COMPARATIVE STUDY OF VAGINAL MISOPROSTOL TABLET VERSUS DINOPROSTONE INSERT IN INDUCTION OF LABOR

A Dissertation submitted by

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In partial fulfilment of the requirements for the award of degree of

MASTER OF SURGERY

In OBSTETRICS AND GYNAECOLOGY

UNDER THE GUIDANCE OF

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I, **DR. VAISHNAVI MAHESH UNNI** hereby declare that this dissertation entitled **"COMPARATIVE STUDY OF VAGINAL MISOPROSTOL TABLET VERSUS DINOPROSTONE INSERT IN INDUCTION OF LABOR"** is a Bonafide and genuine research., work carried out by me under the guidance of **DR. SUBHASHCHANDRA R. MUDANUR,** Professor, Department of Obstetrics and Gynaecology, Shri. B. M. Patil Medical College, Hospital & Research Centre, Vijayapura.

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ABSTRACT

Introduction:

Induction of labor (IOL) is an essential obstetric procedure for maternal and fetal safety when continuing pregnancy is risky. Prostaglandin analogues like Misoprostol (PGE1) and Dinoprostone (PGE2) are commonly used for cervical ripening and labor induction. This study compares the efficacy and safety of vaginal tablet Misoprostol versus Dinoprostone insert in IOL.

Aims and objectives : To compare efficacy and safety of the vaginal Tablet Misoprostol with Dinoprostone insert in induction of labour and to compare fetal and maternal outcomes.

Materials and Methods:

This prospective, interventional study was conducted at BLDE University, Vijayapura, from April 2023 to February 2025, involving 106 pregnant women at term. Participants were divided into two groups: Group 1 received a 10 mg Dinoprostone vaginal insert, while Group 2 received 25 mcg vaginal Misoprostol every four hours. Key outcomes included vaginal delivery rates, cesarean section rates, and maternal and neonatal complications. Data were analyzed using SPSS (Version 20).

Results:

Vaginal delivery occurred in 73.58% of the Dinoprostone group compared to 50.94% in the Misoprostol group ($p=0.02$). The mean induction-to-delivery interval was significantly shorter in the Misoprostol group (15.2 ± 4.9 hours vs. 18.3 ± 4.29 hours, $p<0.001$). Maternal complications, including postpartum hemorrhage (PPH), were more common in the Misoprostol group (24.5%). Neonatal complications, such as NICU admissions and lower Apgar scores, were also significantly higher in the Misoprostol group.

Conclusion:

Both Misoprostol and Dinoprostone are effective for labor induction, but Misoprostol shortens the induction-to-delivery interval at the cost of increased cesarean rates and fetal complications. Dinoprostone, while slower, shows better fetal outcomes and fewer complications, making it a preferable option for high-risk pregnancies. Tailoring the choice of induction agent based on patient-specific factors is essential.

Keywords: Misoprostol, Dinoprostone, Labor Induction.

ABBREVIATIONS

S No.	ABBREVIATIONS	EXPANSION
1	IOL	INDUCTION OF LABOR
2	PG	PROSTAGLANDINS
3	WHO	WORLD HEALTH ORGANISATION
4	FIGO	INTERNATIONAL FEDERATION OF OBSTETRICS AND GYNAECOLOGY
5	SL	SUBLINGUAL
6	IUFD	INTRAUTERINE FETAL DEMISE
7	PROM	PREMATURE RUPTURE OF MEMBRANES
8	LSCS	LOWER SEGMENT CAESAREAN SECTION
9	NST	NON STRESS TEST
10	ACOG	AMERICAN COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS
11	PPH	POST PARTUM HEMORRHAGE
12	OM	ORAL MISOPROSTOL
13	MVI	MISOPROSTOL VAGINAL INSERT
14	DVI	DINOPROSTONE VAGINAL INSERT

15	**NICE**	NATIONAL INSTITUTE FOR HEALTH AND CARE EXCELLENCE GUIDELINES
16	**NSAIDS**	NON STEROIDAL ANTI-INFLAMMATORY DRUGS
17	**AA**	ARACHIDONIC ACID
18	**COX**	CYCLOOXYGENASE
19	**SRS**	SAMPLE REGISTRATION SYSTEM

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INTRODUCTION

INTRODUCTION

Induction of labor is defined as, "stimulation of contractions before the spontaneous onset of labor, with or without ruptured membranes." When the cervix is closed and uneffaced, labor induction is often preceded by *cervical ripening*, a process to soften and open the cervix. *Augmentation* refers to "enhancement of spontaneous contractions that are considered inadequate because of failed cervical dilation and poor fetal descent".[1]

About ten percent of pregnant women globally undergone this procedure, with some parts of the world experiencing induction rates as high as 33%.[2]A recent multicenter study indicates that inducing labor after 39 weeks of gestation may improve maternal outcomes and decrease the incidence of cesarean deliveries without adversely affecting neonatal outcomes, even for nulliparous women in low-risk pregnancies, compared to expectant management. [3]

Annually, over 500 women succumb to labour-related problems, and around 4 million fetuses are stillborn in developing countries. According to the SRS data report of 2018, the perinatal death rate stands at a concerning 22 per 1000 live births.[4] Labour induction is steadily growing as one of the most prevalent obstetric procedures in these circumstances. Induction prevalence is 22% in India and 24.5% in the USA. [5,6]

Cervical ripening is an ongoing process that occurs during pregnancy and peaks immediately ahead of childbirth. The process is assisted by cytokines and interleukins such as IL-8, IL-6, and G-CSF. Hormones such as estrogen and prostaglandins have been identified to have significance in the process.[7] As the cervix matures, collagen solubility escalates, and proteoglycans endure breakdown. Inflammatory mediators such as interleukins enhance neutrophil activity. Neutrophils release enzymes such as collagenase and elastase, which degrade collagen and proteoglycans. [7]

The Bishop score, specifically assesses cervical examination result and has historically been utilized to assess cervical favorability and has been included into institutional protocols for risk stratification purposes.[8]

THE HISTORY OF LABOR INDUCTION

Hippocrates became the first to make mention of mammary stimulation and mechanical dilatation of the cervical canal.

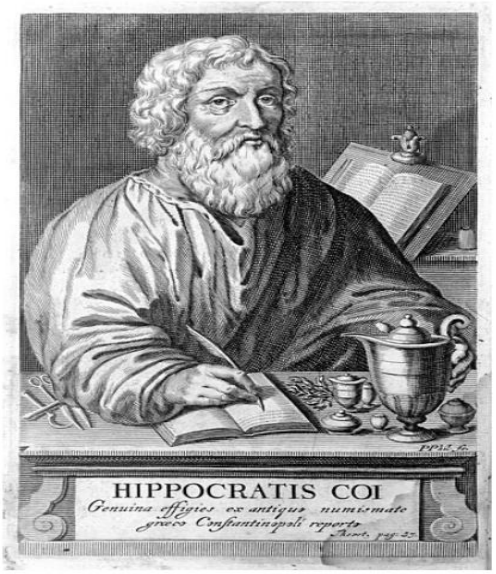

FIGURE 1: Hippocrates, **"Father of medicine"** , did the first cervical dilatation.

In the second century AD, **Soranus** employed several methods, such as artificial membrane rupture, to induce birth. Medical professionals debated the morality and effectiveness of inducing childbirth early in 1756 in London by rupturing the membranes.

Moshion was the first to describe the procedure for manually dilating the cervix, and **Casis** developed machinery that could perform so.

Mechanical techniques to induce labor became more popular starting in the 17th century. **James** was the first American to use an amniotomy to induce early childbirth in 1810.

Until the 20th century, labor induction was most frequently accomplished using amniotomy and other mechanical techniques.

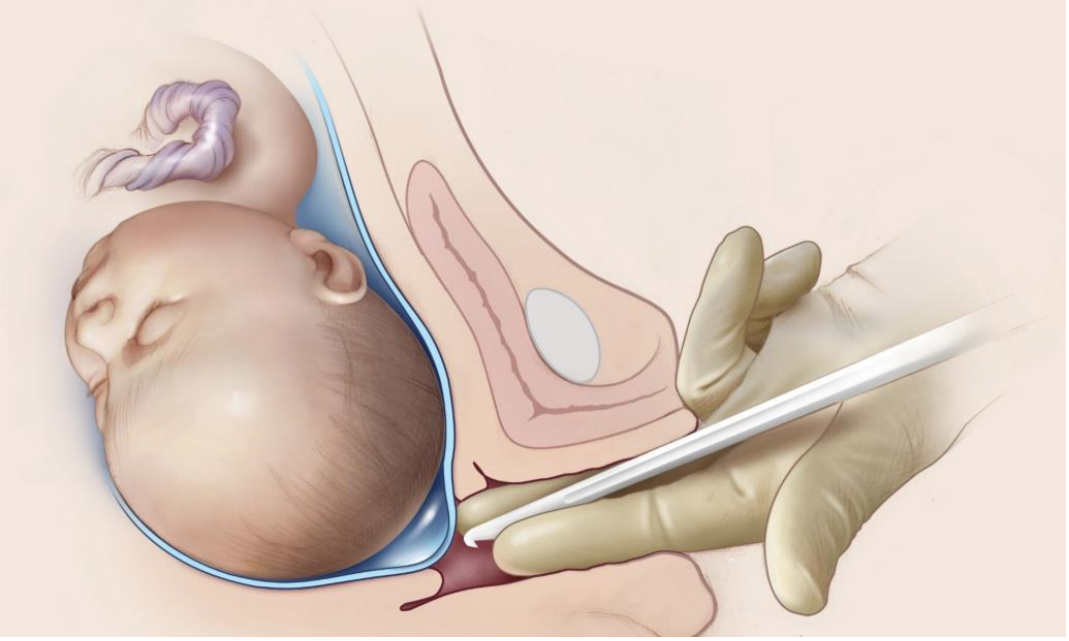

FIGURE 2: Amniotomy for induction of labour

Dale found in 1906 that preparations from the infundibular lobe within the pituitary gland caused myometrial contractions. Three years later, Bell reported the use of a pituitary extract for inducing labor. The pituitary extract was first used as a hormonal method of labor induction in 1913, and since then, obstetricians have accepted its use. However, various adverse effects were recorded due to a result of the usage of high doses and the extract's impurity. The application of pituitary extract began to decline in several institutions as the likelihood of uterine rupture soared.

The initial analysis on the application of prostaglandins for labor induction was issued in 1968 by Karim and colleagues. Since then, labor induction has commonly employed prostaglandins in various forms and sorts.

Several methods for cervical ripening are available, spanning from mechanical, pharmacological, surgical, or a combination of these.[11]

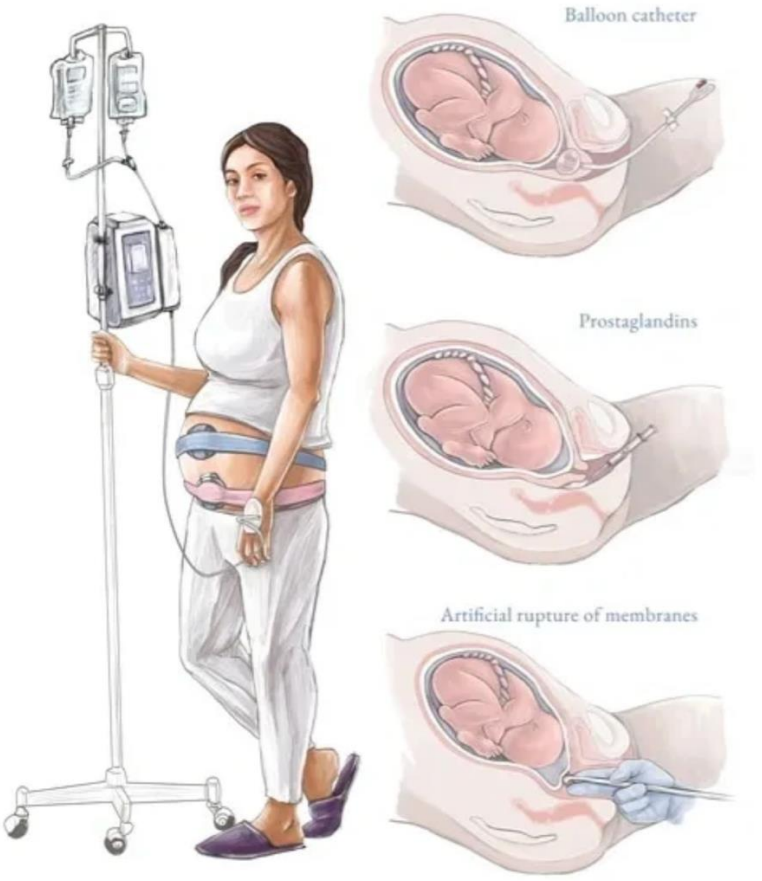

FIGURE 3: Various methods for induction of labour.

Prostaglandin analogues, dinoprostone (PGE2) and misoprostol (PGE1), are widely used in labor induction to aid in cervical ripening and promote uterine contractions, resulting in favoring vaginal birth. Although dinoprostone is FDA-approved for cervical ripening in women at or near term, misoprostol awaits FDA approval for this kind of usage, despite its benefits of reduced cost, no refrigerated specifications, and perhaps greater efficacy. [10]

Dinoprostone, a form of prostaglandin E2, is an FDA-approved drug employed for the evacuation of uterine contents and to promote the onset of labor.[9] The dinoprostone vaginal pessary is a controlled-release pharmaceutical device containing 10 mg of dinoprostone, facilitating a continuous release at a rate of 0.3 mg per hour throughout a period of twenty-four hours. The simplicity of administration and lesser requirement for vaginal assessments boost patient comfort. [11]

FIGURE 4: Dinoprostone ***Propess*** vaginal pessary

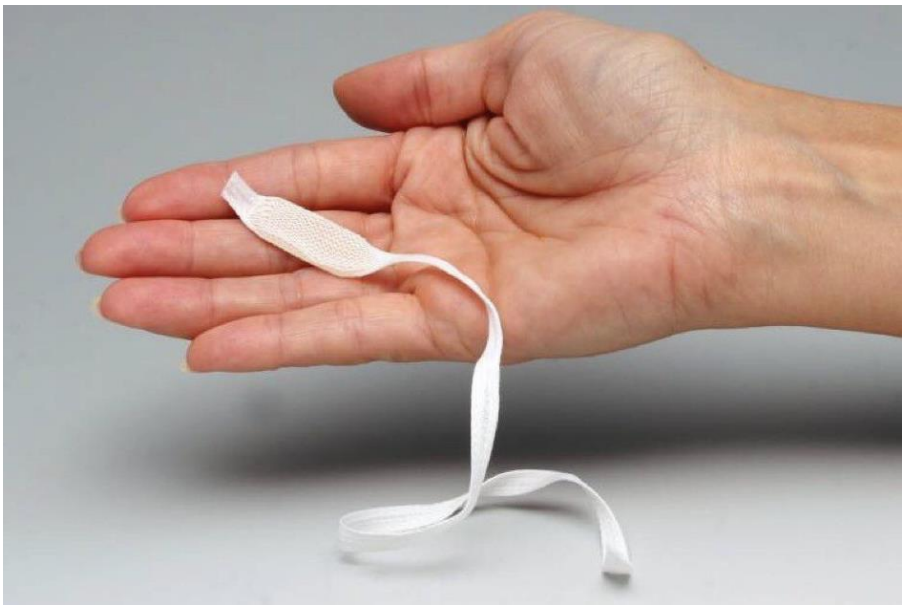

FIGURE 5: Structure of Dinoprostone ***Propess*** pessary.

Misoprostol, a cost-effective and room temperature stable PGE 1 analogue approved for the treatment of NSAID-induced ulcers, has garnered interest in obstetrics and Gynaecology in recent years. Misoprostol is beneficial for labor induction, regardless of whether it is administered orally or vaginally. The interval to vaginal delivery declines with vaginal application. [12]The World Health Organization (WHO) and FIGO authorized a vaginal misoprostol dosage of 25 micrograms every 4 hours, with a maximum of 6 doses, adhering to a thorough investigation. [13]

FIGURE 6:Tablet Misoprostol 25 mcg

Therefore, the goal of this study is to evaluate the vaginal Tablet Misoprostol 25 mcg vs vaginal dinoprostone vaginal pessary for cervical ripening and induction of labor.

AIMS AND OBJECTIVES

THE PRIMARY OBJECTIVE

To compare efficacy and safety of the vaginal Tablet Misoprostol vs Dinoprostone insert in induction of labour.

THE SECONDARY OBJECTIVE

To compare neonatal and maternal outcomes.

REVIEW OF LITERATURE

1. Daniele Bolla, Saskia Vanessa Weisslender et al.(2018)

This retrospective cohort research had 200 consecutive women induced with a 200 mcg Misoprostol 24-hour vaginal insert and 200 women induced with 25 mcg Misoprostol vaginal tablets administered every 4-6 hours. The primary outcome factors encompassed induction-to-delivery time, incidence of vaginal delivery within 24 hours, occurrence of tachysystole, method of delivery, and neonatal outcomes. The induction time for the misoprostol pill was 1048 minutes, whereas the misoprostol vaginal insert had an induction time of 1510 minutes. Vaginal birth within 24 hours occurred in 127 participants from the misoprostol vaginal insert group and 110 women from the misoprostol vaginal group. Tachysystole occurred more frequently in the vaginal implant group. The rates of Caesarean section births and vaginal operation deliveries were not substantially different between the two groups.

