other medicines which causes an elevation in serum lactate levels

Sample size calculation

With anticipated Sensitivity and specificity of Serum lactate as an early predictor of puerperal sepsis 53.8% and 100% respectively, considering the prevalence of sepsis 64% [(5)], at a precision of 1% and 98% confidence, the required sample size is 210.

210+40 more cases will be studied with a 20% dropout rate with 125 in each group.

The formula used is—

$$N=\frac{Z^2(1-p)}{\Delta^2}$$

N will be (a+c) if we use sensitivity as pN= (a+c)/Prevalence **Statistical analysis**

The data obtained will be entered in a Microsoft Excel sheet, and statistical analysiswill be performed using statistical package for the social sciences (Version 20).

Results will be presented as Mean (Median) ±SD, counts and percentages anddiagrams.

- For normally distributed continuous variables between the two groups will be compared using

Independent t-test For not normally distributed variables Mann Whitney U test will used.

- Categorical variables will be compared using the Chi-square test.
- Sensitivity, Specificity, PPV and NPV will be used to find the diagnostic tests.
- P<0.05 will be considered statistically significant. All statistical tests will perform two-tailed.

METHODOLOGY

This is a clinical observational study.

All the patients who fulfill inclusion criteria were studied. Consents were taken in accordance with the declaration of Helsinki once the patient is admitted.

The patient who delivered vaginally were tested for serum lactate levels, Total Leucocyte Count and neutrophils to lymphocyte ratio from 48 to 72hrs after vaginal delivery.

After vaginal delivery, patients were observed for 72hrs for signs and symptoms of sepsis land discharged on 3rd day if no symptoms. Patients were followed up on 7th day personally or through a phone call for assessment of symptoms of puerperal sepsis like fever, foul smelling vaginal discharge and any other.

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OBSERVATION:

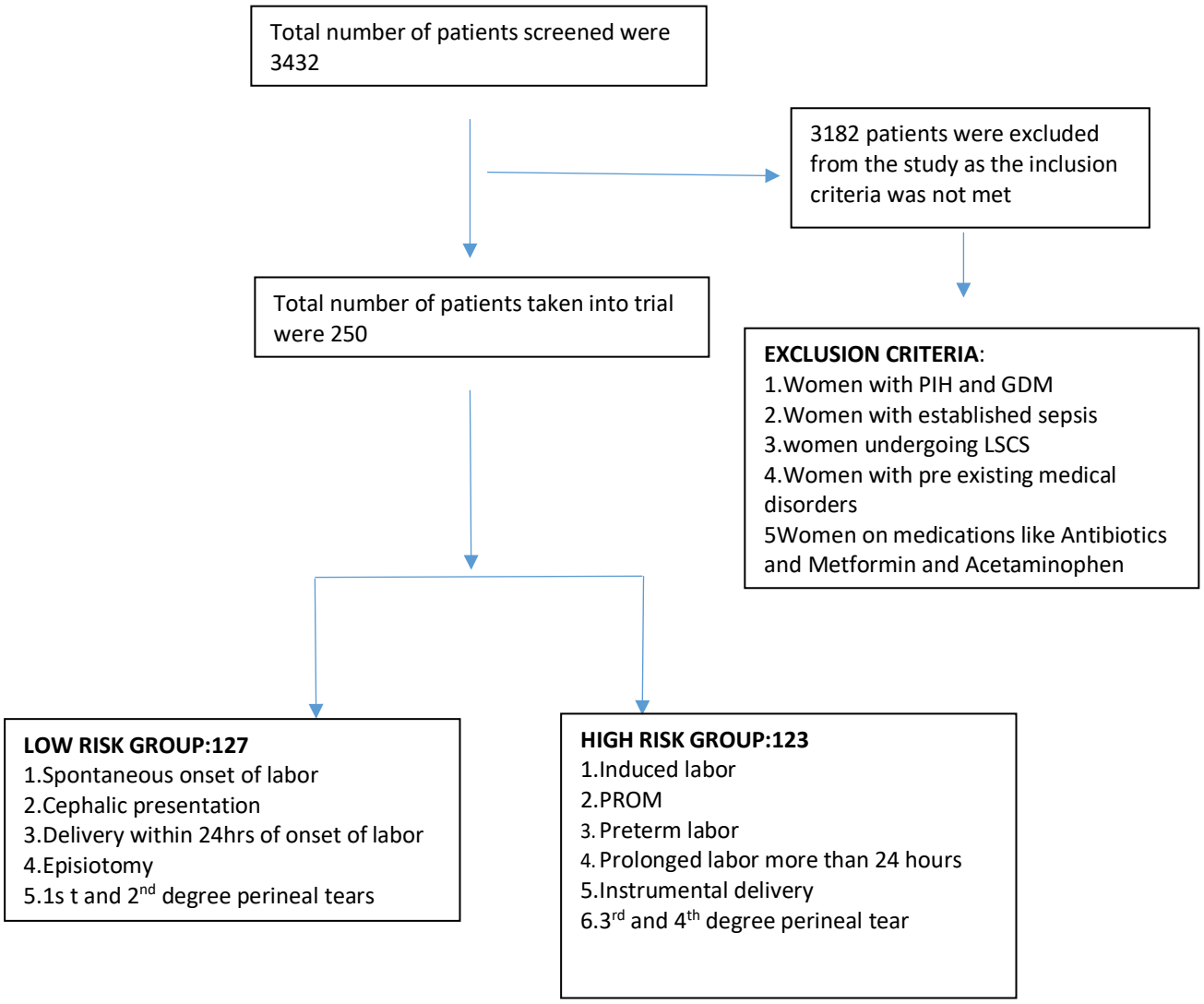
Outcomes: 1) To know the cut off levels of serum lactate, total leucocyte count and neutrophil to lymphocyte ratio as predictor of puerperal sepsis.

2) To know the difference in the predictors between low risk and high-risk cases.

Investigations:

- Serum lactate levels
- Total Leucocyte Count
- Neutrophil to Lymphocyte Ratio

RESULTS:



A) DEMOGRAPHIC DATA

a) AGE GROUP DISTRIBUTION

LOW RISK GROUP

Table 7: Distribution of age in low risk and high risk group

AGE	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
<20	5(3.9)	9(7%)	0.23
20-29	114(89.8)	105(85.4%)	
30 and above	8(6.3%)	9(7.65%)	
MEAN	24.56	24.02	

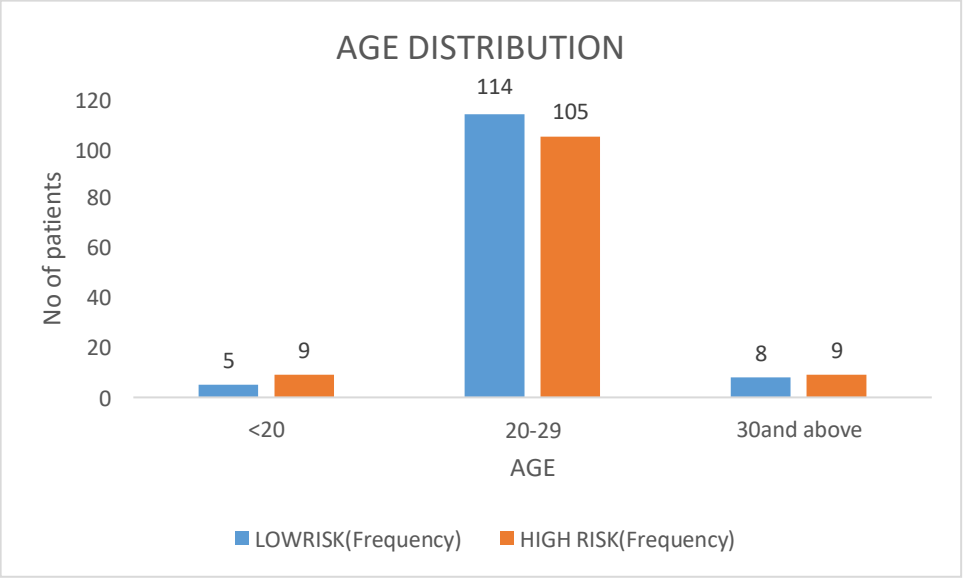
The above table show the age distribution of the patients in low risk and high-risk groups. In low-risk group, 5(3.9%) patients are <20 years of age,114(98.8%) of them were 20-29 years of age (89.8%) and 8(6.3%) of them belong to 30 years and above.

In high-risk group, 9(7%) of the patients were<20 years old age, 105(85.4%) were 20-29 years of age and 9(7.65%) of them are 30 years and above. Majority of the patients in both the groups were between 20 -29 years of age.

The mean age in both the group was 24.56 in low-risk group and 24.02 in high-risk group.

The p value for age is 0.23 which is insignificant.

Graph 3: Distribution of age among low risk and high risk group



b) GESTATIONAL AGE DISTRIBUTION

Table 8: Distribution of gestational age in low risk and high risk group

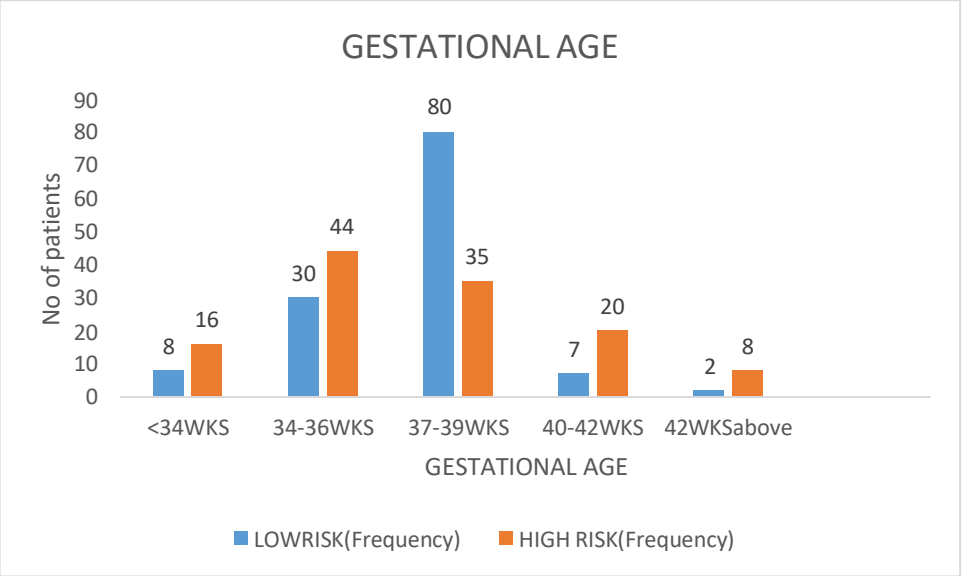
GESTATIONAL AGE	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
<34 WEEKS	8(6.2%)	16(13%)	0.38
34-36 WEEKS	30(23.6%)	44(35.77%)	
37-39 WEEKS	80(62.9)	35(24.27%)	
40-42 WEEKS	7(5.5%)	20(16.26)	
>42 WEEKS	2(1.57%)	8(6.5%)	
MEAN	38.46	37.02	

The above table shows the distribution of the gestational age in both the groups in comparison. In low-risk group 8(6.2%) were < 34 weeks of period of gestation, 30(23.6%) of them were 34-36 weeks of gestation,80(62.9%) of them were 37-39 weeks of gestation, 7(5.5%) of them were of 40-42 weeks of gestation and 2(1.57%) were more than 42 weeks of gestation.

In high risk group,16(13%) of them were <34weeks,44(35.77%) were 34-36 weeks of gestation,35(24.27%) were belonging to 37-39weeks of gestation and 20(16.26%) of them were

40-42 weeks of gestation,8(6.5%) of them were> 42 weeks of gestation. The mean gestational age for low-risk group is 38.46 and for high-risk group it is 37.02. In low-risk group majority of the patients were of 37-39 weeks of gestation and in high-risk group it is 34-36weeks of gestation.

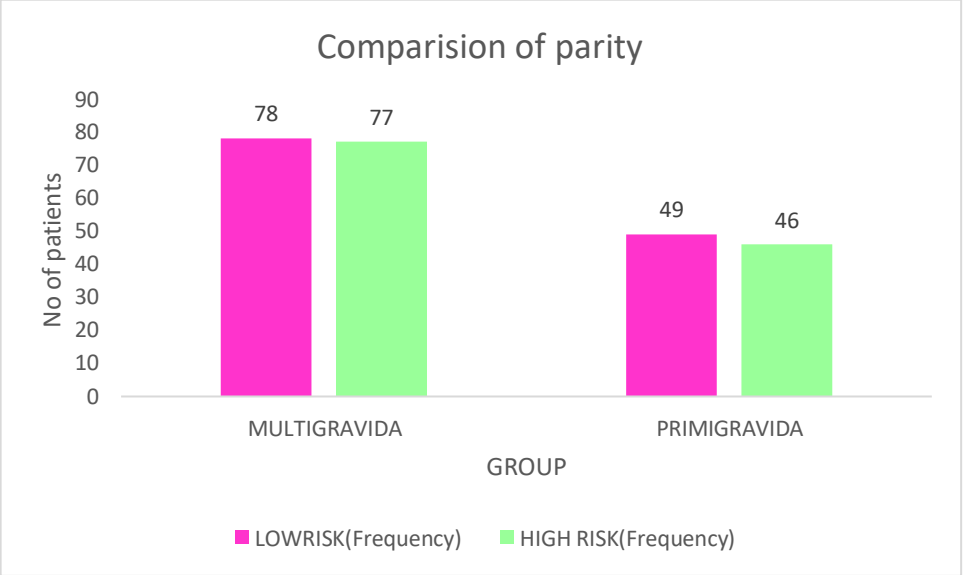
Graph 4: Distribution of gestational age in low risk and high risk group



c) PARITY DISTRIBUTION

Table 9: Distribution of parity in low risk group and high risk group

PARITY	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
MULITGRAVIDA	78(61.4%)	77(62.6%)	0.26
PRIMI GRAVIDA	49(38.6%)	46(37.45)	
TOTAL	127	123	



Graph 5: Distribution of parity among low risk group and high risk group

In low-risk group out of 127 patients 78(61.4%) of them were multigravida and 49(38,6%) of them were primigravida. In high-risk group 77(62.6%) of them were multigravida and 46(37.45%) were primigravida. In both the groups majority of them are multigravida. The p value is 0.26 which is not significant.

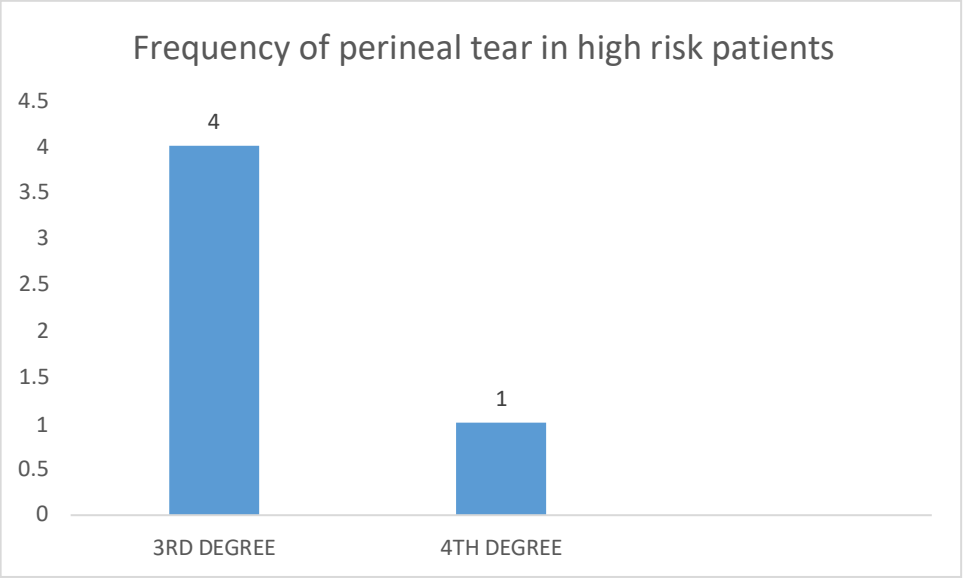
d) FREQUENCY OF PERINEAL TEAR DEGREE IN HIGH RISK GROUP

Table 10

DEGREE OF TEAR	3^{RD} DEGREE	4^{TH} DEGREE
FREQUENCY	4	1

The above table shows the frequency of perineal degree tears in high risk group. A total of 5 pateints had perineal tears in high risk group of which 4 of them had 3^{rd} degree perineal tear and 1 of them had 4^{th} degree perineal tear.

Graph 6



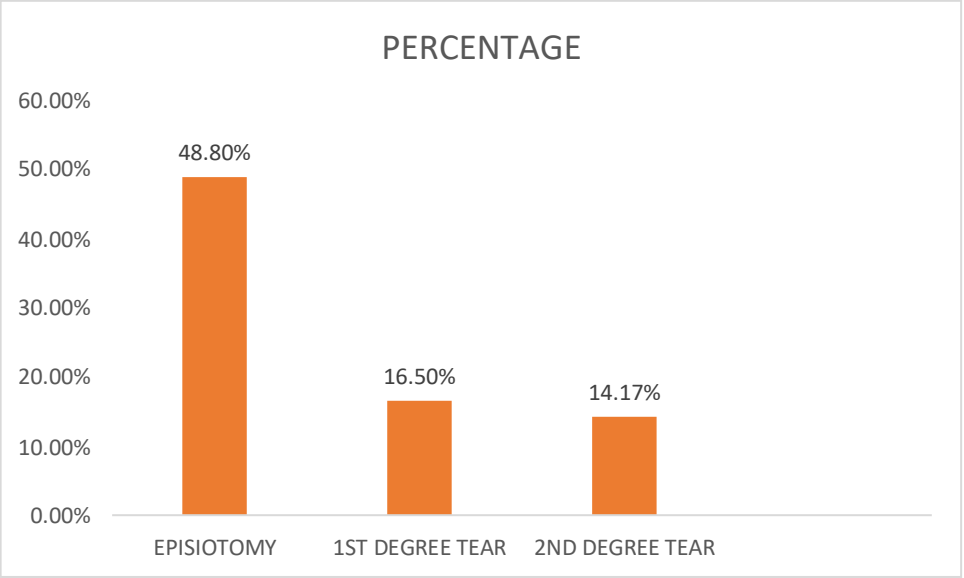
e) EPISIOTOMY/ PERINEAL TEAR/EPISIOTOMY IN LOW-RISK GROUP

Table 11: Frequency of Episiotomy/ perineal tear in low risk group

EPISIOTOMY/PERINEAL TEAR	FREQUNECY	PERCENTAGE
EPISIOTOMY	62	48.8%
1ST DEGREE TEAR	21	16.5%
2ND DEGREE TEAR	18	14.17%

The above table depicts the frequency of episiotomy and perineal tear in low risk group.In low risk group 62 patients were given episiotomy and 21 of them had 1st degree perineal tear and 18 of them had 2nd degree perineal tear.

Graph 7



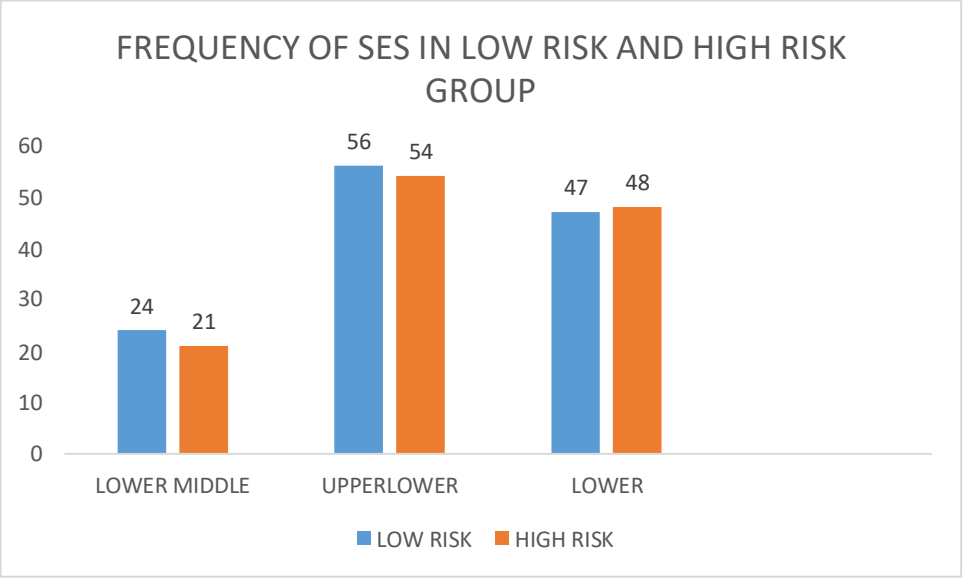
d) COMPARISION OF SOCIOECONOMIC STATUS IN LOW RISK AND HIGH RISK

Table 12

GROUP	LOWER MIDDLE	UPPER LOWER	LOWER
LOW RISK	24(18.8%)	56(44.09%)	47(37%)
HIGH RISK	21(17%)	54(43.9%)	48(39.02%)

The above table depicts the socioeconomic status in both low risk and high risk patients.In low risk group, 24(18.8%) of them belong to lower middle class,56(44.09%) of them belong to upper lower class, 47(37%) of them belong to lower class. In high risk group,21(17%) belong to lower middle class,54(43.9%) of them belong to upper lower class, and 48(39.02%) of them belong to lower class.