2. Katharine Rankin, Rohan Chondakar-l et al.(2018)

This research included 200 women in the UK who were induced with Misoprostol and Dinoprostone vaginal inserts for identical purposes, primarily targeting women with a modified Bishop's score of less than 4.Outcome assessments encompassed the incidence of tachysystole and hyperstimulation, administration of prostaglandins, utilization of pre-delivery oxytocin, method of delivery, and admission to the NICU.A significantly higher rate of tachysystole and hyper stimulation is noted in Misoprostol vaginal insert group ,no difference is noted in modes of birth; the median induction period is longer in DVI group (33hrs) when compared to the MVI group (15hrs), no difference in neonatal outcomes is noted and no difference in use of predelivery oxytocin or Caesarean section rate is noted.

3. Claudia Maggi, Georgia Mazzoni et al.(2019)

A study was done on a cohort of 220 women (109 got vaginal Misoprostol insert and 111 received Dinoprostone vaginal insert) with a Bishop score of less than 4, admitted for labor induction at a single hospital, with the primary result being the vaginal delivery rate.The vaginal delivery rate was 88% in the Misoprostol group and 74% in the Dinoprostone group, with a shorter delivery time seen in the Misoprostol group. However, uterine tachysystole is seen to be more prevalent with Misoprostol.

4. **H. Bagory, C Dr Broucker et al.(2021)**

A retrospective study included 5,238 pregnant women who were induced at term using prostaglandins with an unfavorable cervix. The primary outcomes measured were the rate of vaginal deliveries within 24 hours, cesarean section rates, reasons for cesarean delivery, uterine contractility issues, and neonatal outcomes. The results showed no significant difference in efficacy between the two induction techniques. However, Misoprostol was found to be better tolerated by both the mother and the fetus.

5.Jana Beyer, Yvonne Jager et al.(2022)

This study is performed on 322 pregnant women in four German tertiary health care centres to measure the safety, efficacy and perinatal outcome of oral Misoprostol (OM) initially started with 50 mcg followed by 100mcg every 4th hourly with maximum dose of 500mcg/day, Misoprostol vaginal insert(MVI) 200mcg/24hr with maximum dose of 2x24hr and a Dinoprostone vaginal insert (DVI) 10mg/24for induction of labor at term with primary aims to study induction birth interval (IBU) ,delivery rates after 12h, 24h and 48h as well as the mode of delivery. It was found that induction of labor at term using prostaglandins was a safe intervention for mother and child. With oral Misoprostol having the highest efficacy.

INDUCTION OF LABOR

DEFINITION-

The World Health Organization defines IOL as, “The process of artificially stimulating the uterus to start labor.”

The uterus comprises the body and the cervix. The body primarily consists of smooth muscle, while the cervix is chiefly built from collagen. Throughout gestation and parturition, the cervix experiences substantial changes involving shortening, effacement, and dilatation. These physiological cervical alterations could have been triggered by mechanical or pharmacological strategies of labor induction. [14,15]

INDICATIONS-

The date of birth for late preterm, early term, late term, and post-term is contingent upon the patient's obstetric and medical history. IOL can be justified when it is believed that the prognosis for the fetus, the mother, or both is superior to that of expectant management, which involves awaiting the spontaneous commencement of labor.[16]

The American College of Obstetricians and Gynecologists (ACOG) provides an extensive set of recommendations regarding the duration of delivery, encompassing numerous prevalent instances of labor outlined below. [17]

- Oligohydramnios occurring between 36 0/7 and 37 6/7 weeks of gestation.
  • Fetal intrauterine growth restriction without abnormal Doppler findings, occurring between 38 0/7 and 39 6/7 weeks of gestation.

• Fetal intrauterine growth restriction with reversed end-diastolic flow, occurring at 32 0/7 weeks of gestation.

• Chronic hypertension, untreated, occurring between 38 0/7 and 39 6/7 weeks of gestation.

- Gestational hypertension with the timing at 37 0/7 weeks of gestation or at the time of diagnosis if diagnosed later.
- Preeclampsia without severe features with the timing at 37 0/7 weeks of gestation or at the time of diagnosis if diagnosed later.
- Preeclampsia with severe features with the timing at 34 0/7 weeks of gestation or at the time of diagnosis if diagnosed later.
- Pregestational diabetes is well-controlled, with the timing at 39 0/7 to 39 6/7 weeks of gestation.
- Gestational diabetes, diet, or exercise controlled, with the timing at 39 0/7 to 40 6/7 weeks of gestation.
- Preterm rupture of membranes with the timing at 34 0/7 weeks of gestation or at the time of diagnosis if diagnosed later.
- Abruptio placentae.
- Chorioamnionitis.
- Intrauterine fetal demise.

The **WHO** promotes,

- The induction of labor for women who exceed 41 weeks of gestation (that is, more than 40 weeks and 7 days).
- Induction of labor is not advised for women with an uneventful pregnancy before to 41 weeks of gestation.
- Induction of labor prior to to 41 weeks of gestation is not recommended if gestational diabetes is the only problem.
- Induction of labor at term is not recommended in cases involving suspected fetal macrosomia.
- Induction of labor is recommended for women with prelabor rupture of membranes at term.[2]

PREREQUISITE FOR INDUCTION:

Assessment of maternal parameters -

- Confirm that induction is required.
- Rule out contraindications of labor and/or vaginal delivery.
- Pelvic assessment
- Favourability of the cervix
- Weigh and explain the benefit and risks of induction of labor to a patient and the family

Assessment of fetal parameters-

- Period of gestation
- Effective fetal weight calculation

- Position of the fetus
- Assess fetal status

CONTRAINDICATIONS OF INDUCTION OF LABOUR-

Contraindications to IOL encompass, but are not restricted to, the following:

- Vasa previa or placenta previa
- Transverse fetal presentation
- Umbilical cord prolapse
- History of a past classical cesarean section
- Active herpes infection
- A previous myomectomy breaching the endometrial cavity [17]

PREDICTION OF LABOR INDUCTION SUCCESS/FAILURE –

According to **NICE guidelines**, induction failure occurs when there is no progress in cervical dilation and ripening, potentially leading to a higher caesarean section rate.
The ACOG consensus advocates permitting an extended latent phase (up to 24 hours) and administering oxytocin for 12-18 hours post-membrane rupture before concluding induction failure.
Additionally, dilatation must reach 6 cm before labor arrest can be diagnosed.
Research conducted by the American Maternal-Fetal Medicine Units Network, spanning more than 10,000 women, corroborates this approach by indicating that induction failure is improbable in the absence of oxytocin to induce contractions.

METHODS OF INDUCTION OF LABOR –

A broad range of approaches available for the induction of labor. The choice of approach could be contingent upon national standards, local regulations, alongside particular clinical parameters. [18]

Inducing labor in women with challenges has been associated to reduced health-service expenditures contrasted to those incurred by expectant management. [19, 20] Nevertheless, there is scant evidence regarding the costs caused by particular induction methods in juxtaposition with others. Randomized research investigations comparing different methods of induction have occasionally integrated economic evaluations. [21]

A wide array of pharmacological, mechanical, complementary, and alternative techniques have been employed to initiate labor.

1.Non-pharmacologic methods:

- Natural methods-

    1. Relaxation methods
    2. Coitus
    3. Tactile stimulation of Nipples
    4. Enema
    5. Cumin Tea
    6. Herbs
    7. Acupressure

- Mechanical methods-

    Osmotic dilators -

1. Laminaria
2. Dilapan

Balloon devices-

1. Foley's
2. Bougie

- Surgical methods-

1. Stripping of membranes
2. Amniotomy

2. Pharmacological methods:

- Oxytocin
- Prostaglandin

1. Misoprostol(1,16-Dihydroxy-16-methyl-9-oxoprost-13-en-1-oic acid methyl ester)
2. Dinoprostone [PGE2]
3. Mifepristone / RU 486

The Bishop score, established by Edward Bishop in 1964, is a cervical evaluation technique employed to forecast the successful outcome of labor induction. This grading method analyzes many clinical aspects of the cervix, including dilatation, effacement, position, consistency, and the fetal head's station within the pelvis. Each parameter generates a score, with the aggregate value varying from 0 to 13.[22] The examination is conducted throughout the late third trimester and at induction of labor. [23]
Several experts have settled on a revised Bishop score, whereby cervical length can substitute for effacement, with an upper limit score of 12.[24]

While the precise standards for classifying a cervix as favorable or unfavorable for induction remain unknown, numerous studies have utilized a score exceeding 8 on the traditional Bishop scoring system to signify a favorable cervix, or a score of at least 5 on a modified Bishop score, particularly among multiparous women at term with uncomplicated pregnancies. [22,24]

The Bishop score is frequently utilized during digital cervical assessments at induction to assess the importance of cervical ripening. Several investigations indicate that the Bishop score may lack predictive value concerning induction failure with contemporary cervical ripening modalities. [25]

BISHOP SCORE =..... (total)			Date of Bishop Score: ...../...../.....	
Score	0	1	2	3
Dilation	Closed	1 - 2	3 - 4	5
Length	> 4	3 - 4	1 – 2	0
Consistency	Firm	Medium	Soft	—
Position	Posterior	Midline	Anterior	—
Head: station	-3	-2	-1, 0	+1,+2

FIGURE 7: Modified Bishop's score

While it is a subjective evaluation approach, the Bishop score continues to be the preferred method to evaluate the cervix prior to labor induction. Multiple mechanical and medication-based treatments may be employed in individuals whose Bishop score implies the necessity of cervical ripening. Given the significant morbidity and death rates for both mother and fetus associated with emergency cesarean births, this method is praised for its

ease of use and accuracy in predicting vaginal delivery. In recent years, transvaginal ultrasonography has become a viable option for cervical assessment. Research indicates that cervical length measures acquired by transvaginal ultrasonography can serve as a reliable predictor of effective labor induction. Research comparing transvaginal ultrasonography and the Bishop score has produced inconclusive findings, lacking a definitive consensus on the superiority of either approach. Nevertheless, the Bishop score stays a prevalent and reliable instrument in obstetrics for assessing cervical suitability and advising labor induction methods.[26,27]

Prior to initiating induction of labor , all pregnant women must provide informed consent and comprehend the associated benefits, maternal and fetal dangers, as well as alternatives to IOL. The dangers associated with IOL parallel those of spontaneous labor, including the necessity for a caesarean section, surgical vaginal birth, chorioamnionitis, non-reassuring fetal heart rate patterns, and postpartum hemorrhage. Indications for caesarean section and surgical vaginal birth should be evaluated prior to proposing induction of labor. A cesarean section may be recommended in cases of failed induction of labor (IOL), where cervical dilatation remains insufficient despite the administration of drugs, with or without amniotomy. The American College of Obstetricians and Gynecologists (ACOG) advises about the use of oxytocin for 12 to 18 hours post amniotomy prior to executing a caesarean section for a failed IOL. [28]

The rates and reasons behind Caesarean sections should be discussed with all pregnant women prior to obtaining consent for induction of labor (IOL). The medical literature and social media place a strong emphasis on cesarean section rates throughout the United States. The New England Journal of Medicine recently released the ARRIVE study, which compared caesarean section rates and perinatal outcomes in nulliparous women having elective induction of labor at 39 weeks of gestation to those undergoing expectant

treatment. The results indicated a markedly reduced caesarean section rate in the induction group, with no statistically significant reduction in detrimental perinatal outcomes. [24]

MISOPROSTOL

Misoprostol was developed in 1973.Misoprostol is an artificial prostaglandin E1 (PGE1) utilized for the prevention and treatment of gastric and duodenal ulcers, induction of labor, facilitation of abortion, and management of postpartum hemorrhage resulting from inadequate uterine contractions. [29,30]

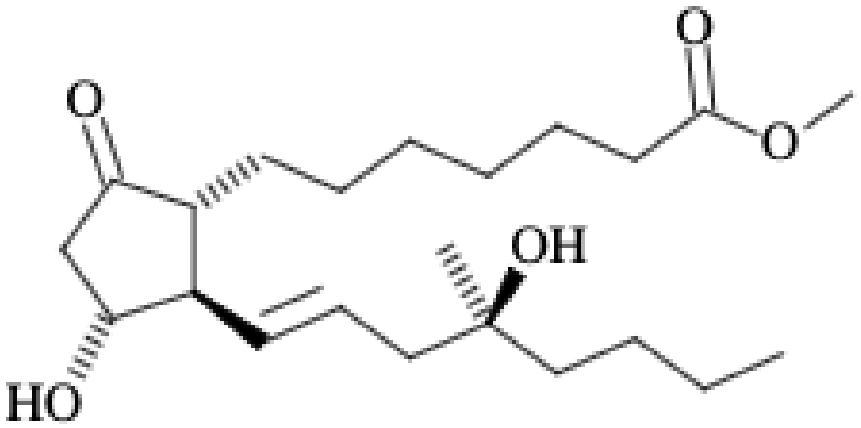

FIGURE 8: Chemical structure of Misoprostol

Administered orally, it is used to avert stomach ulcers in individuals consuming nonsteroidal anti-inflammatory medicines (NSAIDs).It is utilized for abortions either independently or in conjunction with mifepristone or methotrexate. [31]For labor induction or abortion, it is administered orally, dissolved sublingually, or inserted vaginally.[31,32,33] For prevention of postpartum bleeding it may also be used rectally.[34]

Frequent adverse effects encompass diarrhoea and stomach discomfort. It is classified as pregnancy category X, indicating the drug has been shown to cause adverse effects on the developing baby if administered during gestation. Uterine rupture may occur in infrequent instances mainly in uterine scars (e.g. LSCS, myomectomy etc).[30]

Misoprostol is included in the World Health Organization's List of Essential Medicines.[35] It is offered as a generic version of the medication.[30]

PHARMACODYNAMICS OF MISOPROSTOL:

ROUTE OF ADMINISTRATION	ONSET OF ACTION	DURATION OF ACTION
Oral	8 min	Approx. 2 hrs
Sublingual	11 min	Approx. 3 hrs
Vaginal	20 min	Approx. 4 hrs
Rectal	100 min	Approx. 4 hrs

TABLE 1- Pharmacodynamics of Misoprostol

CLINICAL PHARMACOLOGY OF MISOPROSTOL:

Misoprostol has three principal effects.

1.Cellular protection of the gastrointestinal mucosa.

2.As a uterotonic

3.Gastrointestinal discomfort (diarrhoea and stomach pain are included as undesirable consequences).

Peptic ulcers-

Misoprostol is employed for the prophylaxis of NSAID-induced stomach ulcers. It affects the parietal cells of the gastric tract, obstructing acid from the stomach secretion by G-protein coupled receptor-mediated suppression of adenylate cyclase, resulting in diminished intracellular cyclic AMP levels and diminished proton pumping function at the apical membrane within the parietal cell. In the management of NSAID-induced ulcers, omeprazole demonstrated efficacy comparable to misoprostol, nevertheless the latter was far better absorbed; hence, misoprostol shouldn't be regarded as a treatment of choice.[37]

Standard Adult Dosage for Duodenal Ulcer: 200 mcg taken orally four times day following meals and at bedtime.

-Maintenance dosage: 100 to 200 mcg administered orally four times daily

Standard Adult Dosage for Gastric Ulcer: 200 mcg orally four times day after meals and at sleep.

-Maintenance dosage: 100 to 200 mcg administered orally four times daily

Standard Adult Dosage for NSAID-Induced Ulcer Prophylaxis: 200 mcg administered

orally four times day following meals and at sleep.
-Maintenance dosage: 100 to 200 mcg administered orally four times daily

Labour induction-

Misoprostol is often used for the induction of labor. It stimulates uterine contractions and facilitates the ripening (effacement or thinning) of the cervix. Oxytocin has traditionally served as the primary agent for labor induction; however, its efficacy diminishes if the cervix is not yet mature. Misoprostol can additionally be utilized alongside with oxytocin.[38]

Guidelines from the American College of Obstetricians and Gynecologists (ACOG):
25 mcg administered vaginally every 3 to 6 hours
-Certain people might need dosages of 50 mcg every 6 hours.

Abortions-

Misoprostol is utilized potentially by itself or in combination alongside a different pharmaceutical agent (mifepristone or methotrexate) for medical abortions as an alternative to surgical abortion.[39]
Mifepristone (Mifeprex and generics) is utilized alongside misoprostol for terminating of an intrauterine pregnancy within seventy days of onset of gestation, calculated from the very first day of the last menstrual period. The time frame of pregnancy might be ascertained by means of menstrual history, clinical examination, or an ultrasonographic scan if the duration is ambiguous or if ectopic pregnancy is suspected.[40]
Mifepristone is a progesterone antagonist that terminates pregnancy.[41] Misoprostol is an artificial analog of prostaglandin E1 that terminates pregnancy by promoting the degradation of the lining of the uterus, inducing contractions, and facilitating cervical ripening to aid in the evacuation of the products of conception.
Misoprostol should be delivered 24–48 hours after mifepristone to stimulate uterine contractions.