Graph 8



e) COMPARISION OF LOCALITY ILOW RISK GROUP AND HIGH RISK GROUP

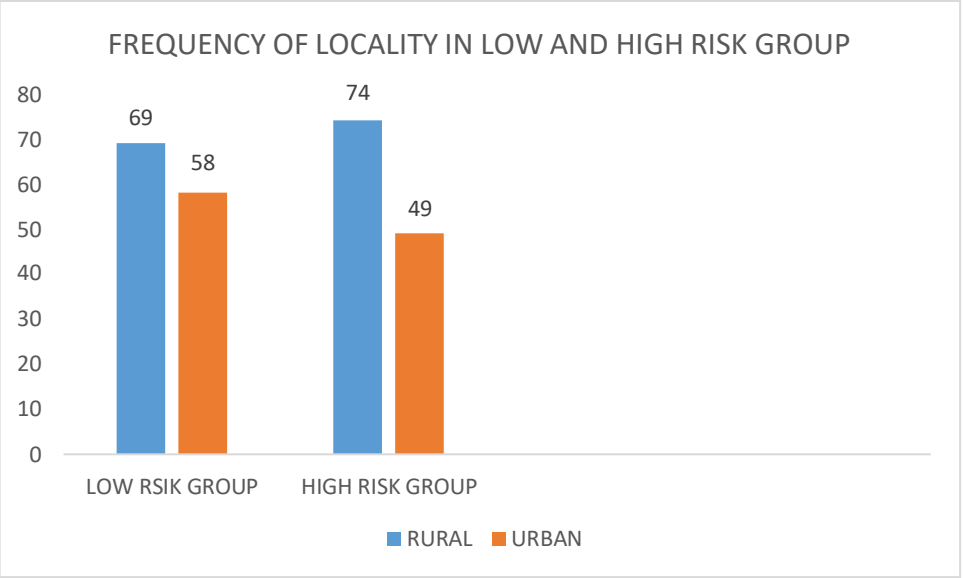
Table 13:

LOCALITY	LOW RISKGROUP	HIGH RISK GROUP
RURAL	69(54%)	74(60%)
URBAN	58(45.6%)	49(39%)

The above table depicts the comparision of locality in low risk and high risk group.

In low risk group,69(54%) of them belong to rural area and 58(45.6%) of them belong to urban area. In high risk group 74(60%) of them belong to rural area and 49(39%) of them belong to urban area.

Graph 9



B) SERUM LACTATE

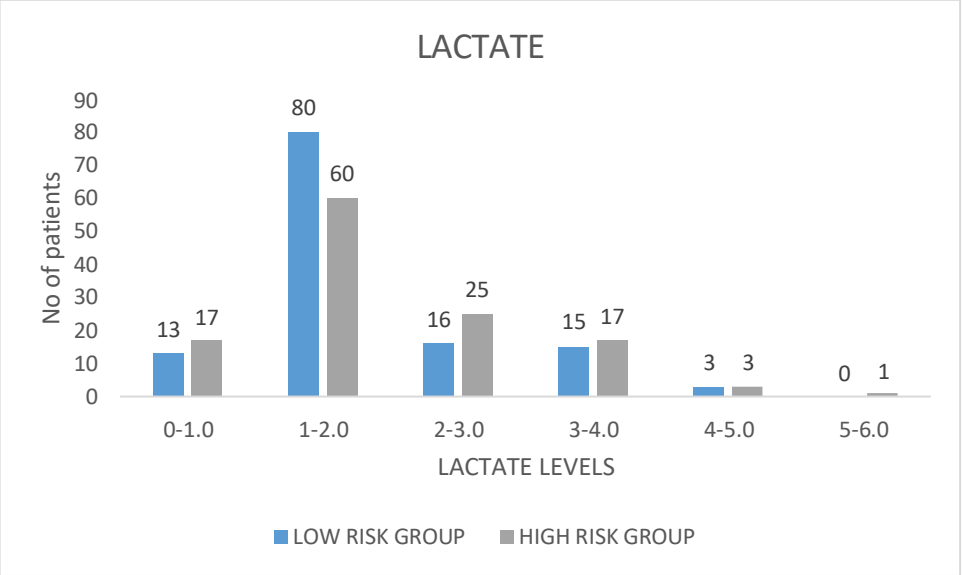
Table 14: Outcome of serum lactate in low risk and high risk group

SERUM LACTATE	LOWRISK GROUP	HIGH RISK GROUP	P VALUE
<2.1	100(78%)	82(66.6%)	0.0320
>=2.1	27(21.2%)	41(33.3%)	
LACTATE (mmol/L)	**LOW RISK GROUP**	**HIGH RISK GROUP**	**P VALUE**
0-1	**13(10.56%)**	**17(13.8)**	
1-2	**80(65%)**	**60(48.7%)**	**0.15**
2-3	**16(12.6%)**	**25(20.3%)**	
3-4	**15(11.8%)**	**17(13.8%)**	
4-5	**3(2.4%)**	**3(2.4%)**	
5-6	**0**	**1(0.8%)**	
MEAN	**1.6**	**1.8**	

The above table depicts the comparison of serum lactate levels in both the groups. In low-risk group 13(10.56%) patients had lactate levels of 0-1mmol/L,80(65%) of them had 1-2mmol/L.16 (12.6%) of them had lactate value of 2-3mmol/L,15(11.8%) of them had 3-4mmol/L,3(2.4%) of them had 4-5mmol/L.

In high-risk group 17(13.8%) of them had lactate levels of 0-1mmol/L,60(48.7%) of them had 1-2mmol/L,25 of them had lactate levels of 2-3mmol/L, 17(13.8%) of them had lactate levels of 3-4mmol/L,3(2.4%) of them had lactate of 4-5mmol/L,1(0.8%) of them had lactate of 5-6mmol/L. In both the groups majority of them had lactate levels of 1-2mmol/L.

Graph 10: Outcome of serum lactate in low risk and high risk group



TOTAL LEUCOCYTE COUNT

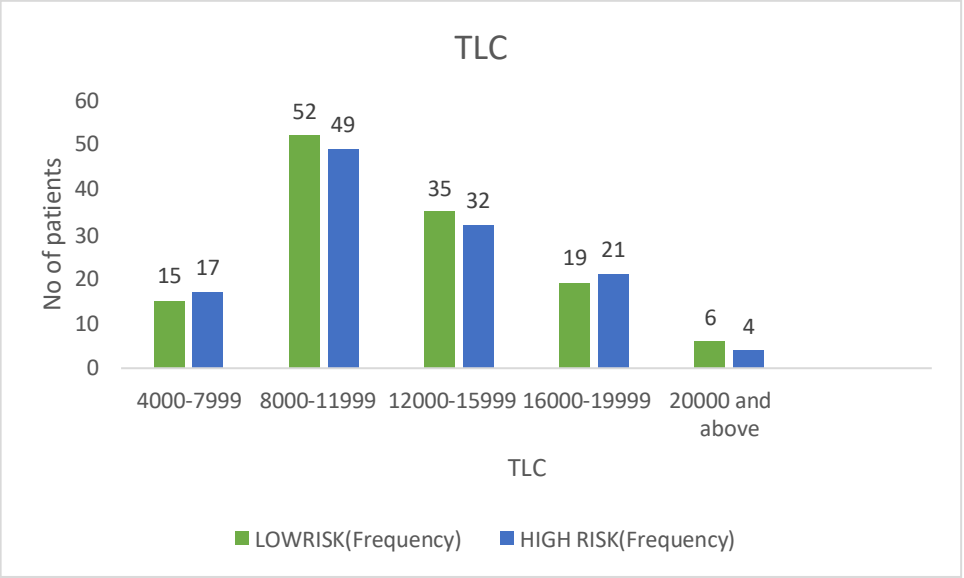
Table 15: Outcome of TLC in low risk and high risk group

TLC	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
<12000	67(52.7%)	66(53.6%)	0.7936
>12000	57(46.3%)	60(47.2%)	

TLC	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
4000-7999	15(11.8%)	17(13.8%)	0.54
8000-11999	52(40.9%)	49(39.8%)	
12000-15999	35(27.6%)	32(26%)	
16000-19999	19(15%)	21(17.1%)	
20000 and above	6(4.7%)	4(3.3%)	
MEAN	12186	11869	

The above table depicts the comparison of Total leucocyte count in both low risk and high-risk groups. In low-risk group 15(11.8%) of them had 4000-7999cell/mm^3,52(40.9%) of them total leucocyte count of 8000-11999, 35(27.6%) of them had 12000-15999, 19(15%) of them had 16000-19999cells/mm^3, 6(4.7%) of them had total leucocyte count of 20000cells/mm^3 and above. In high-risk group 17(13.8%) of them total leucocyte count of 4000-7999cells/mm^3,49(39.8%) had count of 8000-11,999cells/mm^3,32(26%) of them had 12,000-15,999cell/mm^3,21(17.1%) of them 16,000-19,999cells/mm^3,4(3.3%) of them had total count of 20000 and above. The mean total leucocyte count in low-risk group is 12,186 and in high-risk group the mean value is 11,869.20. In both the groups most of the patients had total leucocyte count of 8000-11999.

Graph 11: Outcome of TLC in low risk and high risk group



N/L RATIO COMPARISION IN LOW RISK AND HIGH RISK GORUPS

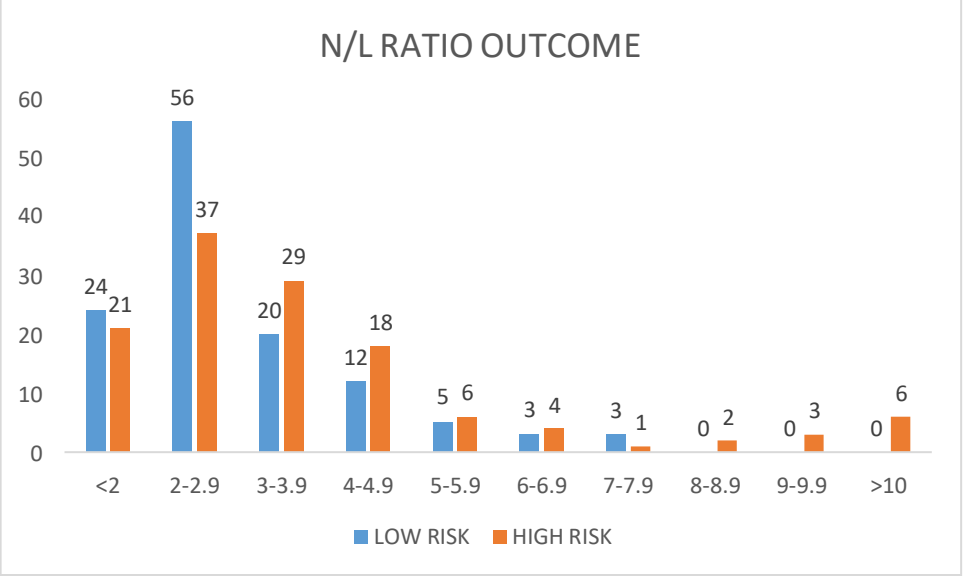
Table 16: Outcome of N/L ration in low risk and high risk group

N/L RATIO	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
<3.6	79(62.2%)	96(78.0%)	0.1801
>3.6	27(21.9%)	48(37.7%)	

N/L RATIO	**LOW RISK GROUP**	**HIGH RISK GROUP**	**P VALUE**
<2	**24(19.5%)**	**21(16.5%)**	**<0.01**
2-2.9	**56(45.5%)**	**37(29.1%)**	
3-3.9	**20(16.3%)**	**29(22.8%)**	
4-4.9	**12(9.8%)**	**18(14.2%)**	
5-5.9	**5(4.1%)**	**6(4.7%)**	
6-6.9	**3(2.4%_**	**4(3.1%)**	
7-7.9	**3(2.4%)**	**1(0.8%)**	
8-8.9	**0**	**2(1.6%)**	
9-9.9	**0**	**3(2.4%)**	
>10	**0**	**6(4.7%)**	
MEAN	**2.69**	**3.6**	

The above table depicts the comparison of Neutrophil to lymphocyte ratio in both the groups. In high risk group 21(16.5%) of them had had N/L ratio of <2,37(29.1%) of them had N/L ratio of 2-2.9, 29(22.8%) of them 3-3.9, 18(14.2%) of them had ratio of 4-4.9,6(4.7%) of them had N/L ratio of 5-5.9%, 4(3.1%) of them a ratio of 6-6.9,1(0.8%) of them had N/L ratio of 7-7.9,2(1.6%) of them had a N/L ratio of 8-8.9, 3(2.4%) of them 9-9.9, 6(4.7%) of them had a ratio of >10.
In low-risk group 24(19.5%) of them had N/L ratio of <2,56(45.5%) of them had ratio of 2-2.9, 20(16.3%) of them had a ratio 3-3.9,12(9.8%) of them had a ratio 4-4.9, 5(4.1%) of them a N/L ratio of 5-5.9, 3(2.4%) of them had a ratio of 6-6.9, 3(2.4%) of them had a ratio of 7-7.9. None of them in the low risk group had a ratio >8. The mean N/L ratio in high-risk group is 3.6 and in low-risk group the mean value is 2.69. Majority of them in both the groups had a ratio of 2-2.9.

Graph 12: Outcome of N/L ratio in low risk and high risk group



B) Primary outcome

1)FEVER

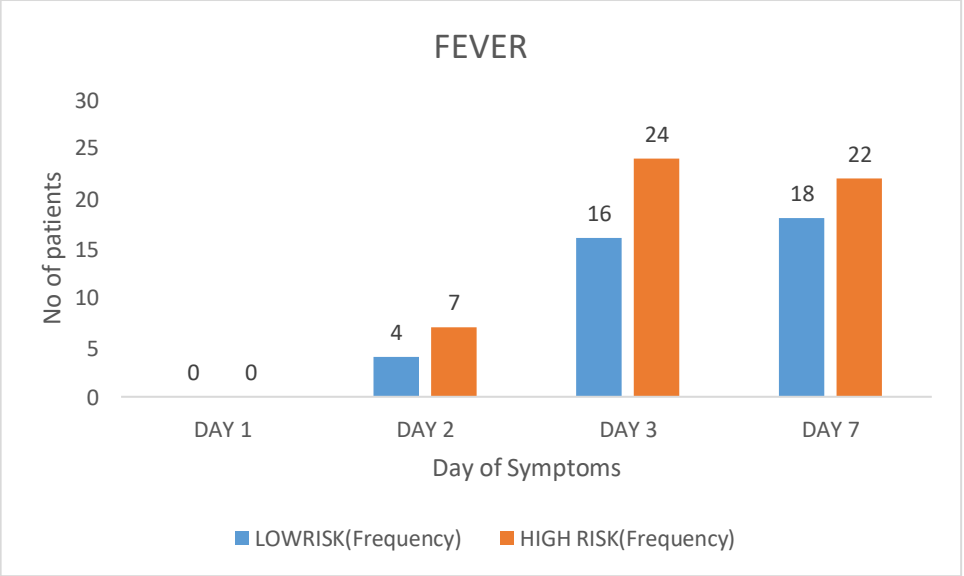
Table 17: Number of patients with fever in low risk and high risk group

FEVER

DAY	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
1	0	0	-
2	4(3.1%0	7(5.7%)	0.32
3	16(12.6%)	24(19.5%)	0.13
7	18(14.2%)	22(17.9%)	0.42
TOTAL	38(29.9%)	53(43.0%)	

The above table depicts the comparison of fever outcome in both the groups.
In low-risk group a total of 38 patients had fever of which none of them had on day 1 of delivery, 4(3.1%) of them had fever on day 2 ,16(12.6%) of them had on day 3 of delivery and 18(14.2%) of them developed fever on day 7. In high-risk group of the 123 patients ,53 of them developed fever. Of them none of them had fever on day 1 of delivery,7(5.7%) developed on day 2 of delivery, 24(19.5%) had fever on day 3 of delivery and 22(17.9%) of them developed fever on day 7. In low-risk group majority of them had fever on day 7 of delivery, whereas in high-risk group majority of them developed on day 3 of delivery.

Graph 13: Frequency of fever in low risk and high risk group



2) FOUL SMELLING LOCHIA

Table 18: Number of patients with foul smelling lochia in low risk and high risk group

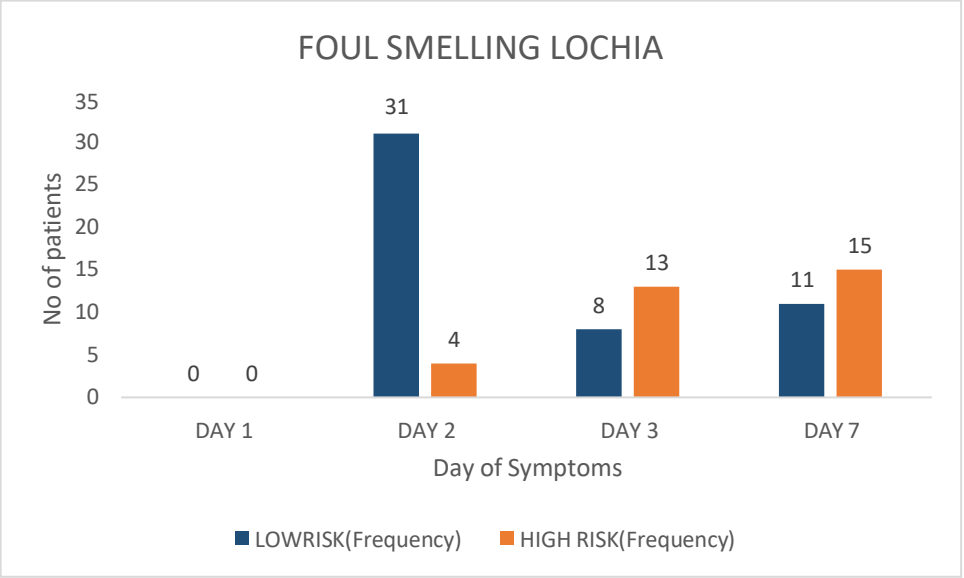
DAY	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
1	0	0	-
2	4(3.3%)	31(24.4%)	0.04
3	8(6.3%)	13(10.6%)	0.22
7	11(8.7%)	15(12.2%)	0.36
TOTAL	23(18.1%)	59(47.9%)	

The above table depicts the comparison rates of foul-smelling lochia in both the groups. In low-risk group out of 127,23 of them had foul smelling lochia. In low-risk group none of them had foul smelling lochia on day 1of delivery,4(3.3%) of them had foul smelling lochia on day 2 of delivery, 8(6.3%) had foul smelling lochia on day 3 of delivery,11(8.7%) had developed foul smelling lochia on day 7.

In high-risk group out of 123 patients,59 of them developed foul smelling lochia of which none of them had foul smelling lochia on day 1 of delivery,31(24.4%) of them had foul smelling

lochia on day 2 of delivery, 13(10.6%) of them had foul smelling lochia on day 3 of delivery and 15(12.2%) of them had on day 7 of delivery. In both the groups majority of them reported foul smelling lochia on day 7 of delivery. The p value for foul smelling lochia on day 2 was 0.04 which is significant.

Graph 14: Frequency of foul smelling lochia in low risk and high risk group



3)EPISIOTOMY WOUND GAPING

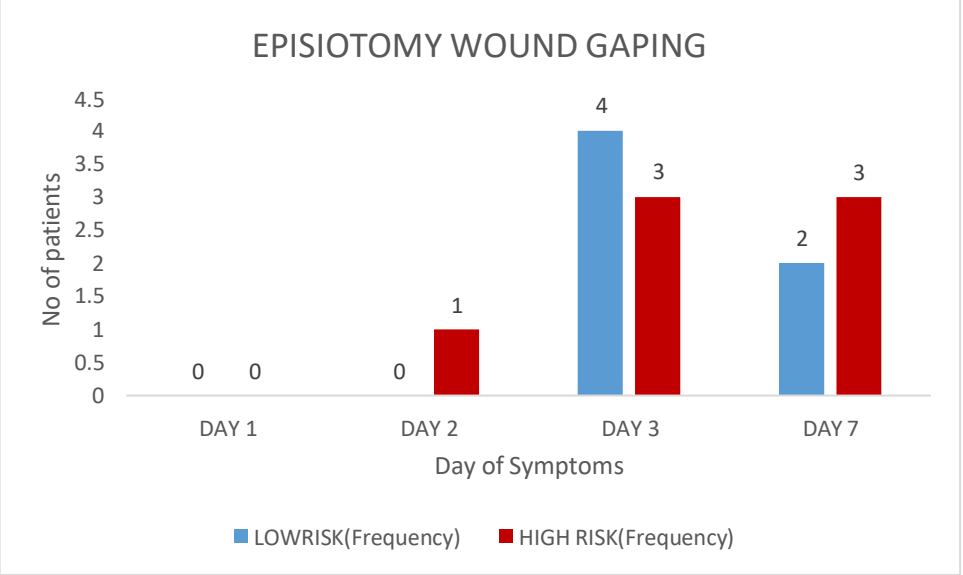
Table 19: Number of patients with episiotomy wound gaping in low risk group and high risk group