The American College of Obstetricians and Gynecologists (ACOG) asserts that the medication abortion protocol endorsed by prominent medical organizations globally and nationally comprises mifepristone and misoprostol; should mifepristone be inaccessible, a misoprostol-only regimen is a viable alternative.[40]

Early pregnancy loss-

Regimens based on misoprostol have been thoroughly investigated for the medical treatment of early pregnancy loss. Numerous studies indicate that a higher dosage of misoprostol is more efficacious than a lower dosage, as well as that vaginal or sublingual administration surpasses oral administration in effectiveness, however the sublingual route is linked to a greater incidence of gastrointestinal upset. [42] The most extensive randomized controlled study in the United States revealed that 71% of women experiencing first-trimester pregnancy loss had full expulsion by day 3 following a single dosage of 800 mcg of vaginal misoprostol. [31]

Postpartum bleeding-

Misoprostol serves for the prevention and treatment of postpartum hemorrhage. Misoprostol has been studied for reducing the risk of postpartum hemorrhage and demonstrates clinical effectiveness by oral, vaginal, and rectal administration. Rectally given misoprostol has been documented in several case reports and randomized controlled studies. [43] Nonetheless, it is economical and thermally stable (hence does not need refrigeration like oxytocin to maintain the temperature of drugs), rendering it a cost-efficient and significant medication for usage in the poor countries. [44]

A study was conducted to compare the relative benefits of different uterotonics and following results were obtained.

ASPECT OF PPH PREVENTION	OXYTOCIN	ERGOMETRINE	MISOPROSTOL
Effectiveness	+++	-/?	++
Needs skilled provider	Yes	No	No
Preparation suitable for home birth	No	Yes	Yes
Serious side effects	Rare	Common	Rare
Contraindications	0%	15%	0%
Heat stability	No	No	Yes
Cost	$0.80	$0.30–$0.50	$0.35–$0.50

TABLE 2: Comparison of various uterotonics in treatment of PPH

In India, Oxytocin costs around 100 rupees, Ergometrine on an average costs 30-50 rupees and misoprostol costs an average of 40 rupees respectively.

Misoprostol is endorsed for its affordability, efficacy, and lesser incidence of adverse effects.[45]Misoprostol is heat stable in contrary to oxytocin and ergometrine.

FIGO guidelines for use of misoprostol in abortion and PPH management-

<13 weeks' gestation	13–26 weeks' gestation	>26 weeks' gestation[8]	Postpartum use
Pregnancy termination[a,b,1] 800µg sl every 3 hours *or* pv*/bucc every 3–12 hours (2–3 doses)	**Pregnancy termination**[1,5,6] 13–24 weeks: 400µg pv*/sl/bucc every 3 hours[a,e] 25–26 weeks: 200µg pv*/sl/bucc every 4 hours[f]	**Pregnancy termination**[1,5,9] 27–28 weeks: 200µg pv*/sl/bucc every 4 hours[f,g] >28 weeks: 100µg pv*/sl/bucc every 6 hours	**Postpartum hemorrhage (PPH) prophylaxis**[i,2,10] 600µg po (x1) *or* **PPH secondary prevention**[i,11] (approx. ≥350ml blood loss) 800µg sl (x1)
Missed abortion[c,2] 800µg pv* every 3 hours (x2) *or* 600µg sl every 3 hours (x2)	**Fetal death**[f,g,1,5,6] 200µg pv*/sl/bucc every 4–6 hours	**Fetal death**[2,9] 27–28 weeks: 100µg pv*/sl/bucc every 4 hours[f] >28 weeks: 25µg pv* every 6 hours *or* 25µg po every 2 hours[h]	**PPH treatment**[k,2,10] 800µg sl (x1)
Incomplete abortion[a,2,3,4] 600µg po (x1) *or* 400µg sl (x1) *or* 400–800µg pv* (x1)	**Inevitable abortion**[g,2,3,5,6,7] 200µg pv*/sl/bucc every 6 hours	**Induction of labor**[h,2,9] 25µg pv* every 6 hours *or* 25µg po every 2 hours	
Cervical preparation for surgical abortion[d] 400µg sl 1 hour before procedure *or* pv* 3 hours before procedure	**Cervical preparation for surgical abortion**[a] 13–19 weeks: 400µg pv 3–4 hours before procedure >19 weeks: needs to be combined with other modalities		

Implantation of an intrauterine device

For women with a history of caesarean section or previous failed IUD insertion, administering misoprostol before the procedure can lower the likelihood of unsuccessful IUD placement. However, due to an increased incidence of side effects, routine use of misoprostol for this purpose in women without these specific risk factors is not supported by the evidence. [46]

Additionally, it can be used to prepare the cervical region for an endometrial biopsy, which in turn minimizes the need for a tenaculum or cervical dilator.
There is insufficient data to support the use of misoprostol as a treatment for trigeminal neuralgia in people with multiple sclerosis. [47]

MECHANISM OF ACTION:

Misoprostol directly stimulates the prostaglandin E1 receptors on the parietal cells of the stomach, resulting in a reduction in baseline and overnight gastric acid secretion. This effect diminishes stomach acid production due to stimulation from carbohydrates, alcohol, NSAIDs, histamine, caffeine, and similar substances. A dose-dependent relationship frequently appears for this effect.
Misoprostol promotes the production of mucus and bicarbonate, in addition to oedema of the mucosa and submucosa, leading to the expansion of the mucosal bilayer. This thickening diminishes hydrogen ion reflux and enhances the regulation of mucosal blood flow, hence maintaining the mucosa's ability to produce new cells.
Prostaglandin attaches to smooth muscle cells in the uterine lining, inducing uterotonic activities by causing calcium influx that contribute to its abortifacient effects and its ability to expedite labor and cervical ripening. A reduction in cervical tone due to greater

and more frequent contractions and collagen breakdown in the stroma's connective tissue are the two primary reasons of cervical dilatation. Its uterotonic properties are also beneficial in mitigating postpartum hemorrhage.

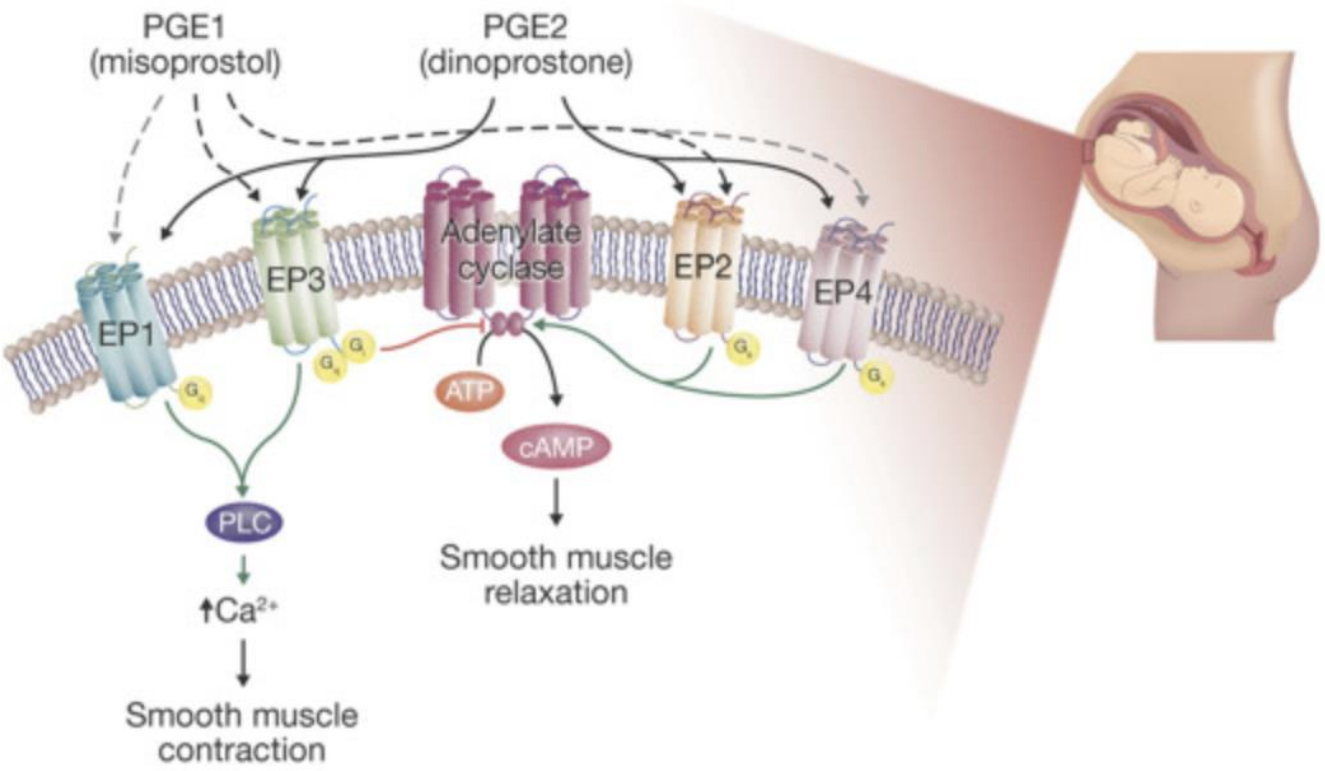

FIGURE 9: Mechanism of action of Misoprostol.

As symptoms seem to be correlated with the plasma concentration of misoprostol acid, it appears that exposure to the misoprostol acid generated during metabolism causes abdominal discomfort and diarrhoea.

ADMINISTRATION:

The only FDA-approved method for administering misoprostol is oral ingestion. However, it can also be delivered sublingually, buccally, vaginally, or rectally through the placement of tablets or suppositories, although these routes are not FDA-approved.[55]

To reduce gastrointestinal discomfort, it is best to take misoprostol orally at night with food. Magnesium-based antacids should be avoided, as they can worsen the drug's diarrheal side effects.[55]

ADVERSE EFFECTS:

1. Diarrhoea
2. Pain Abdomen
3. Nausea
4. Flatulence
5. Headache
6. Dyspepsia
7. Constipation
8. Vomiting
9. Cramps
10. Spotting
11. Hypermenorrhoea
12. Dysmenorrhea

MISOPROSTOL DOSAGE AND ADMINISTRATION

INDICATIONS DOSES

INDICATIONS	DOSES
NSAID ulcer prophylaxis	200 mcg × 4 times
Induced abortion (0-12 wks)	800 µg vaginal 12 hrly × 3 times
Missed abortion (0-12 wks)	800 µg vaginal 13 hrly or sublingual 600 µg 3 hrly
Incomplete abortion (0-12 wks)	600 µg single oral dose
Induced abortion (13-22 wks)	400 µg vaginal 3 hrly × 5 times
Intrauterine fetal death	13-17 wks:200 µg PV 6th hrly;18-26 wks:100 µg PV 6th hrly; >27wks:25-50µg PV 4th hrly
Induction of labor	25 µg vaginal 4 hrly or 50 µg oral 4 hr or 20 µg oral solution 2 hrly
PPH prophylaxis	600 µg oral or sublingual stat

TABLE 3: Misoprostol dosage and indications

CONTRAINDICATIONS:

In people who have experienced prior allergic reactions or prostaglandin hypersensitivity, misoprostol is not recommended. NSAID users who are at risk for developing stomach ulcers. Aside from allergic reactions, misoprostol is not contraindicated for use in gynecology or obstetrics. Contraindications are specific to the medication's intended impact and should be considered for each patient's risk factors. Misoprostol, which has a higher risk of uterine rupture, should not in those who have had prior Caesarean sections for medical abortions.

ROLE OF MISOPROSTOL IN CERVICAL RIPENING AND LABOUR INDUCTION:

❖ DRUG MONITORING

Misoprostol is an effective and well-accepted pharmaceutical agent. At present, there are no defined monitoring protocols for its FDA-sanctioned use. Similarly, its applications in obstetrics and gynaecology are not governed by standardized protocols. However, fetal monitoring is recommended when it is used for labor induction.

❖ TOXICITY

Misoprostol frequently serves as a safe and effective medication, and the toxic dose remains undetermined.

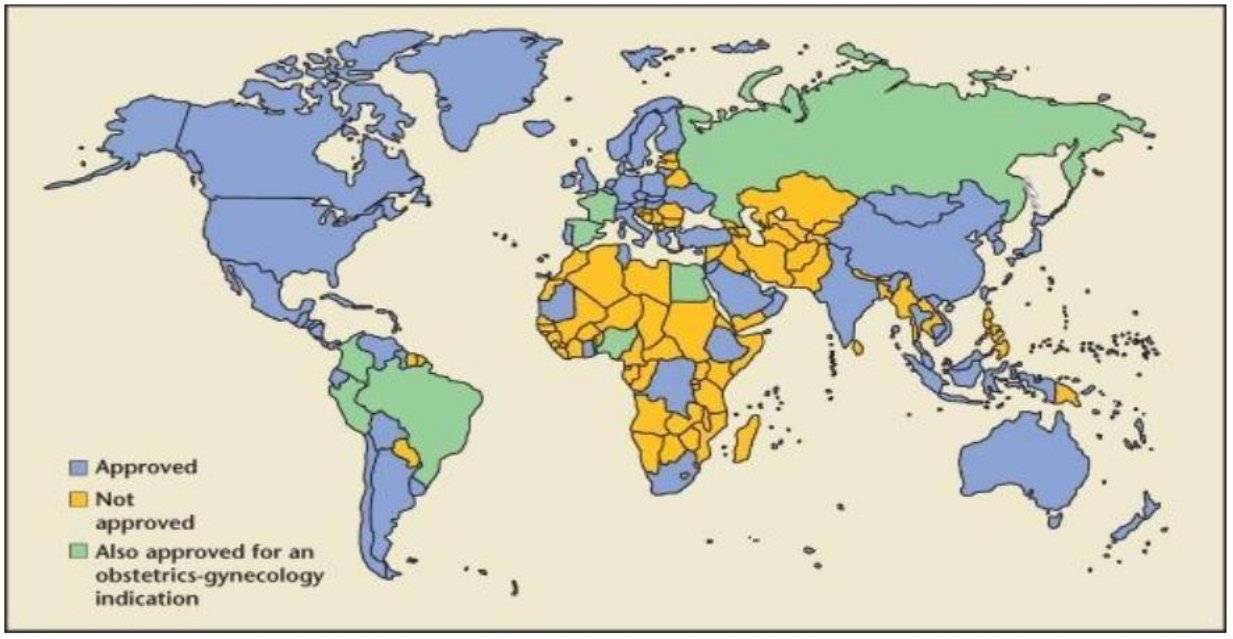

FIGURE 10: WORLD MAP OF MISOPROSTOL APPROVAL

PHARMACOKINETIC CHARACTERISTICS OF VARIOUS ADMINISTRATION ROUTES OF MISOPROSTOL

Misoprostol can be administered by oral, vaginal, sublingual, buccal, or rectal routes. Pharmacokinetic research comparing oral and vaginal delivery indicate that vaginal misoprostol exhibits delayed absorption, reduced peak plasma concentrations, and delayed clearance, akin to an extended-release formulations. [49-51]

Vaginal misoprostol is linked to increased total drug exposure (area under the curve [AUC]) and stronger impacts on the cervix and uterus. [48]The mechanism of absorption of misoprostol via the vaginal epithelium varies significantly across people. [52]