DAY	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
1	0	0	-
2	0	1(0.8%)	0.3
3	4(3.2%)	3(2.4%)	0.62
7	2(1.6%)	3(2.4%)	0.72
TOTAL	6(4.7%)	7((5.6%)	

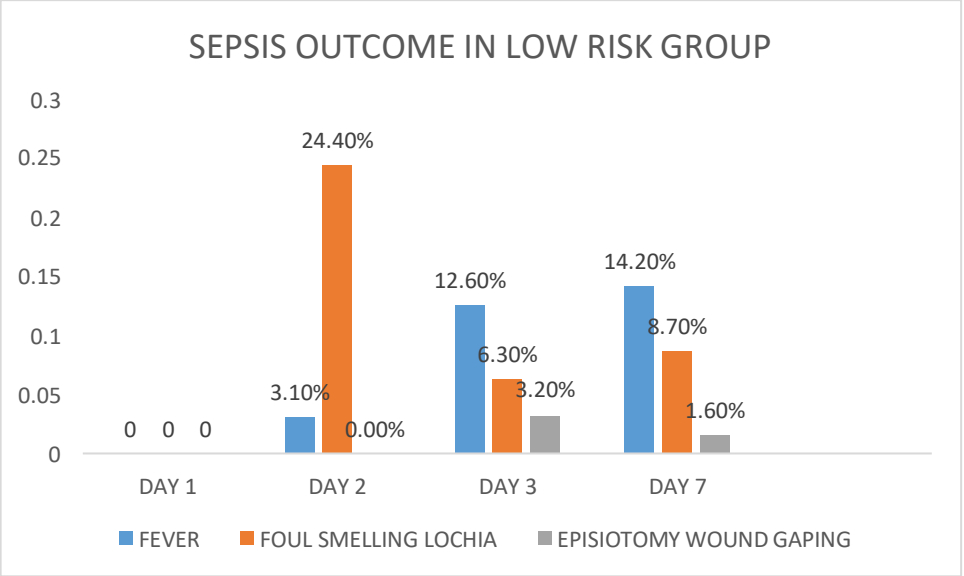
The above table depicts the comparison of episiotomy wound gaping rates in both the groups. In low-risk group, out of 127 patients ,6 (4.7%)of them had episiotomy wound gaping. 4(3.2%) of them developed on day 3 of delivery and 2(1.6%) of them developed episiotomy wound gaping on day 7 of delivery.

In high-risk group, out of 123 patients 7(5.6%) of them had episiotomy wound gaping. 1(0.8%) of them developed on day 1 of delivery. 3(2.4%) of them on day 3 of delivery and another 3(2.4%) of them on day 7 of delivery.

Graph 15: Frequency of episiotomy wound gaping in low risk and high risk group



Graph 16: Outcome of sepsis in low risk group



Graph 17: Outcome of sepsis in high risk patients

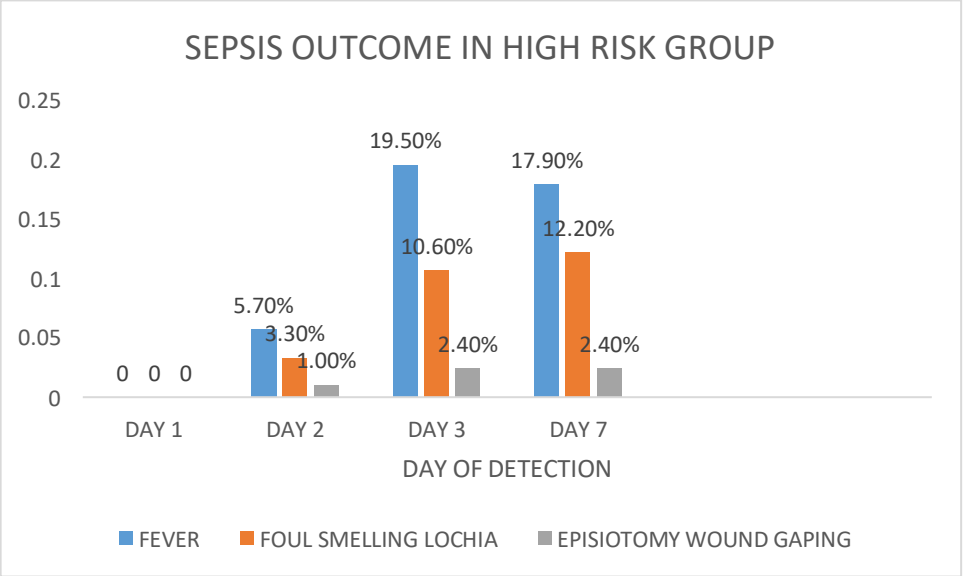


Table 20: Lactate level in individual high risk factors.

	FREQUENCY	MEAN	(PERCENTAGE)%
INDUCED LABOR	**35**	**1.7**	**33.9**
PROM	**76**	**1.7**	**61.7**
PRETERM LABOR	**61**	**1.8**	**48.7**
PROLONGED LABOR	**20**	**1.6**	**16.26**
INSTRUMENTAL DELIVERY	**19**	**2.0**	**15.44**

The above table depicts the number of high-risk group. Out of 123 35(33.9%) of them were indued labour and 76(61.7%) of them had PROM, 61(48.75%) of them had preterm labour and 20(16.26%) of them had prolonged labour more than 24 hours and 19(15.44%) of them had instrumental delivery.

Graph 18: Percentage of high risk patients

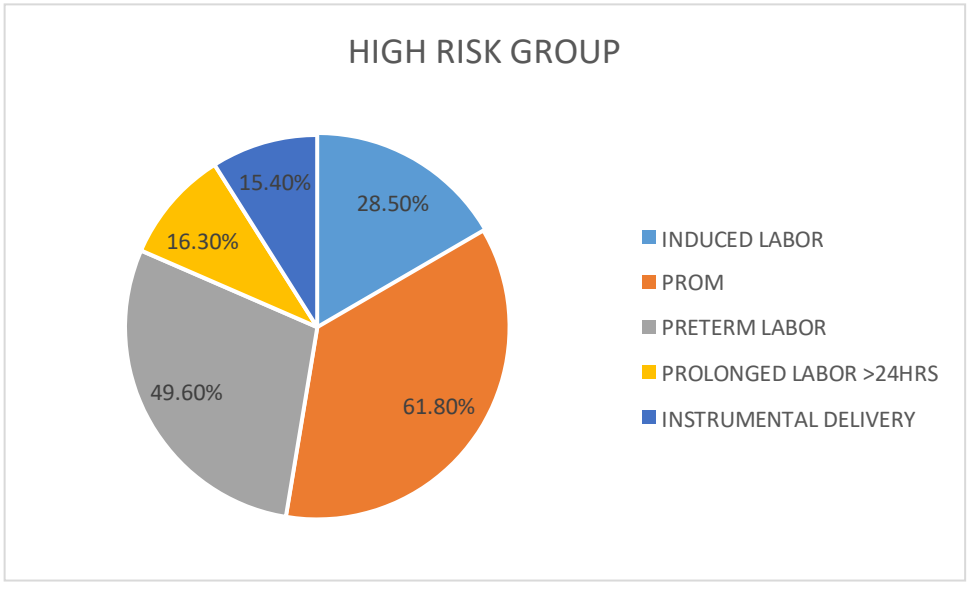


Table 21: Statistics of Serum lactate, Total leucocyte count and N/L ratio.

STATISTIC	LACTATE	TLC	N/L RATIO
SENSITIVITY	66.67%	53.66%	78.05%
SPECIFICTY	21.26%	47.24%	37.80%
PPV	45.05%	49.62%	54.86%
NPV	39.71%	51.28%	64.00

Table 22: Outcome of maternal sepsis in low risk group

FEVER	YES/NO	N	LACTATE (mmol/L)	TLC (cells/mm3)	N/L RATIO
	YES	18	2.7	15487	3.2
	NO	109	1.6	11366	2.6
	P VALUE		**0.000**	**0.001**	**0.106**
FOUL SMELLING LOCHIA	YES	11	2.7	15866	3.7
	NO	116	1.6	10998	2.4
	P VALUE		**0.000**	**0.000**	**0.002**
EPISIOTOMY GAPING	YES	2	3	17255	2.3
	NO	125	1.7	11734	2.7
	P VALUE		**0.22**	**0.35**	**1.000**

This table shows the outcome of maternal sepsis in low risk group. 18 patients developed fever in low risk group and 109 were asymptomatic with mean lactate value of 2.7mmol.L, TLC of 15487 cells/mm3 and N/L ratio of 3.2. The p values obtained for serum lactate is 0.000 and for TLC it is 0.001 there by showing its significancy. The p value obtained for N/L ratio is 0.106 showing that it is not significant. 11 patients developed foul smelling lochia with lactate value of 2.7. TLC of 15866 and N/L ratio of 3.7. The p values obtained for lactate is 0.000, TLC is 0.000 and for N/L ratio it is 0.002 there by showing that all the three tests are significant. 2 of the low risk patients had episiotomy wound gaping with lactate value of 1.7. TLC of 11734cells/mm3

and N/L ratio of 2.7. The p values obtained for lactate is 0.22, for TLC it is 0.35 and for N/L ratio it is 1 there by showing that non of the tests are significant.

Table 23: Outcome of maternal sepsis in high risk group

FEVER	YES/NO	N	LACTATE (mmol/L)	TLC (cells/mm3)	N/L RATIO
	YES	22	2.8	17083	6.4
	NO	101	1.4	11378	3.1
	P VALUE		**0.000**	**0.002**	**0.002**
FOUL SMELLING LOCHIA	YES	31	2.9	158681	5.7
	NO	98	1.5	11837	3.4
	P VALUE		**0.000**	**0.003**	**0.002**
EPISIOTOMY GAPING	YES	3	3	14330	2.5
	NO	120	1.6	12152	3.6
	P VALUE		**0.27**	**0.6**	**0.637**

This table shows the outcome of maternal sepsis in high risk group. 22 patients developed fever in low risk group and 101were asymptomatic with mean lactate value of 2.8mmol.L, TLC of 17083 cells/mm3 and N/L ratio of 6.4. The p values obtained for serum lactate is 0.000 and for TLC it is 0.002 and for N/L ratio is 0.002 showing that it is significant. 25 patients developed foul smelling lochia with lactate value of 2.9. TLC of 158681 and N/L ratio of 5.7. The p values obtained for lactate is 0.000, TLC is 0.003 and for N/L ratio it is 0.002 there by showing that all the three tests are significant. 3 of the low risk patients had episiotomy wound gaping with lactate value of 1.6. TLC of 12512cells/mm3 and N/L ratio of 3.6. The p values obtained for lactate is 0.27, for TLC it is 0.6 and for N/L ratio it is 0.637 there by showing that non of the tests are significant.

Table 24: Outcome of maternal sepsis in both groups

FEVER	YES/NO	N	LACTATE (mmol/L)	TLC (cells/mm3)	N/L RATIO
	YES	40	2.3	14465	3.3
	NO	210	1.6	10917	2.4
	P VALUE		**0.000**	**0.000**	**0.024**
FOUL SMELLING LOCHIA	YES	36	2.3	11112	2.5
	NO	214	1.6	14560	3.3
	P VALUE		**0.000**	**0.000**	**0.24**
EPISIOTOMY GAPING	YES	5	2.5	14815	3.22
	NO	245	1.67	11314	2.56
	P VALUE		**0.000**	**0.001**	**0.051**

This table shows the outcome of maternal sepsis in both groups. 40 patients developed fever in low risk group and 210were asymptomatic with mean lactate value of 2.3mmol.L, TLC of 10917cells/mm3 and N/L ratio of 3.3. The p values obtained for serum lactate is 0.000 and for TLC it is 0.000 and for N/L ratio is 0.024 . 36 patients developed foul smelling lochia with lactate value of 2.3mmol/L. TLC of 11112 and N/L ratio of 2.5 The p values obtained for lactate is 0.000, TLC is 0.000 and for N/L ratio it is 0.24. 5 patients had episiotomy wound gaping with lactate value of 2.5 TLC of 11314 cells/mm3 and N/L ratio of 2.56. The p values obtained for lactate is 0.000, for TLC it is 0.001 and for N/L ratio it is 0.051.

NEONATAL OUTCOME

Table 25: Neonatal outcome in low risk and high risk group

NEONATAL OUTCOME	LOW RISK GROUP	HIGH RISK GROUP	P VALUE
MOTHERSIDE	93(73.25)	53(43.5%)	<0.01
NICU	33(26%)	65(52.8%)	<0.01
NEONATAL SEPSIS	1(0.8%)	3(2.9%)	0.32
DEATH WITHIN 7 DAYS	0	5(4.1%)	0.09

The above table depicts the comparison of neonatal outcome in both the groups**.** In low-risk group 93(73.25%) of the neonates were given to mother after delivery, and p value obtained was <0.01 which is significant.33(26%) of them were admitted to NICU and the p value obtained using t test is <0.01 ,1(0.8%) baby was diagnosed with neonatal sepsis and none of them died within 7 days.

In high-risk group 53(43.5%) of babies were given to mother side immediately after delivery, 65(52.8%) of them were admitted to NICU and 3(2.9%) babies had neonatal sepsis and 5(4.1%) of them died within 7 days of birth. The cause for the death was mostly due to prematurity and not due to sepsis.

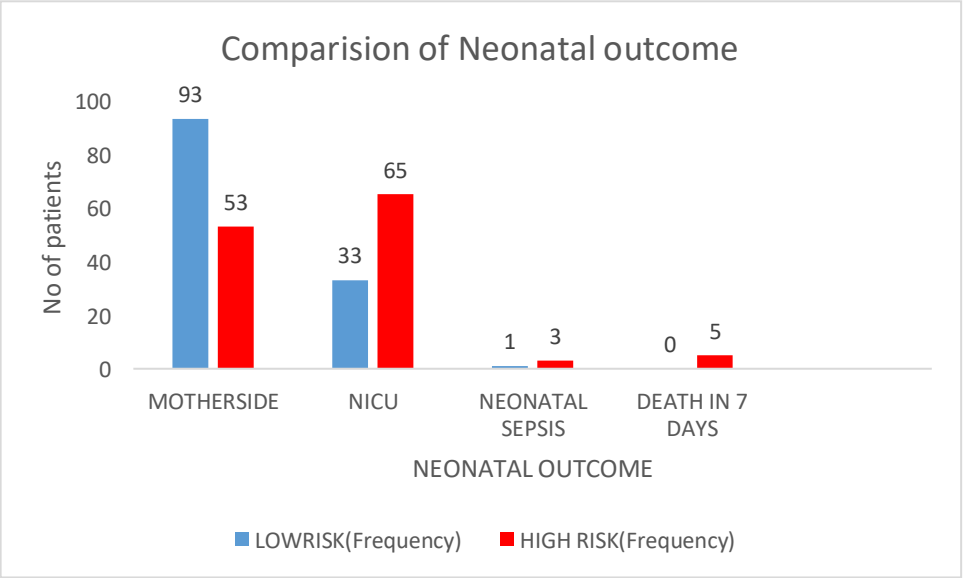
Lactate values in neonates who went into sepsis in low risk and high risk group

Table 26:

LOW RISK GROUP	HIGH RISK GROUP
3.0mmol/L	2.9mmol/L
	3.1mmol/L
	4.3mmol/L

A total of 4 neonates were diagnosed with neonatal sepsis of which 1 in low risk group with a lactate value of 3.ommol/l, and in high risk group 3 neonates were diagnosed with sepsis with lactate values of 2.9mmol/L, 3.Immol/L and 4.3mmol/L respectively.

Graph 19: Comparision of neonatal outcome in low risk group and high risk group



DISCUSSION

The study was conducted for 1 year 6 months at BLDE(DU), Shri B.M.Patil Medical College, Vijayapura, Karnataka, India. It is a cross sectional study. In this study the efficacy and feasibility of serum lactate. Total leucocyte count, and neutrophil to lymphocyte ratio as a predictor of puerperal sepsis are studied and their role as a screening test are studied.

In our study, a total of 250 patients have been taken into the study of which 127 of them were taken into low-risk group and 123 into high-risk group.

The factors determining low risk group are spontaneous onset of labor, cephalic presentation, delivery within 24hours of onset of labor and 1st and 2nd degree perineal tears. The factors determining high risk factors are Induced labour, PROM, preterm labour, delivery more than 24hrs of onset of labour, instrumental delivery.

All of the patients were delivered by vaginal delivery and the follow up was done for 7days post-delivery for any symptoms of sepsis like fever, foul smelling lochia, episiotomy wound gaping. Blood samples for serum lactate, total leucocyte count and neutrophil to lymphocyte ratio was collected with 48 to 72 hours of the delivery.

In both the groups none of them developed fever on day 1 of delivery. In low-risk group 4 of them and on high-risk group 7 of them developed fever on day 2 with a p value of 0.32. On day 3 of delivery, 16 in the low-risk group and 24 in the high-risk group developed fever with a p value of 0.13. On day 7, 18 of the low-risk group patients and 22 of the high-risk group patients had fever with a p value of 0.42. In high risk group, 31 of the patients had foul smelling lochia on day 2, while in low risk group 4 of them had foul smelling lochia with a p value of 0.04 which is significant. On day 3, 8 of the low-risk group and 13 of the high-risk group patients developed foul smelling lochia with a p value of 0.22.

On day 7 of delivery ,11(8.7%) of the low-risk group patients and 15(12.2%) of the high-risk group patients had foul smelling lochia with a p value of 0.36. In low-risk group none of them

had episiotomy wound gaping on day 1 and 2 of delivery. In high-risk group 1 patients had episiotomy wound gaping on day 2 of delivery with a p value of 0.3. In low-risk group, 4(3.2%) of them had episiotomy wound gaping on day 3 whereas in high-risk group 3(2.4%) of them had episiotomy wound gaping on day3 with a p value of 0.62. On day 7 ,2(1.6%) of them in low risk and 3(2.4%) of them in high-risk group had episiotomy wound gaping with a p value of 0.72.

In low-risk group 27 patients had raised serum lactate levels whereas in high-risk group 41 of them had raised lactate levels with a p value of 0.15. In low-risk group, 60(47.2%) patients had raised total leucocyte count and in high-risk group 57 patients had raised total leucocyte count with a p value of 0.54. In low-risk group, 27(21.9%) patients had raised neutrophil to lymphocyte ratio, whereas in high-risk group 48(37.7%) of them had raised neutrophil to lymphocyte ratio with a significant p value of <0.01.

Majority of the patients in our study were of the age group of 20-29 with a mean of 24.56, and in high-risk group the mean age group was 24.02 with a p value of 0.23. In a study conducted by **Meharun-Nissa Khaskheli**(ps-2) et al, on Risk factors and complications of puerperal sepsis at a tertiary healthcare centre, majority of the patients (65%) of them in their study were of the age of 31 years and above. In a study conducted by **Ezechi MD (**puerperium articles**)** et al, majority of the patients in their study were of the age group of 20-24 years.

In our study, most of the patients were multiparous women in both groups being 62%, and primigravida being 38% of them. In a study conducted by **CD Acosta** (lactate article) et al, most of the patients in their study were primiparous women.

In our study, Total leucocyte count was raised in 46.3% of the low-risk patients and 47.24% of the high-risk patients and the p value is 0.54. In study conducted **by Meharun-Nissa Khaskheli** et al Total leucocyte count was raised in 72.09% of the patients with a p value of 0.001. Fever

was seen in 29.9% of the low-risk patients and 43.0% of the high-risk patients in our study. whereas in this study fever was seen in 90.69% of the patients.

In our study, most of the patients were of the gestational age of 37-39 weeks in low-risk group and in high risk most of them are 34-36 weeks. In a study conducted by **Alveera (**3rd article) et al, on Serum lactate: an independent predictor of severe sepsis in obstetric patients, most (61%) of the patients were of the gestational age of 28-35 weeks.

In this study, 33.9% of the patients had induced labour and 61.7% of the patients had PROM, and 16.26% of the patients had prolonged labour more than 24hrours.In a study conducted by **Dare MD** (lactate article) et al,30.8% of them were induced labour, and prolonged labour more than 24 hours was seem in 21.9% and PROM was seen in 31.5% of the patients.

In our study, preterm birth was seen in 48.7% of the high-risk patients and NICU admission among them was seen in52.8% of the patients. In a study conducted by **Acosta** (serum lactate article) et al, preterm birth was seen in 19.1% of the patients and NICU admission was seen in 42.2% of the babies. In both the studies it was observed that the NICU admission rates were more in the babies whose mothers had PROM and also the signs and symptoms of sepsis was more in the patients who had PROM and prolonged labour for more than 24 hours.

In our study episiotomy was given for 48.8%% of the patients and 1st and 2nd degree perineal tears was seen in 16.5% and 14.17% respectively. In a study conducted by **Dare et al,** episiotomy was given in 27.4% of the patients and perineal tears were seen in 11.1% of the patients.

In our study serum lactate was raised in 21.2% of the low-risk patients and 33.35 of the high-risk patients. In a study conducted by **Alveera(3rd article)** et al, 35% of the patients had raised serum lactate value of >2.2mmol. In our study for serum lactate test, the PPV was 45.05%, NPV of 39.71%, Sensitivity of the test was 66.6% and Specificity was 21.26% with a p value of

0.00 where as in the study conducted by Alveera, the PPV was 100, NPV was 53.8%, Sensitivity of 53.8% and specificity was 100% with a p value of 0.001.