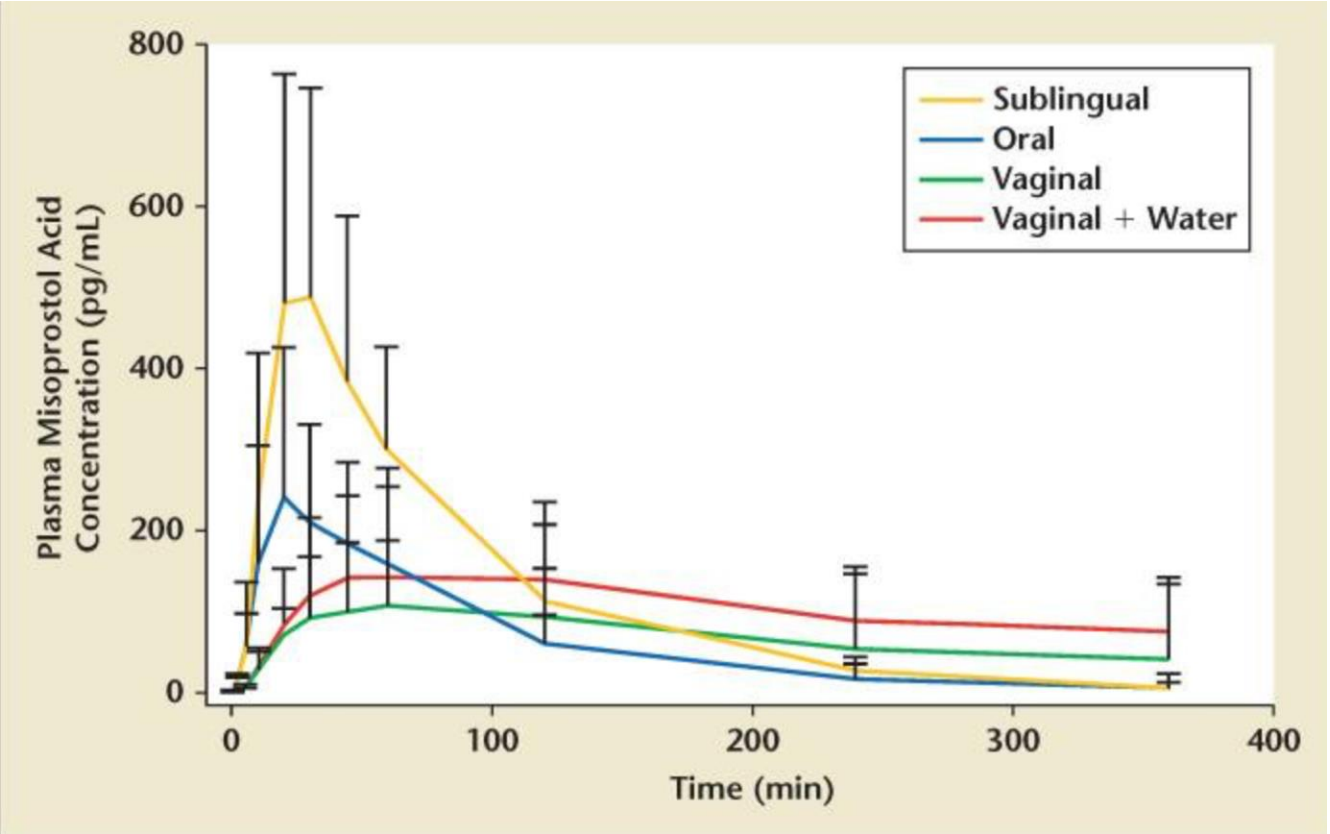

FIGURE 11: Pharmacokinetics of different methods of misoprostol

The rectal route of administration has a comparable profile to vaginal administration, although demonstrates a reduced AUC, including a substantially reduced maximum peak concentration. [52] The sublingual delivery method has an AUC comparable to that of vaginal administration, although it demonstrates more fast absorption and elevated peak levels than both vaginal and oral routes. [52] This results in increased occurrences of gastrointestinal adverse effects. Nonetheless, the sublingual route induces uterine contractions at a pace comparable to vaginal dosing and has reduced variability in absorbing.

There is no clinically meaningful distinction between dry vaginal misoprostol and vaginal misoprostol wet with water, salt, or acetic acid. [52 to 54]

TERATOGENIC EFFECTS

Misoprostol is classified as a teratogen. Congenital anomalies resulting from prenatal exposure to misoprostol during early gestation including cranial abnormalities, bladder exstrophy, arthrogryposis, cranial nerve palsies, facial deformities, terminal transverse limb defects, and Moebius sequence.. [55,56,57] These congenital malformations are thought to arise from vascular disruptions triggered by uterine contractions induced by misoprostol. Nonetheless, research on population registries indicates that the occurrence of such abnormalities is relatively rare, even in groups where misoprostol use is widespread. [52,58]

The overall risk of congenital abnormalities following prenatal exposure to misoprostol is estimated to be around 1%.

Pharmacokinetic research indicates that misoprostol is secreted into breast milk, with its concentrations rising and declining swiftly.

The medication is no longer detectable in breast milk within 5 hours of maternal consumption. [59] Breastfeeding mothers should be informed that misoprostol may induce diarrhoea in infants.[60]

DINOPROSTONE

Prostaglandin E2 was initially created in 1970 by American scientist E.J. Corey and received FDA approval for medicinal usage in the US in 1977.

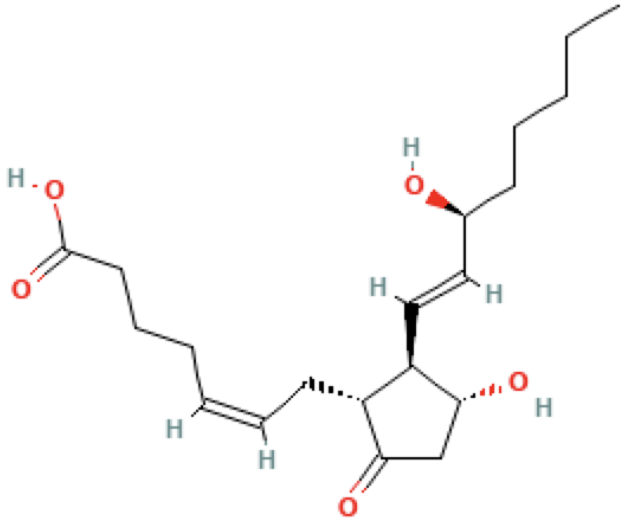

FIGURE 12: Chemical structure of Dinoprostone

Dinoprostone or Prostaglandin E2 (PGE2) is a naturally produced substance having oxytocic capabilities employed as a pharmaceutical agent.[61] PGE2 synthesis in the body begins when phospholipase A2 activates arachidonic acid (AA).

The activated arachidonic acid is then oxygenated by cyclooxygenase (COX) enzymes to produce prostaglandin endoperoxides. In this process, prostaglandin G2 (PGG2) is turned into prostaglandin H2 (PGH2) by the peroxidase activity of the COX enzyme, and PGH2 is then changed into PGE2. [62,63]

PHYSIOLOGICAL EFFECTS-

Dinoprostone is crucial in labor as it facilitates cervical softening and triggers uterine contractions

It further facilitates the secretion of substances from osteoblasts that enhance the breakdown of bones by osteoclasts.[64]

Endogenous prostaglandins such as PGE1 and PGE2 are essential for the anatomical integrity and functionality of the ductus arteriosus in fetuses and neonates. They facilitate the maintenance of the ductus arteriosus, allowing blood to circumvent the fetus's lungs that are underdeveloped and reroute to the placenta for oxygenation.[65] The ductus arteriosus frequently commences closure postnatally as a result of enhanced metabolism of PGE2. In neonates with congenital cardiac disease, prostaglandins are administered to maintain the patency of the ductus arteriosus, hence ensuring adequate oxygen concentration in the bloodstream.[62,65]

PGE2, like PGE1, functions as an intrinsic vasodilator by influencing smooth muscle to induce the dilatation of blood vessels. PGE2 also suppresses aggregation of platelets. [62] PGE2 inhibits T cell receptor signaling and proliferation, perhaps aiding in the resolution of inflammation.[62-66]It diminishes the immunological response by obstructing B-lymphocyte differentiation and their ability to deliver antigens.[66]

Prostaglandin E2 (PGE2) has several effects on the central and peripheral nerve systems. It raises body temperature and develops fever by binding to EP3 receptors. PGE2 plays an essential role in inflammation by facilitating edema and proliferation of leukocytes through enhanced vascular permeability, thus boosting blood flow to inflamed regions via

EP2 receptors. Nonsteroidal anti-inflammatory medications (NSAIDs) inhibit COX-2, hence diminishing PGE2 synthesis and alleviating fever and inflammation.[62]

Effect on Central and peripheral nervous system

Prostaglandin E2 (PGE2) has various effects on both the central and peripheral nervous systems. By interacting with EP3 receptors, it raises body temperature, causing fever. PGE2 also plays a key role in inflammation by promoting oedema and the infiltration of leukocytes through increased vascular permeability, which allows more blood to reach inflamed areas, via EP2 receptors. Nonsteroidal anti-inflammatory drugs (NSAIDs) reduce PGE2 production by blocking COX-2, thereby helping to reduce fever and inflammation.[62]

Additionally, PGE2 interacts with EP1 and EP4 receptors, contributing to pain perception via inflammatory sensory perception. [67]

Neurological effects

Prostaglandin E2 (PGE2) is implicated in several inflammatory and immunological pathways in reaction to both physical and psychological stress. PGE2, one of the most prevalent prostaglandins in the body, has a significant role in typical inflammatory manifestations, including erythema, edema, and nociception. It modulates these actions by interacting with G protein-coupled prostaglandin E2 receptors (EP1, EP2, EP3, and EP4). The stimulation of these receptors is contingent upon the particular stressor and results in varied stress responses. When PGE2 activates EP1, it suppresses impulse behaviours triggered by psychological stress. PGE2 also influences memory impairment during illness through EP2 activation. EP3 activation by PGE2 helps regulate fever caused by illness, while EP4, similar to EP2, is involved in hypothermia and anorexia. In addition to its pro-inflammatory effects, PGE2 also has anti-inflammatory actions, depending on which receptor it activates.[68,69]

Immunity

PGE2, an important homeostatic factor, is also a key mediator in the immune response to chronic infections and cancer. Its effects depend on the balance between its COX-2-regulated production, 15-hydroxyprostaglandin dehydrogenase-mediated breakdown, and the expression pattern of its receptors. PGE2 enhances its own synthesis while inhibiting acute inflammatory mediators, making it more prominent in the later or chronic phases of immunity.

It facilitates dendritic cell activation but diminishes their capacity to attract naïve, memory, and effector T cells. PGE2 selectively dampens the functions of macrophages, neutrophils, and type 1 immune responses (like Th1, CTL, and NK cells), while promoting Th2, Th17, and regulatory T cell responses. It also influences chemokine production, limiting the attraction of proinflammatory cells and encouraging the accumulation of regulatory T cells and myeloid-derived suppressor cells. Targeting PGE2 production, degradation, and receptor activity could offer therapeutic approaches to regulate immune responses in conditions ranging from autoimmunity to cancer. [62,70]

Effect on smooth muscles

Prostaglandin E2 (PGE2) is key in regulating vascular smooth muscle tone. Produced by endothelial cells, it acts as a vasodilator by raising cyclic adenosine monophosphate (cAMP) levels, which reduces intracellular calcium through the activation of IP and EP4 receptors, leading to smooth muscle relaxation.[62]

PGE2 regulates vascular smooth muscle tone by receptor activation. Vasoconstriction elicited by PGE2 following the activation of the EP1 and EP3 receptors is caused by the stimulation of intracellular Ca2+ channels or reductions in cAMP, accordingly.[71]

Effects on renal system

Prostaglandin E2 (PGE2), in along with other prostaglandins, is synthesized in the renal cortex and medulla. Its function in the kidney, originating from COX-2, is to facilitate renal blood flow and glomerular filtration rate (GFR) by localized vasodilation. Prostanoids generated from COX-2 enhance medullary blood flow and diminish salt reabsorption in renal tubules. Furthermore, PGE2 helps control systemic blood pressure by regulating the excretion of water and sodium. It is believed to activate EP4 or EP2 receptors, stimulating renin release, which increases GFR, enhances sodium retention, and raises systemic blood pressure.[62]

Dinoprostone for ripening of cervix

Cervical ripening, or cervical effacement, is a natural process occurring before to labor, during which the cervix softens, thins, and dilates, easing the passage of the fetus through the cervix.[61]A mature cervix is desirable ahead of labor induction, since it enhances the likelihood of a successful induction. When cervical ripening does not occur spontaneously, pharmacological interventions are occasionally employed to facilitate the process.[23]

Cervical ripening naturally occurs through prostaglandins, such as PGE2 or dinoprostone, and hence are are commonly used as a pharmacological method.[61]

A comprehensive study and meta-analysis found that outpatient cervical ripening with dinoprostone or single-balloon catheters did not elevate the probability of cesarean births.[72]

Dinoprostone for termination of pregnancy

PGE2 is commonly used as a pharmacological method for pregnancy termination, especially in the in cases of missed abortion, where the fetus remains in the uterus despite the miscarriage. [73,74] Dinoprostone (PGE2) is not directly feticidal; rather, it promotes abortion by inducing uterine contractions. It is typically administered as a 20 mg vaginal

suppository every 3 to 5 hours to help expel the pregnancy tissue. The abortion is expected to occur within 24 hours of starting the treatment. If the procedure is not completed within this time frame, further methods, such as dilation and curettage (D&C), may be needed to finish the process. Dinoprostone may sometimes be combined with other drugs like misoprostol to enhance its effectiveness. This regimen is generally used for medical abortions during the early stages of pregnancy, though dosages and protocols may vary depending on the clinical situation and patient needs. [62,75]

ADVERSE EFFECTS :

Nausea

Vomiting

Diarrhoea

Headache

Shivering

Chills

Fever

Uterine hyperstimulation

Fetal distress

MECHANISM OF ACTION:

Prostaglandin E2 (PGE2) works by binding to specific G protein-coupled receptors (GPCRs)—EP1, EP2, EP3, and EP4—on cells in various tissues, including the uterus. This binding activates a signaling cascade that leads to an increase in intracellular calcium levels, resulting in uterine contractions. This mechanism is why PGE2 and its synthetic forms, like dinoprostone, are used in medical abortions to induce uterine contractions and expel pregnancy tissue.

In the kidneys, PGE2 has a role in regulating fluid balance by inhibiting sodium (Na+) absorption in the Thick Ascending Limb (TAL) of the Loop of Henle and reducing the effect of antidiuretic hormone (ADH) in the collecting tubules, thus promoting diuresis (increased urine production).

When NSAIDs inhibit PGE2 synthesis by blocking cyclooxygenase (COX) enzymes, this can reduce the effectiveness of loop diuretics (like furosemide). PGE2 helps loop diuretics work by enhancing sodium excretion and limiting water reabsorption, so reducing PGE2 levels can interfere with these effects, making the diuretics less effective at promoting fluid and sodium loss.[75]

In summary, while PGE2 is known for causing uterine contractions during abortion, it also plays a significant role in kidney function. When NSAIDs block its production, they can decrease the efficacy of loop diuretics, which depend on PGE2's action for their diuretic effects. This interaction emphasizes the need to consider drug interactions and their broader impacts on multiple organ systems.[75]

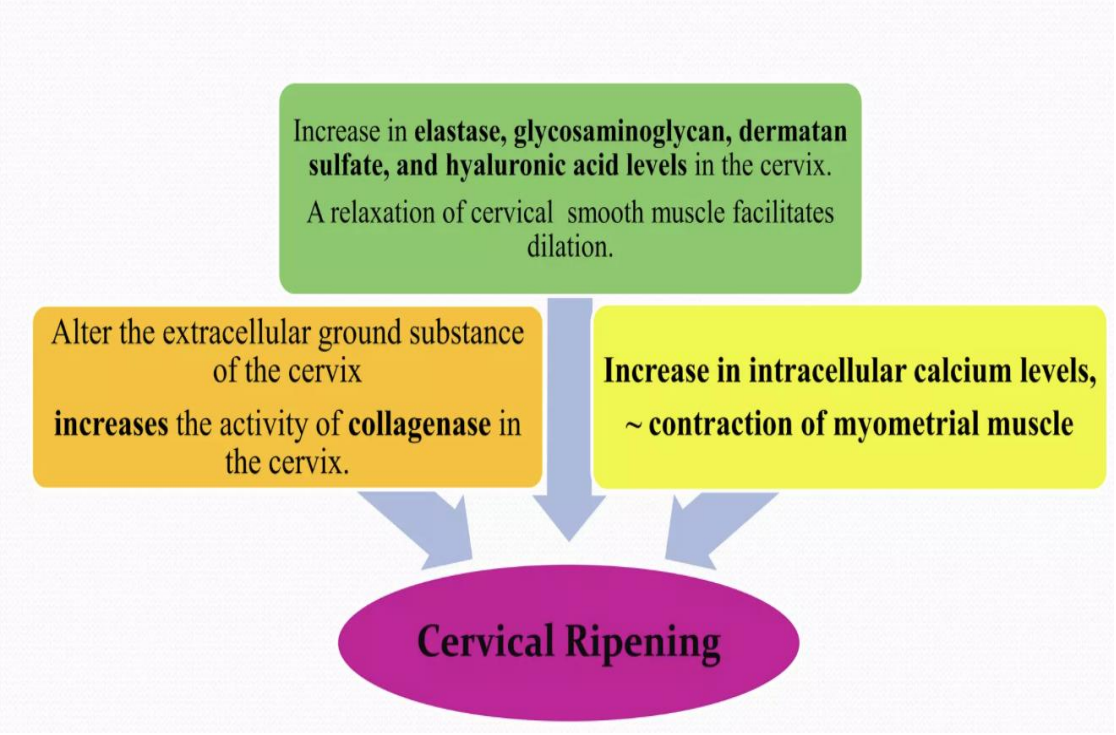

FIGURE 13: Mechanism of action of Dinoprostone

CONTRAINDICATIONS :

In case where spontaneous vaginal deliveries are contraindicated.

- Allergies
- Acute PID or active disease of the cardiovascular, respiratory, hepatic, or renal systems.
- History of cervical cancer, hypotension or hypertension, anemia, epilepsy, jaundice, asthma, or pulmonary disorders.
- Previous history of cesarean sections or significant uterine surgery.
- Fetal distress
- Unexplained vaginal bleeding during pregnancy.
- History of challenging labors and births, presence of cephalopelvic disproportion, less than six prior term infants with nonvertex presentation, and hypertonic or hypotonic uterine patterns.[75]

PHARMACOKINETICS OF DINOPROSTONE

Dinoprostone, a synthetic form of PGE2, possesses a plasma half-life of around 2.5–5 minutes following vaginal treatment, with the majority of metabolites eliminated via urine. [62]

MATERIALS AND METHODS

STUDY SETTINGS:

Patients admitted to the "Department of Obstetrics and Gynecology of BLDE (DEEMED TO BE UNIVERSITY) SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA" for induction of labor fulfilling the inclusion criteria.

Complete written consent was acquired from the patients after full disclosure of the study to them.

Period of study -APRIL 2023 TO MARCH 2025

STUDY DESIGN:

A PROSPECTIVE INTERVENTIONAL STUDY

METHOD OF COLLECTION OF DATA:

Inclusion Criteria:

Singleton, term pregnancy (>37 weeks) with fetus in cephalic presentation and no signs of labour before induction of labour.

Exclusion Criteria:

All contraindications to vaginal delivery -

1.Placenta previa or vasa previa.

2.Surgical procedures on uterine body (Previous LSCS, Intrapartum uterine rupture and myomectomy).

3.Invasive cancer of cervix .

4.Active genital herpes infection .

5.Malpresentations and malpositions.

6.PROM.

SAMPLE SIZE CALCULATION :

SAMPLE SIZE – 106 patients.

With Anticipated Proportion of Caesarean section among MVI group 40.60 % and among DVI group 20.18% (ref) respectively,. the study would require a total sample size of 106. (i.e. a total sample size of 53 for MVI group and 53 for DVI group), to achieve a power of 90% for detecting a difference in proportions between two groups at a two sided p-value of 0.05 .

Formula used

• $n = \frac{(z\alpha + z\beta)2\ 2\ p*q}{MD2}$

Where Z= Z statistic at a level of significance

MD= Anticipated difference between two proportions

P=Common Proportion

q= 100-p

Statistical Analysis

• The collected data is entered into a Microsoft Excel spreadsheet, and statistical analysis is conducted using the Statistical Package for the Social Sciences (Version 20)

• Results is be displayed as Mean±SD, counts, percentages, and graphs.

• Normally distributed continuous variables between two groups are compared using an Independent t-test. The Mann-Whitney U test is employed for variables that are not

regularly distributed. Categorical variables between two groups are analyzed using the Chi-square test.

• Odds ratio and 95% confidence interval The interval are computed to determine the correlation between two groups.

• A p-value of less than 0.05 are deemed statistically significant. All statistical tests are conducted as two-tailed.

METHODOLOGY:

This is a single blinded randomised prospective and interventional study.
Every patient who meet the inclusion criteria and consent to participate in the study will be examined and categorized into two groups.
Detailed history, examination, investigation, and monitoring will be done as per the hospital protocol.

Group 1- Tablet Misoprostol 25mcg will be vaginally inserted in the patients (including PROM). The dose is repeated every 4th hourly.
Group 2 - Dinoprostone 10mg vaginal insert will be used in the patients . Reassessment is done every 12 hours.

This study is to compare the two drugs and their role in induction of labor.

Prostaglandins in the form of vaginally inserted tablet Misoprostol and Dinoprostone insert will be used in the patients. It will be inserted high up into the posterior vaginal fornix.

The vaginal tablet will be repeated 4^{th} hourly and insert will be removed after a maximum of 24hrs or when active phase of labour begins (regular contractions with cervical

dilatation >4 cms)[2]. Dinoprostone insert will also be removed in case of membranes rupture .

Induction of labour was considered ineffective when oxytocin infusion lasted for 12 hrs with no active phase of labour.

Arrested labour was defined as 4 hours no progression of cervical dilatation or no descent of head or rotation of head for 2 hours in second stage of labour.

This study is to compare percentage of vaginal delivery to caesarean sections and vacuum assisted births. Also, to compare the outcome in terms of meconium stained liquor, postpartum haemorrhage and neonatal outcome.

RESULTS AND ANALYSIS

A total of 106 women who came to Department of Obstetrics and Gynaecology at Shri B. M. Patil Medical College Hospital and Research Centre, Vijayapura with Singleton, term pregnancy (>37 weeks) with fetus in cephalic presentation and no signs of labour before induction of labor. They were divided into two equal (53 each) group as per computer generated randomisation (www.randomizer.org) and were given the prostaglandins according to the study criteria.

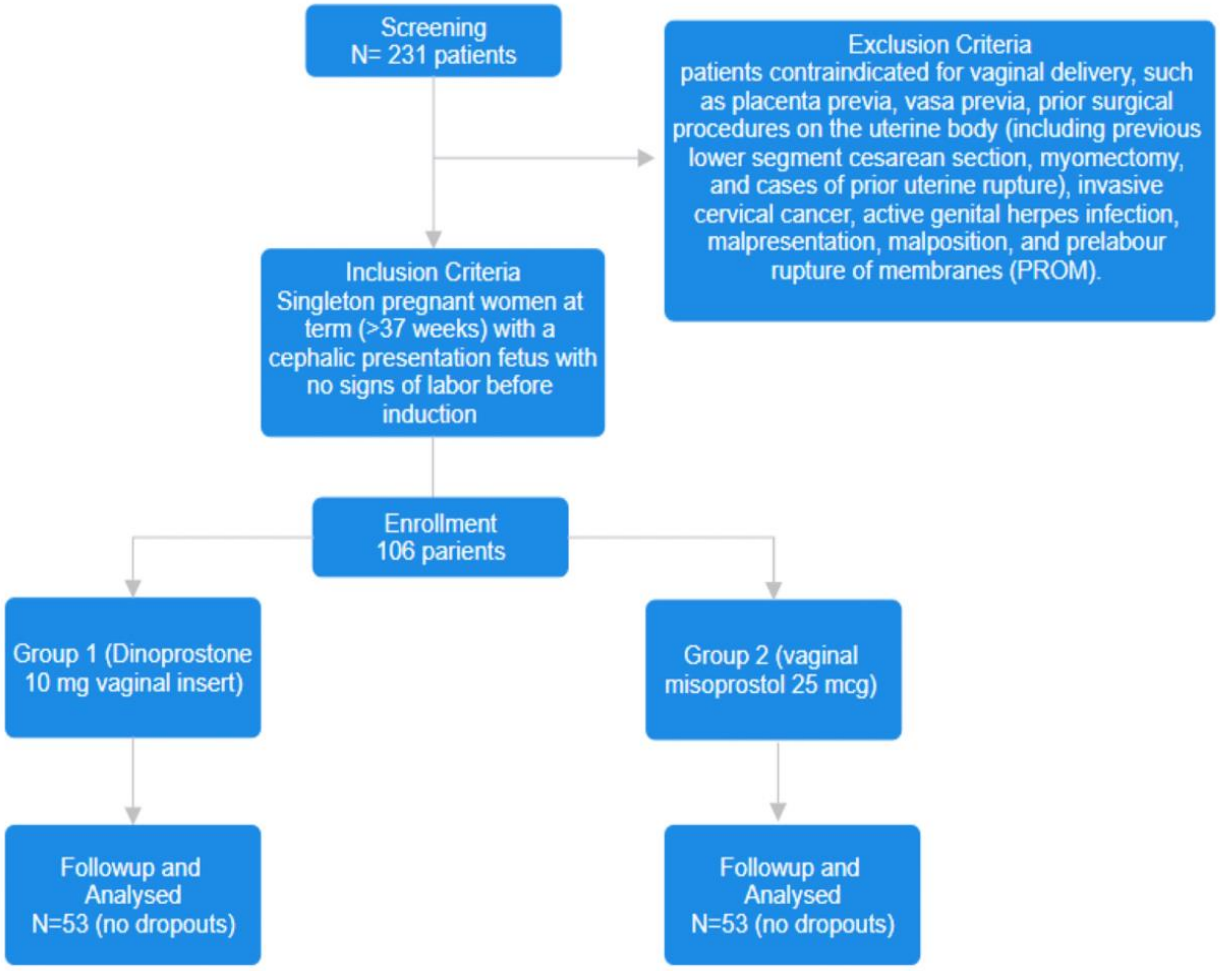

TABLE 3: DISTRIBUTION OF CASES ACCORDING TO MATERNAL AGE IN BOTH GROUPS

Age Groups (Years)	Dinoprostone (53)		Vaginal misoprostol (53)		
	Number	Percentage	Number	Percentage	p-value
≤ 20	10	18.9 %	11	20.8 %	0.695
21-30	39	73.6%	40	75.5%	
≥ 31	4	7.5%	2	3.8 %	

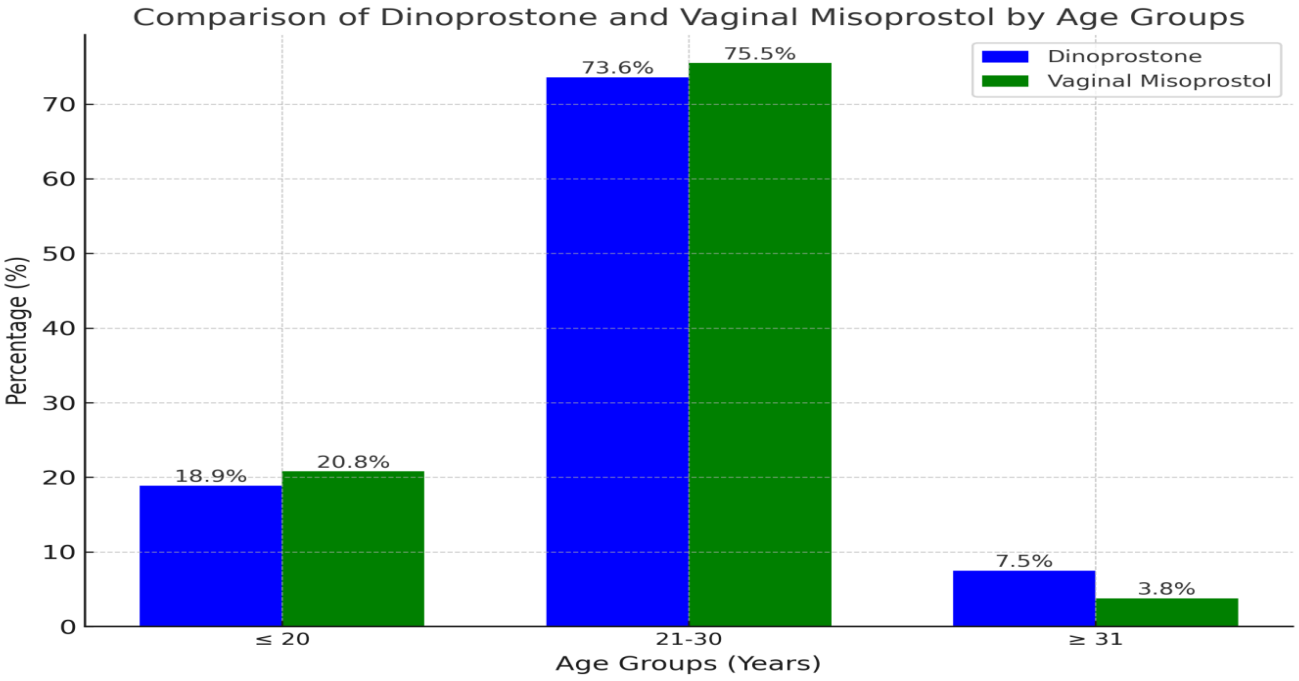

Figure 14: Age distribution bar char

In the Dinoprostone cohort, 10 (18.9%) patients were aged ≤20 years, 39 (73.6%) patients were aged 21-30 years, and 4 (7.5%) patients were aged ≥31 years.

In the Misoprostol group, 11 patients (20.8%) were aged ≤20 years, 40 patients (75.5%) were aged 21-30 years, and 2 patients (3.8%) were aged ≥31 years.

The correlation between maternal age in both groups was not statistically significant.

In both the study groups, most patients belonged to age 21-30 years.

TABLE 4: PARITY IN BOTH STUDY GROUPS

Parity	Dinoprostone(53)		Vaginal misoprostol (53)		
	Number	Percentage	Number	Percentage	p-value
Primiparous	26	41.9	28	52.8	0.698
Multiparous	27	50.9	25	47.2	

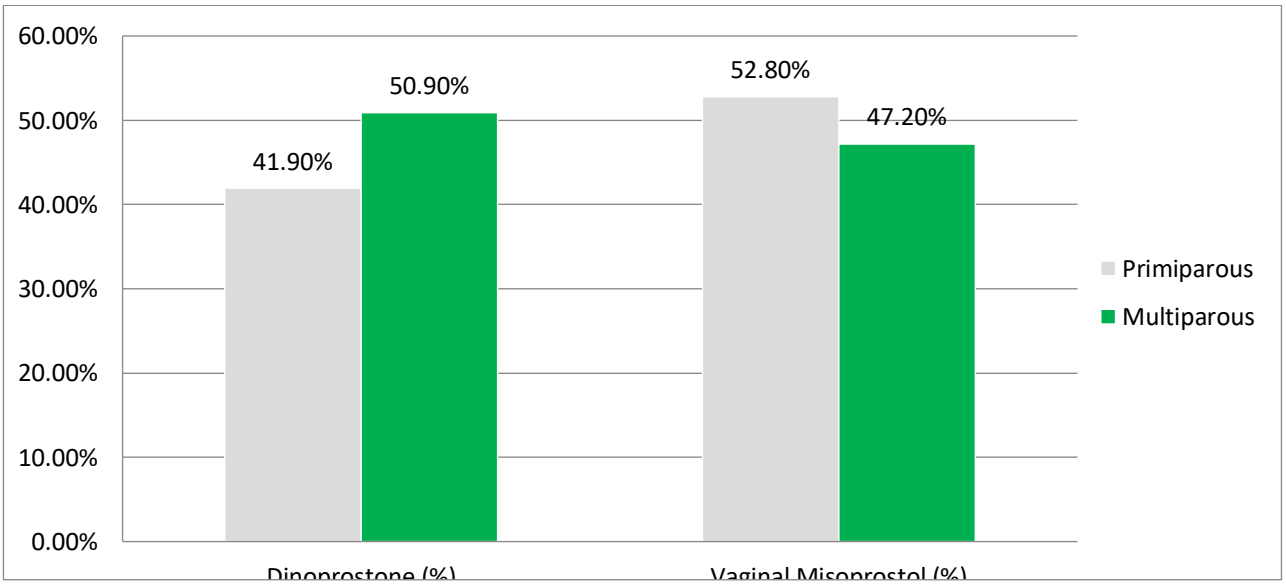

Figure 15: Parity distribution bar chart

In Dinoprostone group, 26 (41.9%) patients were Primiparous and 27 (50.9%) patients were multiparous. In the Misoprostol group, 28 (52.8%) patients were primiparous and 25 (47.2%) patients were multiparous.The parity in both the groups was found to be statistically insignificant.

TABLE 5: COMPARISON OF MEAN GESTATIONAL AGE

	Number	Mean	SD	Minimum	Maximum	Median	p-value
Dinoprostone	53	39.5	1.15	37	41	40	
Vaginal misoprostol	53	39	1.13	37	41	39	**0.29**

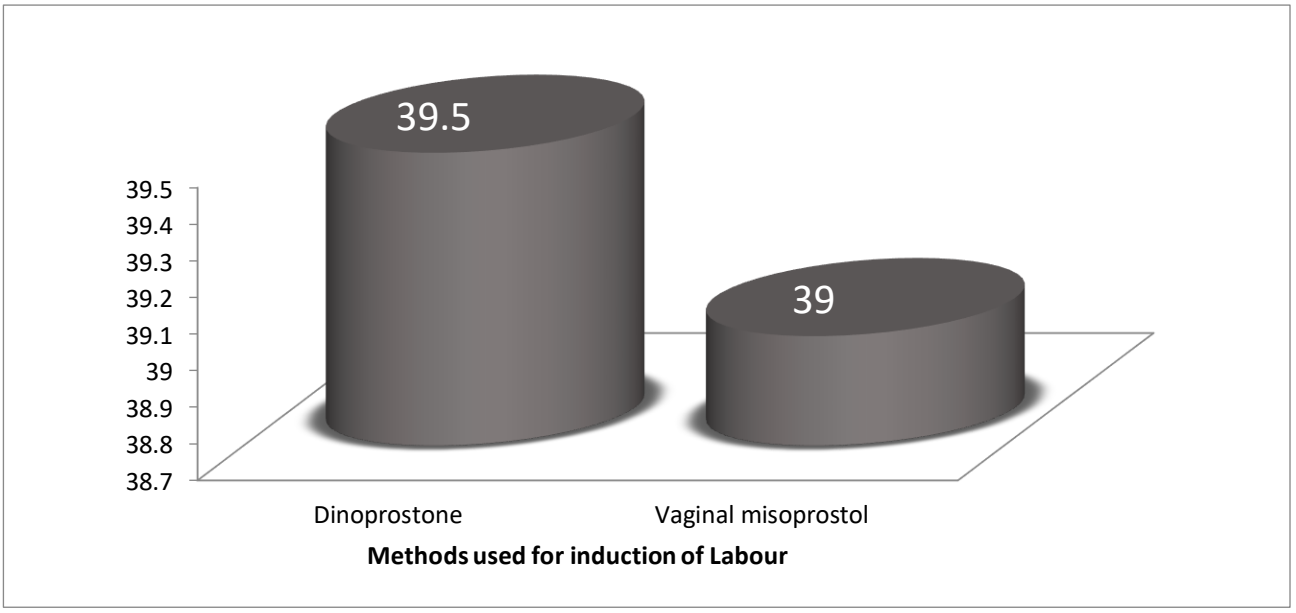

Figure 16: Mean Gestational Age distribution Chart in Both Groups

In the Dinoprostone group, the mean gestational age (mean ± SD) of patients was 39.5 ± 1.15 weeks. In the Misoprostol group, the mean gestational age (mean ± SD) of patients was 39 ± 1.13 weeks. The mean Gestational Age distribution among groups was not statistically significant (p=0.29).