This difference might be because of the difference in sample size and due to the different inclusion and exclusion criteria as high-risk factors like hypertension, diabetes, pre-eclampsia and eclampsia are excluded in our study. In our study fever was reported in 29.9% of the low-risk patients and 43% of the high-risk patients and leukocytosis was seen in 47% of the high risk and 46.3% of the low-risk patients. Whereas in their study fever was seen in 38.10% and leukocytosis was seen in 10.71% of the patients.

In our study serum lactate value of > 4mmol was seen in 2.4% of the low-risk patients and 3.2% of the high-risk patients. In study conducted by **R Agarwal et al (2**nd article), 18.5% of them had serum lactate of more than 4mmol in non-severe sepsis and 81% of the severe sepsis. More over in their study it was seen that patients with serum lactate value of >3mmol/L had multiorgan failure in most of them. The sensitivity and specificity of serum lactate in their study was 37.93% and 88.10% respectively with a p value of 0.006 in patients with lactate value of > 4 mmol/L.

In a study conducted by **Orr, K (5th article)** et al the median lactate level was 2.5mmol/L.67.8% of them had elevated lactate of which 18.7% of them had serum lactate value of > 4mmol/L. It was concluded in their study that in the absence of infection a significant number of low-risk women will have a lactate concentration greater than the current RCOG standard for sepsis recognition. In the absence of clinical signs or features of sepsis, elevated CRP and lactate should not be considered as diagnostic of maternal infection.

CONCLUSION

Studies available so far have been conducted in patients who are already in established sepsis, hence this study was conducted to evaluate the efficacy of these laboratory tests to detect sepsis even before the onset of clinical features

- Puerperal sepsis can be diagnosed by various laboratory tests and biochemical markers. The parameters used in this study for the screening of puerperal sepsis are Serum lactate, Total leucocyte count, Neutrophil to lymphocyte ratio.
- These parameters are selected as they are easy to perform, noninvasive tests and also less expensive.
- The sensitivity of serum lactate is more than its specificity as per the results of our study
- The results obtained with total leucocyte count were not significant.

Based on the results obtained in this study, Serum lactate and Total leucocyte count can be used as screening tools for patients with fever and foul smelling lochia in all patients irrespective of risk category, whereas N/L ratio can be used only in patients with fever. In high risk patients, these tests could predict fever and foul smelling lochia and in where as in low risk patients they could predict only foul smelling lochia.

Larger studies are needed to prove the usefulness and efficacy of these tests for prediction of puerperal sepsis.

LIMITATIONS

Limitations of this study are

1) Small sample size
2) Specific cause of fever and increased leucocyte counts could not be made out as they occur in many other conditions also other than sepsis.

SUMMARY

This study included a total of 250 pregnant women who came for delivery at BLDE(DU), Shri B.M.Patil Medical College Hospital and Research Institute , Vijayapura, Karnataka, India. Of the 250 patients, 127 were taken in the low-risk group study and 123 were taken into high-risk group.

All the patients after giving consent were included in the study as per the inclusion and exclusion criteria.

All the three tests were performed according to the methodology.

The tests include evaluation Serum lactate, Total leucocyte count, Neutrophil to lymphocyte ratio within 48 to 72 hours of delivery.

All the patients were followed up till 7 days after delivery to see for any symptoms of sepsis like fever, foul smelling lochia and episiotomy wound gaping.

A total of 250 women were taken into study, of which 127 of them were taken into low-risk group and 123 of them into high-risk group. Most of the patients in both the groups belong to upper lower class of modified kuppuswamy classification and rural area.

In low-risk group, lactate was raised >2.1mmol/L in 27(21.2%) patients, in high-risk group in 41(33.3%) patients with a p value of 0.032. In low-risk group TLC was raised>12,000 in 57(46.3%) patients, in high-risk group it was raised in 60(47.2%) patients with p value of 0.7936. In low-risk N/L ratio was raised >3.8 in 27(21.9%) patients and in high-risk group it was raised in 48(37.7%) patients with a p value of 0.1801.

- Serum lactate had a sensitivity of 66.67%, Specificity of 21.26% positive predictive value of 45.05%, and negative predictive value of 39.71%.
- Total leucocyte count had a sensitivity of 53.66%, specificity of 47.24%, positive predictive value of 49.62%, and negative predictive value of 51.285.
- Neutrophil to lymphocyte ratio had a sensitivity of 78.05%. specificity of 37.08%, positive predictive value of 54.86% and negative predictive value of 64.0%.

- A total of 4 neonates had neonatal sepsis, of which 1 is in lowrisk group and 3 are of high risk group. The serum lactate levels in neonate of low risk group with neonatal sepsis was 3mmol/L, in high risk group the values are 2.9mmol/l, 3.1mmol/l and 4.3mmol/l respectively.
- Lactate and Total leucocyte count were significant as per the results of the study. The results obtained with N/L ratio were not significant.

Based on the results of all the three tests, serum lactate and TLC can be used as screening tools for screening of puerperal sepsis. The efficacy of N/L could not be proved. Further studies are needed to prove the efficacy of these tests as screening tools. In our study almost same number of patients were symptomatic in both the groups and hence these tests can be used in patients with low risk and high factors.

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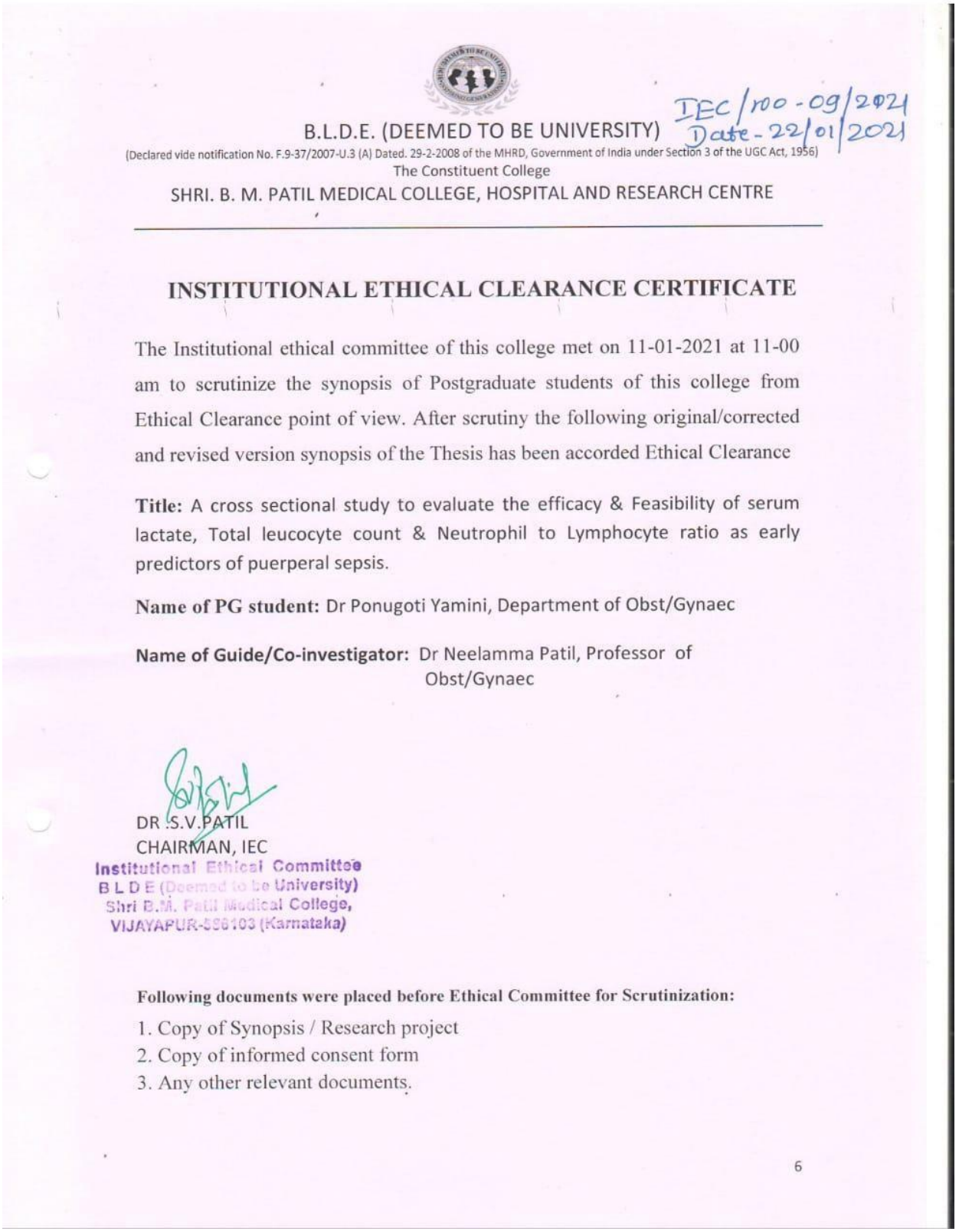
ANNEXURE-I

ETHICAL CLEARANCE



B.L.D.E. (DEEMED TO BE UNIVERSITY)

IEC/100-09/2021
Date - 22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11-00 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: A cross sectional study to evaluate the efficacy & Feasibility of serum lactate, Total leucocyte count & Neutrophil to Lymphocyte ratio as early predictors of puerperal sepsis.

Name of PG student: Dr Ponugoti Yamini, Department of Obst/Gynaec

Name of Guide/Co-investigator: Dr Neelamma Patil, Professor of Obst/Gynaec

DR .S.V.PATIL
CHAIRMAN, IEC
Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

6

13.

ANNEXER-II

CONSENT FORM

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B.M. PATIL MEDICAL COLLEGE

HOSPITAL AND RESEARCH CENTER, VIJAYAPURA-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned,_____, D/O W/O_____, aged_____years, ordinarily resident of_____do hereby state/declare that Dr PONUGOTI YAMINI of Shri. B. M. Patil Medical College Hospital and Research Centre has examined me thoroughly on_____at_____(place) and it has been explained to me in my own language that I am suffering from_____ disease (condition) and this disease/condition mimic following diseases. Further **Dr PONUGOTI YAMINI** informed me that he/she is conducting dissertation/research titled **"A CROSS SECTIONAL STUDY TO EVALUATE THE EFFICACY AND FEASIBILITY OF SERUM LACTATE, TOTAL LEUCOCYTECOUNT AND NEUTROPHIL TO LYMPHOCYTE RATIO AS EARLY PREDICTORS OF PUERPERAL SEPSIS"** under the guidance of **Dr. NEELAMMA PATIL** requesting my participation in the study. Apart from routine treatment procedure, the pre-operative, operative, post-operative and follow-up observations will be utilized for the study as reference data. Doctor has also informed me that during conduct of this procedure like adverse results may be encountered. Among the above complications most of them are treatable but are not anticipated hence there is chance of aggravation of my condition and in rare circumstances it may prove fatal in spite of anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this study would help in evaluation of the results of the study which is useful reference to treatment of other similar cases in near future, and also, I may be benefited in getting relieved of suffering or cure of the disease i am suffering.