TABLE 6: MODE OF DELIVERY (MOD) IN BOTH GROUPS

MOD	Dinoprostone (53)		Vaginal misoprostol (53)		
	Number	Percentage	Number	Percentage	p-value
Vaginal Delivery	39	73.58	27	50.94	
LSCS	14	26.42	25	47.17	
Assisted Vaginal Delivery	0	0	1	1.89	0.02

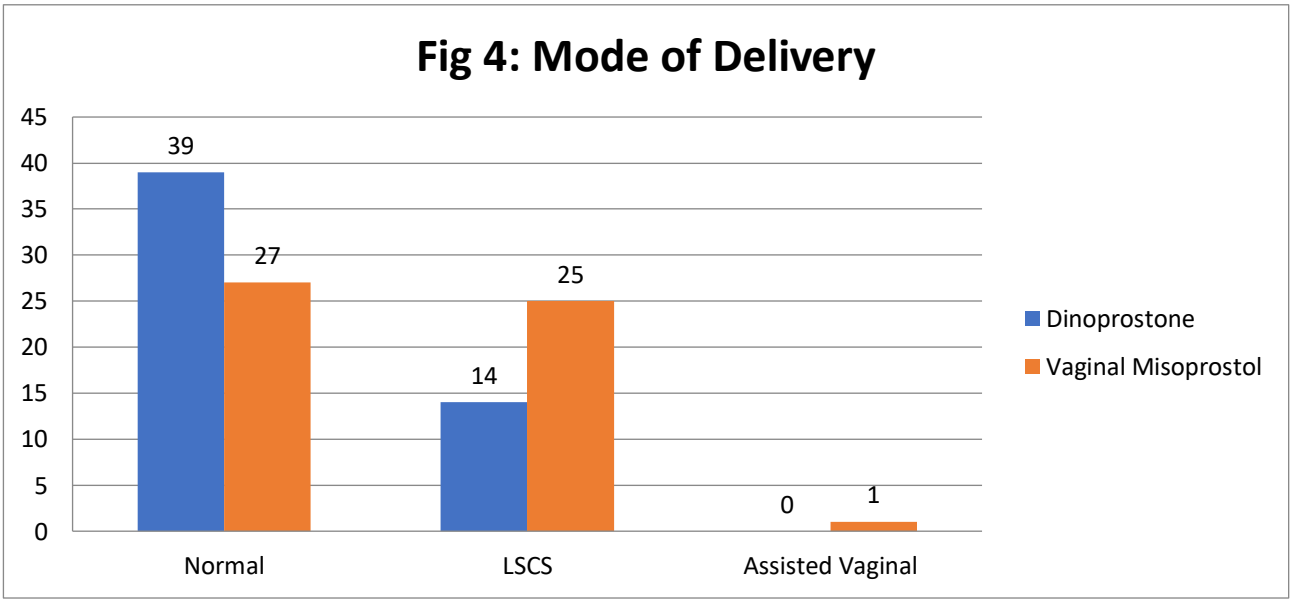

Figure 17: Mode of delivery bar chart

In Dinoprostone group, 14 (26.42 %) patients underwent LSCS and 39(73.58 %) patients underwent Vaginal Delivery. In the Misoprostol group, 25 (47.17%) patients underwent LSCS ,27 (50.94 %) patients underwent Vaginal Delivery and 1 (1.89%) patients

underwent Assisted Vaginal delivery.Hence, Dinoprostone group showed more vaginal deliveries and was statistically significant.

TABLE 7: INDICATION OF INDUCTION IN BOTH STUDY GROUPS

Indication of Induction	Dinoprostone (53)		Vaginal misoprostol (53)	
	Number	Percentage	Number	Percentage
IUGR	0	0	4	7.5
PIH	6	11.3	7	13.2
Cholestatic Jaundice	1	1.9	0	0
Gestational Diabetes Mellitis	1	1.9	3	5.7
Postdated	31	58.5	21	39.6
Others	14	26.4	18	34.0

(p-value):0.131

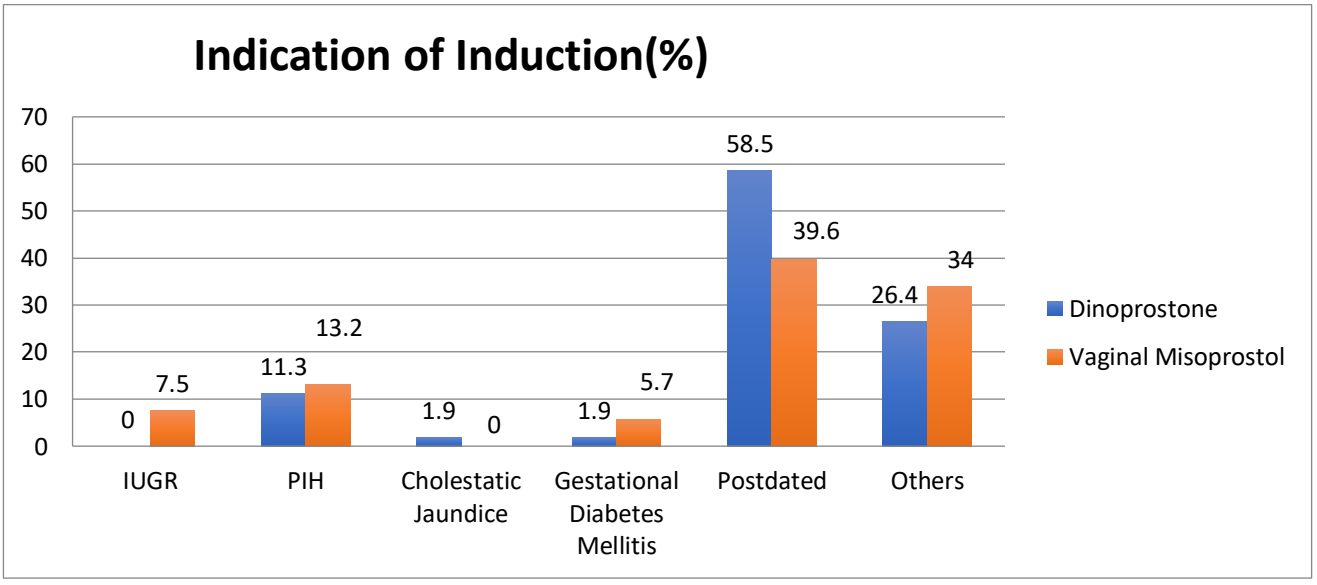

Fig 18: Indication of Induction in both groups

In Dinoprostone and Misoprostol groups, the most common indication for induction was found to be postdatism. Hence, it was found to be statistically insignificant.

TABLE 8: INDICATION OF LSCS IN BOTH GROUPS

	Dinoprostone (13)		**Vaginal Misoprostol(26)**	
INDICATION OF LSCS	**Number**	**Percentage**	**Number**	**Percentage**
2nd stage arrest	0	0.00	1	3.85
Fetal distress	2	15.38	12	46.15
MSL	1	7.69	2	7.69
NPOL	0	0.00	2	7.69
Thick MSL	1	7.69	2	7.69
Antepartum eclampsia	0	0.00	1	3.85
Failed induction with non resurring NST	1	7.69	3	11.54
Gestational hypertension	1	7.69	0	0.00
Imminent eclampsia	2	15.38	0	0.00
Non ressuring nst with fetal distress	0	0.00	1	3.85
Nonreasuring nst	1	7.69	1	3.85
Pe wout severe features	1	7.69	0	0.00
Persistant occipitoposterior with fetal distress	1	7.69	0	0.00
Persistant tachycardia with Fetal tachycardia	1	7.69	0	0.00
Persistant uterine tachysystole	1	7.69	0	0.00

Thin MSL	0	0.00	1	3.85
Total	13	100	26	100

(**p-value):**0.083

In Dinoprostone group : 2 (15.38 %) patients indication for LSCS was fetal distress , In 1 (7.69 %) patient had failed induction with non reassuring NST, and in 1 (7.69% %) patient has persistent uterine tachysystole and underwent LSCS.

In Misoprostol group: 12 (46.15 %) patients indication for LSCS was fetal distress and 3 (11.54 %) patients had failed induction with non reassuring NST, underwent LSCS.

TABLE 9: OCCURRENCE OF MECONIUM-STAINED LIQUOR

Meconium stained liquor	Dinoprostone(53)		Vaginal misoprostol (53)		
	Number	Percentage	Number	Percentage	P -value
Present	6	11.3	13	24.5	0.076

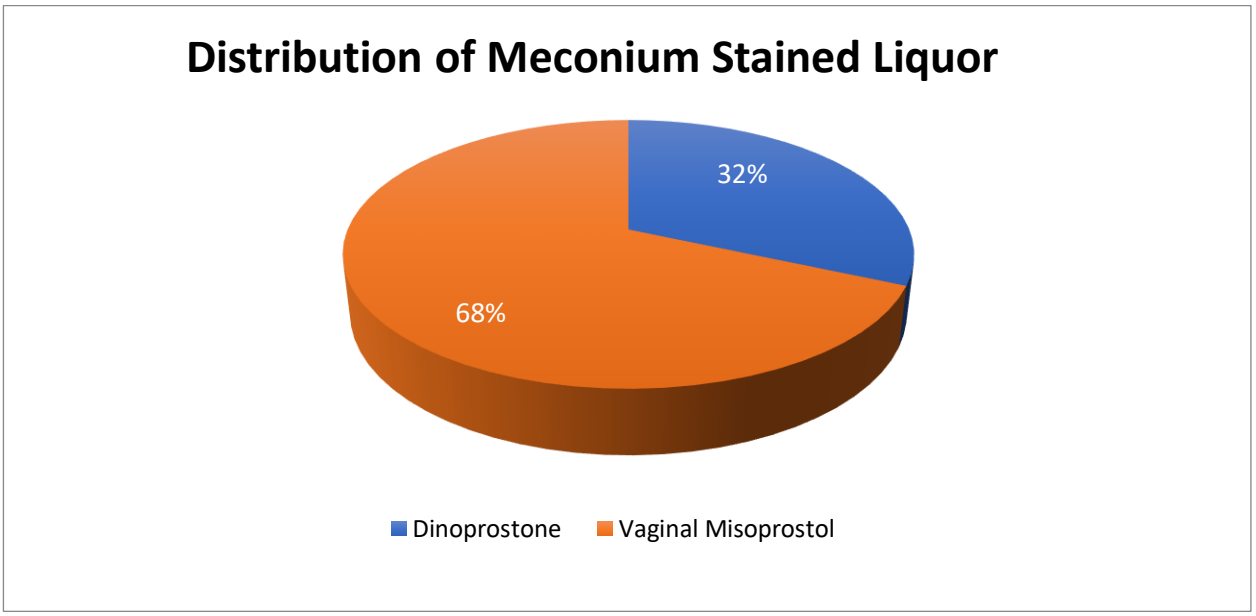

Figure 19: Meconium-stained liquor pie chart

Within the Dinoprosne group, 6 patients (11.3%) had meconium-stained liquid.
In the Misoprostol cohort, 13 (24.5%) individuals had meconium-stained amniotic fluid. The correlation between Meconium-stained liquor and the groups was not statistically significant (p=0.076).

TABLE 10: INCIDENCE OF NICU ADMISSION

NICU Admission	Dinoprostone(53)		Vaginal misoprostol (53)		
	Number	Percentage	Number	Percentage	p-value
Present	9	16.98	25	47.17	**<0.001**

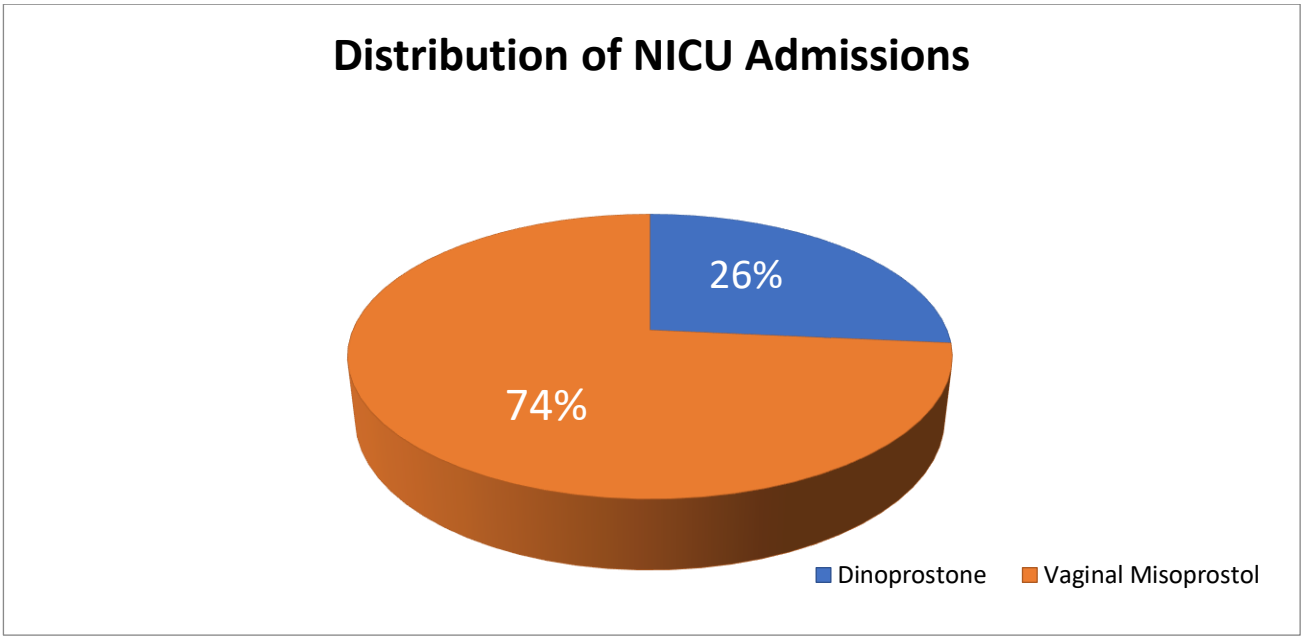

Figure 20: NICU admission chart

In Dinoprostone group, 9 (16.98%) babies were taken for NICU admission. In the Misoprostol group, 25 (47.17 %) babies were taken for NICU admission. The incidence of NICU Admission was statistically significant (p=<0.001).

TABLE 11: DISTRIBUTION OF FINAL MODIFIED BISHOP'S SCORE IN TWO GROUPS

	Number	Mean	Mode	SD	Minimum	Maximum	Median	p-value
Dinoprostone	53	10.3	12	2.66	4	13	10	
Vaginal misoprostol	53	10	10	2.41	5	13	10	0.64

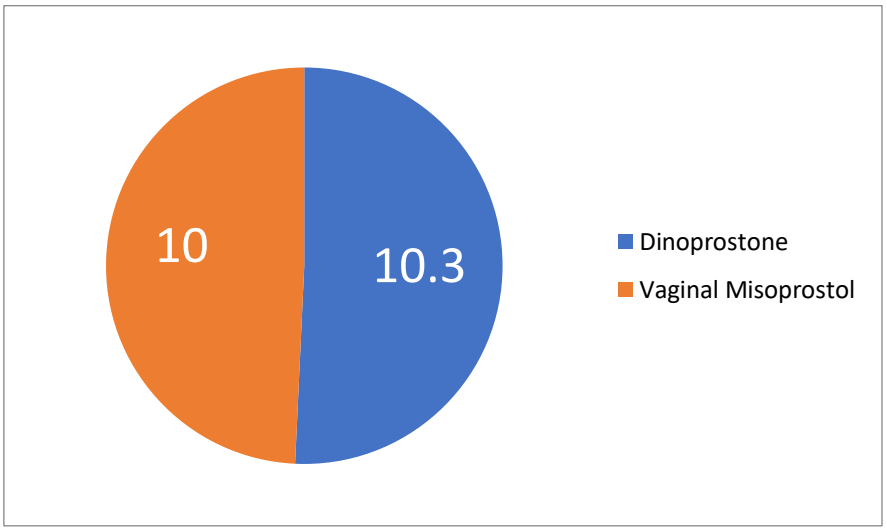

Figure 21: Mean bishop's score pie chart

In Dinoprostone group, the mean Bishop's Score(mean ± SD) of patients was 10.3 ± 2.66.In Misoprostol group, the mean Bishop's Score of patients was 10 ± 2.41.The distribution of the mean Bishop's Score with the group was not statistically significant (p=0.64).

TABLE 12: MEAN INDUCTION TO DELIVERY INTERVAL IN HOURS

	Number	Mean (hours)	SD	p-value
Dinoprostone	53	18.3	4.29	
Vaginal misoprostol	53	15.2	4.9	<0.001

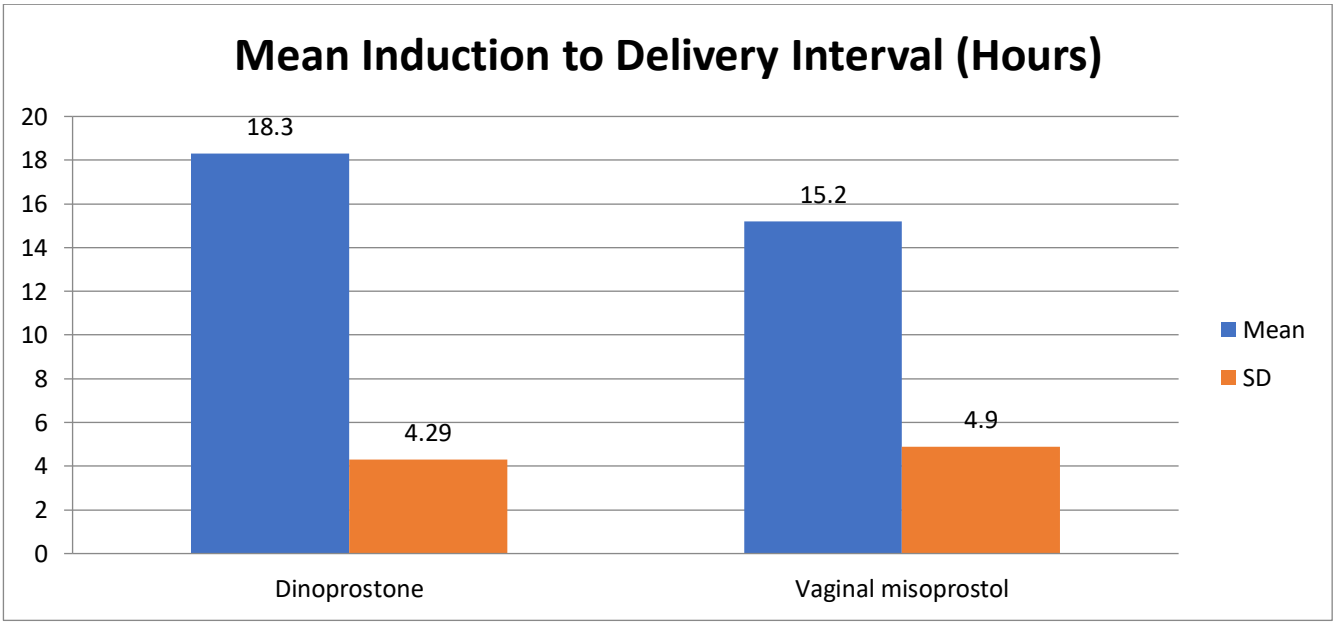

Figure 22: Mean induction to delivery interval bar chart

In the Dinoprostone cohort, the mean induction-to-delivery interval was 18.3 ± 4.29 hours.The mean induction to delivery interval in hours (mean ± SD) for patients in the Misoprostol group was 15.2 ± 4.9.The mean Induction to Delivery Interval in hours among the group exhibited statistical significance ($p<0.001$)

TABLE 13: DISTRIBUTION OF APGAR SCORE<8 AFTER 1MIN IN BOTH STUDY GROUPS

Parity	Dinoprostone(53)		Vaginal misoprostol (53)		
	Number	Percentage	Number	Percentage	P-value
APGAR SCORE<8	9	17	22	41.5	0.006
APGAR SCORE >8	44	83	31	58.5	

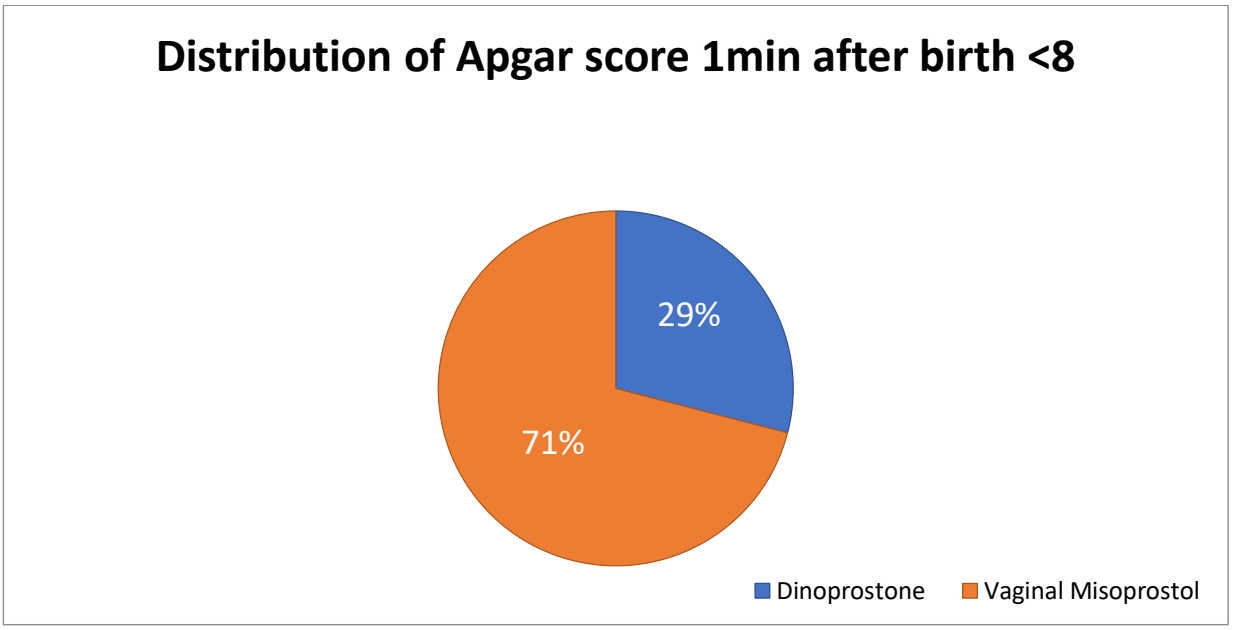

Figure 23: Distribution of APGAR SCORE<8 AFTER 1MIN

In Dinoprostone group, APGAR score<8 after 1min was seen in 9 (17%) patients. In Misoprostol group, APGAR score<8 after 1min was seen in 22 (41.5%) patients. The distribution of APGAR scores within the groups was statistically significant (p=0.006)

TABLE 14: MATERNAL OUTCOME IN BOTH STUDY GROUPS

Maternal Outcome	Dinoprostone(53)		Vaginal misoprostol (53)		
	Number	Percentage	Number	Percentage	P-value
PPH	6	11.3	13	24.5	0.22
Placental Abruption	2	3.8	3	5.7	
Uterine tachysystole	10	18.87	3	5.66	

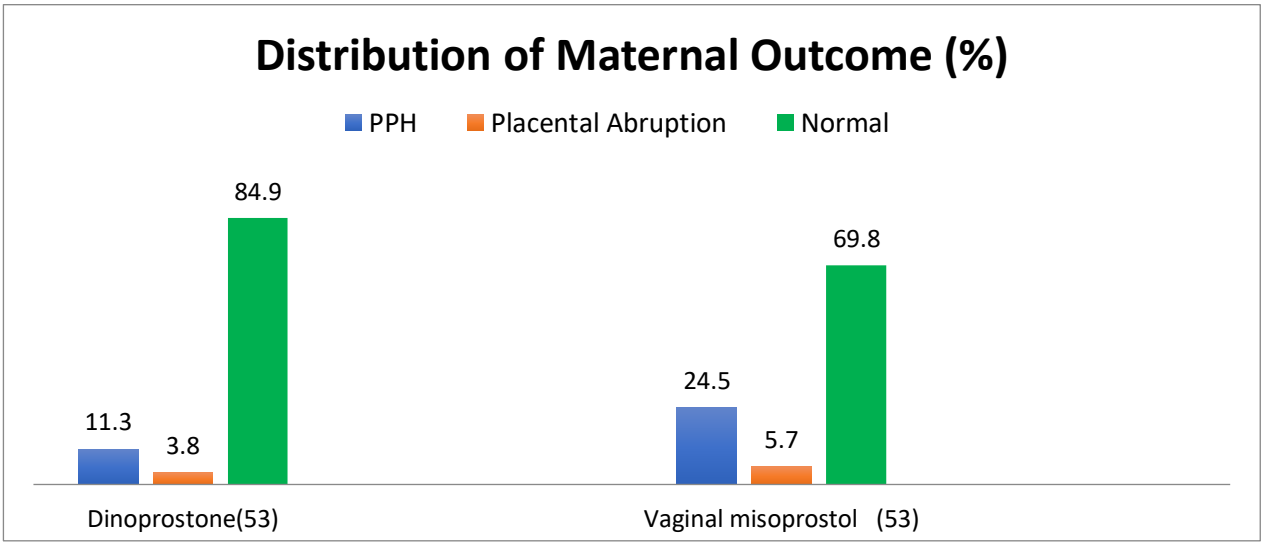

Fig 24: Distribution of Maternal Outcome in Both Groups

In Dinoprostone group, 6 (11.3 %) patients had PPH and 2 (3.8%) patients had a placental abruption. In the Misoprostol group, 2(3.8 %) patients had PPH and 3 (5.7%) patients had a placental abruption. The association of PPH and placental abruption with the groups was not statistically significant (p=0.22).

TABLE 15: INCIDENCE OF UTERINE TACHYSYSTOLE IN BOTH STUDY GROUPS

Uterine Tachysystole	Vaginal misoprostol(53)		Dinoprostone(53)		
	Number	**Percentage**	**Number**	**Percentage**	**P value**
Present	10	18.87	3	5.66	0.038

In misoprostol group, 10 (18.87 %) patients had uterine tachysystole. In Dinoprostone group, 3 (5.66% %) patients had uterine tachysystole. It was found to be statistically significant.

DISCUSSION

The present study is a prospective interventional study. This study was conducted from APRIL 2023 TO FEBRUARY 2025 at the "Department of Obstetrics and Gynaecology of BLDE (DEEMED TO BE UNIVERSITY) SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE."A total of 106 patients were included in this study. All patients were randomised into two groups. Group A: Dinoprostone pessary PV and Group B: T Misoprostol 25 mcg PV. They were divided into two equal (53 each) group as per computer generated randomisation (www.randomizer.org) and were given the prostaglandins according to the study criteria.

The induction of labor (IOL) is a common obstetric procedure aimed at initiating labor in pregnant women who are at risk or have medical indications for delivery before spontaneous labor begins. Several methods exist for cervical ripening and induction, including pharmacologic agents such as **misoprostol** and **dinoprostone**. Both have been extensively studied for their efficacy, safety, and adverse effects, though differences in their clinical use, mechanisms of action, and outcomes remain subjects of debate. The current study aims to compare the effectiveness, safety, and complications of **vaginal misoprostol tablet** and **dinoprostone insert** in the induction of labor.

The present study found that misoprostol significantly reduced the induction-to-delivery interval (15.2 ± 4.9 hours) compared to Dinoprostone (18.3 ± 4.29 hours, $p<0.001$). This is consistent with findings by Patabendige M et al. (2024), who reported that low-dose vaginal misoprostol (≤50 mcg every 4 hours) led to faster labor progression compared to vaginal Dinoprostone. Similarly, Valvi SS et al. (2021) showed a shorter labor duration with misoprostol (9.54 hours) than Dinoprostone (13.45 hours), reinforcing its efficacy in accelerating labor. [84] However, despite its faster action, misoprostol was associated with a higher cesarean section rate (47.17%) compared to Dinoprostone (26.42%), with fetal

distress being the primary indication (46.15% vs. 15.38%, p=0.083). These findings align with those of Sire F et al. (2022), who reported an increased cesarean rate with misoprostol due to abnormal fetal heart rate patterns. [85]

Regarding vaginal delivery rates, the present study observed that Dinoprostone led to a higher rate of vaginal delivery (73.58%) compared to misoprostol (50.94%) (p=0.02). This contrasts with Papanikolaou EG et al. (2004), who reported higher vaginal delivery rates with misoprostol (98.7%) compared to Dinoprostone (91.4%), indicating that misoprostol may be more effective in achieving vaginal birth when labor progresses without complications. [86] Similarly, Valvi SS et al. (2021) found that misoprostol resulted in a vaginal delivery rate of 80.35%, compared to 62.5% with Dinoprostone. [86] The higher vaginal delivery rate with Dinoprostone in our study may be due to lower doses of misoprostol (25 mcg every 4 hours) compared to higher doses used in previous studies, differences in Bishop scores at induction, and variations in labor augmentation protocols

Maternal complications, particularly uterine tachysystole, were more frequent in the misoprostol group (18.87%) compared to the Dinoprostone group (5.66%) (p=0.038). These findings are similar to those of Papanikolaou EG et al. (2004), who reported that uterine tachysystole was more common in the misoprostol group (22.5%) compared to the Dinoprostone group (12%). [84] Similarly, Valvi SS et al. (2021) found that misoprostol had a higher incidence of uterine tachysystole (7.8%) compared to Dinoprostone (2.56%), suggesting that misoprostol, while effective, carries a higher risk of uterine hyperstimulation. This is a significant concern as excessive uterine contractions can compromise fetal oxygenation, leading to distress and increased neonatal morbidity. [86]

Fetal outcomes in the present study further supports the higher incidence of neonatal complications with misoprostol. The study found that NICU admissions were significantly higher in the misoprostol group (47.17%) compared to the Dinoprostone group (16.98%,

p<0.001). Additionally, Apgar scores below 8 at 1 minute were more frequent in the misoprostol group (41.5%) than the Dinoprostone group (17%, p=0.006). These results align with those of Sire F et al. (2022), who found that fetal distress was more frequent in the misoprostol group, leading to higher NICU admissions. [85]

While misoprostol effectively reduces labor duration, it is known to cause higher incidence of fetal distress, leading to increased NICU admissions and cesarean deliveries. In contrast, Dinoprostone, though slower in action, provides better fetal outcomes and a higher vaginal delivery rate.

The dosages used in this study align with standard clinical guidelines. However, evidence suggests that misoprostol's safety profile is dose-dependent. Patabendige M et al. (2024) emphasized that lower doses of misoprostol (≤25 mcg every 4–6 hours) could achieve similar efficacy while reducing complications. Similarly, Sire F et al. (2022) found that higher doses of misoprostol (50 mcg every 6 hours) were associated with increased fetal distress and cesarean rates, reinforcing the need for careful dose titration. [85] Swami KS et al. (2023) noted that Dinoprostone's controlled cervical ripening effect may contribute to safer labor progression, leading to better fetal outcomes. [86]

Based on these findings, the choice of induction agent should be decided based on individual patient characteristics. Misoprostol may be more suitable for patients requiring a faster labor progression, but its higher risk of hyperstimulation and fetal distress necessitates close monitoring. Conversely, Dinoprostone, despite its longer induction duration, appears safer for fetal outcomes and is preferable for cases with a high risk of fetal compromise. These findings agree with Valvi SS et al. (2021) and Patabendige M et al. (2024), both of whom recommended that clinicians should weigh the trade-off between induction speed and fetal safety when choosing between these agents.

Cost-Effectiveness and Accessibility

An often-overlooked aspect of labor induction is the **cost-effectiveness** and **accessibility** of the induction agent. Misoprostol, due to its lower cost and ease of administration (vaginal tablets), is often considered a more **economically viable** option, particularly in resource-limited settings. **World Health Organization (2018)** has recommended misoprostol for labor induction in settings where other options, like dinoprostone, may not be readily available [83]. Conversely, dinoprostone pessary, being a more expensive(3250 rupees) and often more complex formulation (insert or gel), may be less accessible in low-resource settings. However, **Fraser et al. (2014)** argued that while misoprostol is more affordable (around 40 rupees), the increased risk of complications may offset these savings, especially in high-risk pregnancies .

Patient Satisfaction and Comfort

A key consideration in IOL is the **patient's experience** with the induction process. Misoprostol has been associated with more intense uterine contractions, which may contribute to greater discomfort or pain, as shown in a study by **Homer et al. (2015)** [84]. On the other hand, dinoprostone's slower, more gradual onset of contractions may be perceived as less painful, and its insert formulation allows for easier management, making it a more comfortable option for some women. However, a study by **Boulvain et al. (2014)** found that while dinoprostone is perceived as more comfortable by some patients, both drugs led to similar levels of maternal satisfaction in the context of labor induction .