The Doctor has also informed me that information given by me, observations made photographs video graphs taken upon me by the investigator will be kept secret and not assessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary, based on information given by me, I can ask any clarification during the course of treatment / study related to diagnosis, procedure of treatment, result of treatment or prognosis. At the same time I have been informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged.

After understanding the nature of dissertation or research, diagnosis made, mode of treatment, I the undersigned Smt_____under my full conscious state of mind agrees to participate in the said research/dissertation.

Signature of patient: Signature of doctor:

Date:

Place:

ANNEXER-III

CASE PROFORMA

"A CROSS SECTIONAL STUDY TO EVALUATE THE EFFICACY AND FEASIBILITY OF SERUM LACTATE, TOTAL LEUCOCYTE COUNT AND NEUTROPHIL TO LYMPHOCYTE RATIO AS EARLY PREDICTORS OF PUERPERAL SEPSIS"

NAME: AGE: DOA

IN PATIENT NUMBER (I.P No.): DOD

DATE OF SAMPLE COLLECTION:

ADDRESS:

PHONE NUMBER :1) 2) 3)

OBSTETRIC HISTORY:

PAST HISTORY:

ON EXAMINATION

TEMPERATURE: PULSE: BLOOD PRESSURE: SPO2:

CVS: RS: PALLOR:

PER ABDOMEN:

DATE OF DELIVERY:

DIAGNOSIS:

EPISIOTOMY: YES / NO

PERINEAL TEARS:

YES/NO

BLOOD GROUP:

CATEGORY: LOWRISK

- SPONATANEOUS INSET OF LABOR
- DELIVERY WITHIN 24HRS OF ONSET OF LABOR
- CEPHALIC PRESENTATION
- EPISIOTOMY
- 1^{ST} AND 2^{ND} DEGREE PERINEAL

HIGH RISK GROUP

- INDUCED LABOR
- PROLONGED LABOR MOREBTHAN 24HRS
- PRETERM LABOR
- PREMATURE RUPTURE OF MEMBRANES
- $3^{RD}/4^{TH}$ DEGREE PERINEAL TEAR
- INSTRUMENTAL DELIVERY

Antibiotics details drug, dose, and duration of course : IV ORAL

DAY1 -FEVER
FOUL SMELLING LOCHIA
EPISIOTOMY WOUND GAPING

DAY 2-
FEVER
FOUL SMELLING LOCHIA
EPISIOTOMY WOUND GAPING

DAY 3

FEVER
FOUL SMELLING LOCHIA
EPISIOTOMY WOUND GAPING

DAY 7

FEVER
FOUL SMELLING LOCHIA
EPISIOTOMY WOUND GAPING

MATERNAL INVESTIGATIONS:

SERUM LACTATE
TOTAL LEUCOCYTE COUNT
NEUTROPHIL TO LYMPHOCYTE RATIO

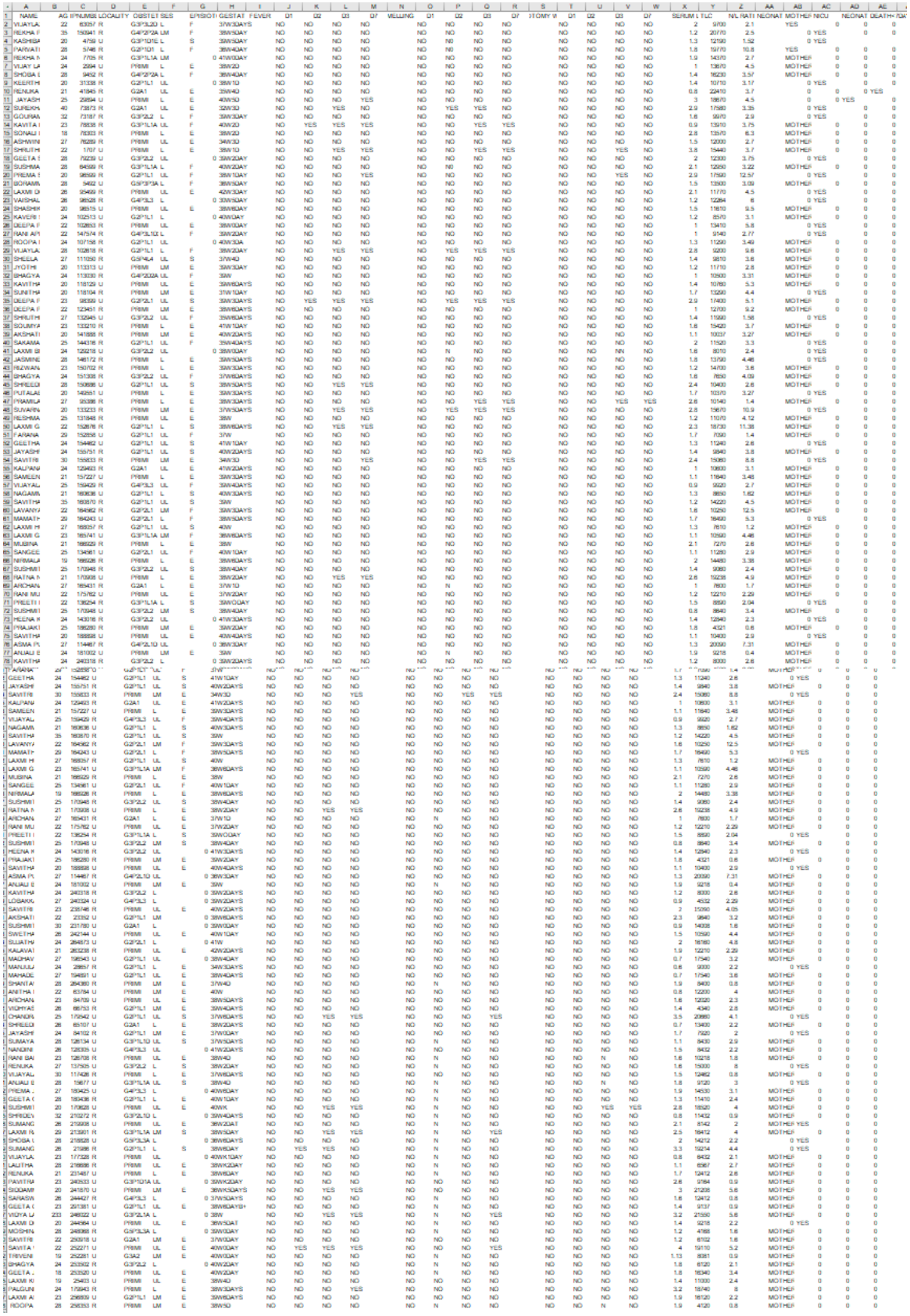
NEONATAL OUTCOME:

MOTHERSIDE NICU NEONATAL SEPSIS DEATH WITHIN 7 DAYS

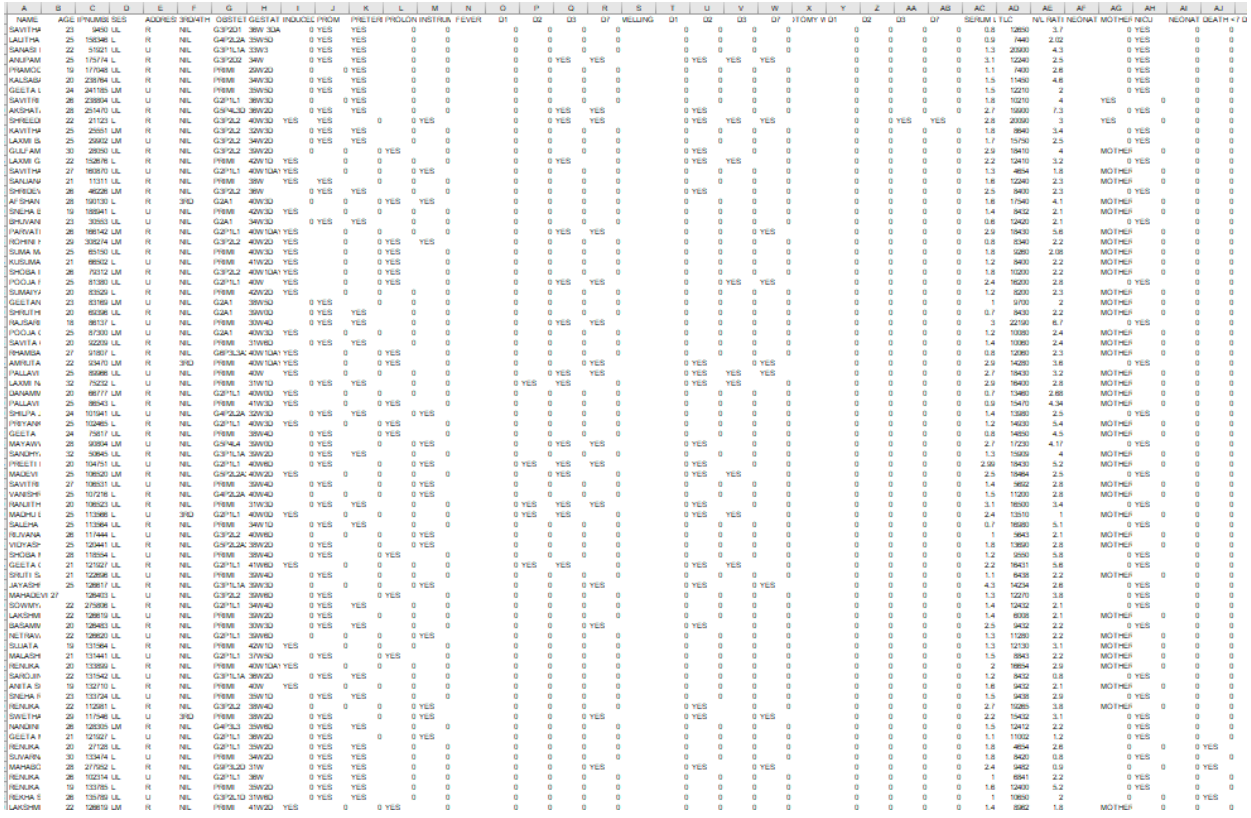
ANNEXER-IV

MASTER CHARTS

LOW RISK GROUP EXCEL SHEET



HIGH RISK GROUP EXCEL SHEET



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