The selection of an induction method should be individualized, considering maternal and fetal risk factors, proper dosing strategies, and the need for continuous fetal monitoring to mitigate adverse effects. Further research is warranted to identify optimal induction protocols that balance efficacy with safety, ensuring the best possible maternal and neonatal outcomes.

As there are no similar studies associated with dinoprostone pessary to our knowledge, comparison with similar studiescouldn't be done for the same.

LIMITATIONS OF THE STUDY

1) Sample Size:

Although 106 participants were included, the sample size might not be large enough to generalize the results to a broader population. A larger sample size could provide more robust data and allow for more granular subgroup analyses.

2) Single-Center Study:

The study was conducted at a single institution (BLDE University, Vijayapura), which may limit the generalizability of the findings to other settings or regions with different patient demographics, healthcare practices, or resources.

3) Short Follow-Up Duration:

The follow-up period appears to be limited to the immediate postpartum period. Longer-term maternal and neonatal outcomes (such as long-term health effects on the mother or child) were not assessed.

FINANCIAL DISCLOSURE

No funds received from any agency for conduct of the study.

CONCLUSION

This study demonstrates that while misoprostol effectively shortens the induction-to-delivery interval but also causes fetal distress and increased chances of cesarean delivery. In contrast, Dinoprostone causes higher normal vaginal delivery with fewer fetal complications, although labor progression is slower. The choice of an induction agent should be individualized based on maternal and fetal conditions, cervical status, and the need for careful fetal monitoring. Optimizing dosage and administration protocols can help balance efficacy and safety, ensuring better outcomes for both mother and baby.

SUMMARY

Induction of labour is defined as "stimulation of contractions before the spontaneous onset of labor, with or without ruptured membranes." Labour induction is increasingly becoming one of the most common obstetric interventions.

Globally, this procedure applies to every 10th pregnant woman, and in some parts of the world even every third labour is induced. The prevalence of induction is up to 22% in India and 24.5% in the USA.

Prostaglandins induce labor by stimulating uterine contractions, softening the cervix, and promoting cervical dilatation.They increase collagenase activity, enhance water content in the cervix, and elevate uterine sensitivity to oxytocin. Modified Bishop's score was used to assess the cervical changes.

To compare efficacy, safety and complications of vaginal Misoprostol 25mcg vs Dinoprostone insert for labor induction, and neonatal and maternal outcomes were the objectives of the study.

It was a single blinded randomised prospective and interventional study with 53 patients in each group, with a total of 106 patients which included ,Singleton ,term pregnancy (>37 weeks) with fetus in cephalic presentation and no signs of labour before induction of labour. The study excluded all the contraindications to vaginal delivery like placenta previa, vasa previa etc.

Group A included dinoprostone 10 mg pessary which was placed in posterior fornix and reassessment was done every 12 hours ,while group B included Tablet misoprostol 25 mcg which was kept 4th hourly upto 6 doses.

A majority of induced patients belonged to 21-30 years of age. Mean bishop's score was 10.3 and 10 in group A and B respectively.73.58% and 50.94% patients delivered vaginally in group A and B respectively and was statistically

significant. Fetal distress was the most common indication for LSCS in two groups.11.3% and 24.5% cases in group A and B were respectively associated with meconium stained liquor and was found to be statistically insignificant. Misoprostol was associated with 47.17% of NICU admissions and was found to be statistically significant (p =<0.001)compared to dinoprostone. The mean induction to delivery time in group A was 18.3 hrs and 15.2 hrs in group B which was statistically significant (p<0.001).Maternal outcomes in terms of PPH, abruption and uterine tachysystole were comparable in the two groups. Our study concluded that, Dinoprostone insert is more effective than misoprostol tablet for labor induction, showing better outcomes in terms of vaginal deliveries, mean Bishop score and maternal and fetal health. This is the first study to directly compare the two methods to our knowledge.

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ANNEXURE

ANNEXURE I

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, _____ , S/O D/O W/O , _____aged_____
years, ordinarily resident of do hereby state/declare that Dr.
VAISHNAVI MAHESH UNNI of Shri. B. M. Patil Medical College Hospital and
Research Centre has examined me thoroughly. Further Dr. VAISHNAVI MAHESH UNNI
informed me that she is conducting a dissertation/research titled
"COMPARATIVE STUDY OF VAGINAL MISOPROSTOL TABLET VERSUS DINOPROSTONE INSERT IN INDUCTION OF LABOR" under the guidance of Dr. Subhashchandra R Mudanur requesting my
participation in the study. According to this, I will be assigned to a parallel randomized trial. I will be administered either of the drugs and evaluated for the induction of labor and outcomes of the pregnancy. Further Doctor has informed me that my participation in this study helped in the evaluation of the results of

the study which is a useful reference for the treatment of other similar cases soon.

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 a 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 the information given by me, I can ask for any clarification during treatment/study related to diagnosis. 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 the dissertation or research, the diagnosis made, mode
of treatment. I am giving consent for the blood investigations and also for the follow-up.

I the undersigned Shri/Smt under my fully
conscious state of mind agree to participate in the said research/dissertation.

Signature of a patient:

Signature of doctor:

Witness: 1.

2.

Date:

Place:

ANNEXURE II

PROFORMA

NAME: AGE: IN PATIENT NUMBER (I.P No.): DATE OF ADMISSION : ADDRESS : PHONE NUMBER :

L.M.P(LAST MENSTRUAL PERIOD) : P.O.G (PERIOD OF GESTATION) : E.D.D (EXPECTED DATE OF DELIVERY): MENSTRUAL HISTORY :

OBSTETRIC HISTORY:

PAST HISTORY:

PERSONAL HISTORY

GENERAL PHYSICAL EXAMINATION:

PALLOR: TEMPERATURE:

PULSE: BLOOD PRESSURE:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

PER ABDOMEN:

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS:

HB- BLOOD GROUP AND RH TYPING -

TC-

PLATELET COUNT :

USG:

PREINDUCTION NST-

POSTINDUCTION NST-

DATE OF DELIVERY: MODE OF DELIVERY:

BIRTH WEIGHT: SEX OF BABY:

COMPLICATIONS:

APGAR SCORE: 1 MINUTE: 5 MINUTES:

NEONATAL RESUSCITATION AT BIRTH:YES/NO

NICU ADMISSION: YES /NO

DAYS OF ADMISSION IN NICU:

PERINATAL DEATH: YES/NO

IF YES,REASON -

NASAL PRONGS/O2/CPAP/HFNC/ROOM AIR :

DAYS OF ADMISSION OF BABY IN HOSPITAL:

ANNEXURE III

ETHICAL CLEARANCE

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 896/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "COMPARATIVE STUDY OF VAGINAL MISOPROSTOL TABLET VERSUS DINOPROSTONE INSERT IN INDUCTION OF LABOR".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.VAISHNAVI MAHESH UNNI

NAME OF THE GUIDE: DR.SUBHASHCHANDRA R. MUDANUR , PROFESSOR, DEPT. OF OBSTETRICS AND GYNAECOLOGY.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE IV

MASTER CHART

	RANDOMISATION NO.	NAME	AGE	IP NO	POG (weeks)	GRAVIDA	PARA	LIVING	ABORTION	Parity	Indication of induction	PER ABDOMEN	PER VAGINA	PREINDUCTION HIST	POSTINDUCTION HIST	INITIAL BISHOPS SCORE	FINAL BISHOPS SCORE	INDICATION OF I.O.S	MODE OF DELIVERY	INDCTION TO DELIVERY TIME	FETAL OUTCOME	APGAR SCORE	UTERINE TACHYSYSTOLE	MECONIUM STAINED LIQUOR	FETAL DISTRESS	MATERNAL OUTCOME	GRO
1	1341-TA7	Akera	24	1629226	40	2	1	0	0	1	postdated	relaxed	os closed,uneffaced		1	1	0	11	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/A
3	1342-DK9	Sangeta	25	2207193	39	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	5	NVDL emgtcs	10hrs	1	7/10,8/10	0	0		1	0	D/B
4	1343-KS6	Vidyashree	21	163035	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	early decelerations +	0	13	0 vaginal	16hrs	0	8/10,9/10	0	0		0	0	D/B
5	1344-ZA6	Veena	25	1221181	40	primi	0	0	0	0	postdated	irritable	os closed,uneffaced		1	1	0	4	NVDL emgtcs	9hrs	1	6/10,8/10	0	0	1	1	0	D/A
6	1221-KB4	Swaranjali	22	1722161	39	3	2	1	1	1	PIH	relaxed	patulous os		1	1	4	13	0 vaginal	15hrs	0	8/10,9/10	0	0		0	0	D/A
7	1222-113	Aishwarya	21	2534098	38	primi	0	0	0	0	Others	irritable	os closed,uneffaced		1	1	0	13	0 vaginal	18hrs	0	8/10,9/10	0	0		0	PPH	B
8	1341-JI07	Sangeeta Gugadadi	20	2192011	37	primi	0	0	0	0	Others	irritable	os closed,uneffaced		1	1	0	13	0 vaginal	16hrs	0	8/10,9/10	0	0		0	0	D/A
9	1342-HP6	Rubiya	24	2794497	39	3	2	2	0	1	Others	relaxed	patulous os		1	1	3	13	0 vaginal	16hrs	0	8/10,9/10	0	0		0	0	D/B
10	1343-PWD	Puja	24	2310358	39	primi	0	0	0	0	Others	irritable	os closed,uneffaced		1	1	0	13	0 vaginal	10hrs	0	8/10,9/10	0	0		0	0	D/A
11	1344-CB5	Prema	19	2796697	39	2	1	1	0	1	Others	irritable	patulous os		1	1	3	13	0 vaginal	16hrs	0	8/10,9/10	0	0		0	PPH	B
12	1421-TKO	Roopa	21	2360911	41	primi	0	0	0	0	postdated	irritable	os closed,uneffaced		1	1	0	13	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/A
13	1422-RRI	Saniya	18	2865440	40	2	1	1	0	1	postdated	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	18hrs	0	8/10,9/10	0	0		0	0	D/B
14	1321-JN5	Bhagamma	23	2301137	39	2	1	1	0	1	Others	relaxed	patulous os		1	1	2	13	0 vaginal	21hrs	0	8/10,9/10	1	0		0	0	D/A
15	1421-GB3	Roopa	23	2922098	39	3	1	0	2	1	Others	relaxed	patulous os		1	1	3	13	0	vacuum assisted vaginal	18hrs	1	6/10,8/10	0	0		1	PPH	B
16	1343-GB2	Reshma	19	2968010	41	primi	0	0	0	0	postdated	irritable	os closed,uneffaced		1	1	0	13	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/B
17	1344-GGD	Sneha	23	3011180	41	2	0	0	1	1	postdated	irritable	patulous os		1	1	3	13	0 vaginal	18hrs	0	8/10,9/10	0	0		0	0	D/A
18	1321-VGS	Wafer	22	3004483	40	3	2	2	0	1	postdated	irritable	patulous os		1	1	4	13	0 vaginal	18hrs	0	8/10,9/10	0	0		0	0	D/B
19	1322-CP6	Bellamma	29	3254476	40	3	1	1	0	1	postdated	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	23hrs	0	8/10,9/10	0	0		0	0	D/A
20	1741-QLB	Jayashri	25	3232236	41	3	2	2	0	1	postdated	irritable	patulous os		1	1	3	13	0 vaginal	18hrs	0	8/10,9/10	0	0		0	0	D/A
21	1742-DY6	Nikita	19	3515233	38	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	8	failed induction with no	emgtcs	10hrs	0	8/10,9/10	1	0		0	PPH	A
22	1743-KT2	Mallamma	27	3045214	38	primi				0	gestational Diabetes mellitus	relaxed	os closed,uneffaced		1	early decelerations +	2	13	2nd stagearrest	emgtcs	12 hrs	1	8/10,9/10	0	0		1	placental disruption	B
23	1744-QD3	Ambika	23	2697760	40	2	0	0	1	1	postdated	irritable	patulous os		1	1	0	10	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/B
24	1641-R27	Nagaveva	21	3297779	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	10	NVDL emgtcs	15hrs	0	8/10,9/10	0	0		0	0	D/B
25	1642-QJO	Ashavari	23	3587779	39	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	18hrs	0	8/10,9/10	0	0		0	0	D/A
26	1643-EI2	Laxmi	25	2653227	37	1	0	0	1	1	Others	relaxed	os closed,uneffaced		1	1	0	4	persistant uterine tatha	emgtcs	10hrs	1	7/10,8/10	1	1		0	PPH	A
27	1644-3NE	Roopa	27	3588891	39	2	0	0	1	1	Others	relaxed	patulous os		1	1	4	13	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/B
28	1922-GH3	Anitha	20	3704419	39	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	20hrs	0	8/10,9/10	0	0		0	PPH	A
29	1922-147	Sumitra	30	4018220	40	2	1	1	0	1	postdated	relaxed	patulous os		1	1	3	13	0 vaginal	18hrs	1	6/10,8/10	0	0		1	0	D/B
30	1021-MR2	Jayashri	19	4004468	38	primi	0	0	0	0	PIH	relaxed	os closed,uneffaced		1	1	0	9	ante partum eclampsia	emgtcs	11hrs	1	6/10,8/10	0	0		1	placental disruption	B
31	1022-R23	Bhagashree	21	2924300	38	primi	0	0	0	0	cholestatic jaundice	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/A
32	1141-DD0	Kaveri	19	3785109	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	10	0 vaginal	16hrs	0	8/10,9/10	0	0		0	0	D/A
33	1142-TMC	Sukanya	25	550384	37	4	2	2	1	1	postdated	relaxed	patulous os		1	1	3	13	0 vaginal	18hrs	1	8/10,9/10	0	0		0	PPH	B
34	1143-ZK2	Lakshmi	24	3977717	39	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	4	fetal distress	emgtcs	17hrs	1	6/10,8/10	0	0		1	0	D/A
35	1144-HHI	Jayashree	19	559888	39	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	late decelerations	0	7	fetal distress	emgtcs	9hrs	1	8/10,9/10	0	0		1	placental disruption	B
36	1221-UP7	Sukanya	25	3946512	37	2	1	1	0	1	Others	relaxed	patulous os		1	1	3	7	fetal distress	emgtcs	8hrs	1	6/10,8/10	0	0		1	0	D/B
37	1222-MMD	Kalpana	26	3903568	41	2	1	1	0	1	postdated	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	21hrs	0	8/10,9/10	0	0		0	0	D/A
38	1344-4A2	Roopa	27	3588891	38	2	0	0	1	1	Others	relaxed	patulous os		1	1	5	13	0 vaginal	22hrs	1	6/10,8/10	0	0		1	0	D/B
39	1341-QC1	Vidya	24	4567789	41	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	10	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/A
40	1342-BI5	aruna	20	3245116	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	early decelerations +	0	9	fetal distress	emgtcs	14hrs	1	6/10,8/10	0	1		1	PPH	B
41	1343-MMK	bagamma	23	2233145	41	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	10	0 vaginal	19hrs	0	8/10,9/10	0	1		1	placental disruption	A
42	1441-TQ1	Shridevi	19	564226	38	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/A
43	1442-UE6	Rekha	28	565567	39	3	2	2	0	1	gestational Diabetes mellitus	relaxed	patulous os			1	3	13	0 vaginal	16hrs	0	8/10,9/10	0	0		0	0	D/B
44	1443-FL2	Radhika	22	74231	40	4	2	1	1	1	postdated	relaxed	patulous os		1	1	3	13	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/A
45	1444-CD9	Rajeshwari	28	4062230	41	2	1	1	0	1	postdated	relaxed	os closed,uneffaced		1	early decelerations +	0	10	pewout seen features	emgtcs	18hrs	0	8/10,9/10	1	0		0	PPH	A
46	1521-VL5	shilpa	34	2940	40	3	2	2	0	1	postdated	relaxed	patulous os		1	1	3	8	0 vaginal	20hrs	0	8/10,9/10	0	0		0	Uterine tachystole	A
47	1522-NE	Pradnya	25	516011	38	2	1	1	0	1	IUGR	relaxed	patulous os		1	1	4	10	0 vaginal	18hrs	1	7/10,8/10	0	0		0	PPH	B
48	1621-PCB	Shweta	22	319611	38	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	10	0 vaginal	20hrs	0	8/10,9/10	1	0		0	0	D/B
49	1622-JD7	Shakuntala	25	3989041	41	4	2	2	1	1	postdated	relaxed	patulous os		1	early decelerations +	4	8	0	emgtcs	10hrs	1	7/10,8/10	1	0		0	0	D/A
50	1721-KJ8	Priyanka	24	458213	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	13	persistant occiptoposte	emgtcs	21hrs	1	6/10,8/10	1	0		0	PPH	A
51	1722-UA8	Sharada	26	3602211	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	8	NVDL	emgtcs	18hrs	1	6/10,8/10	0	0		0	0	D/B
52	1841-WM6	Laxmibai	21	226693	40	3	2	2	0	1	postdated	relaxed	patulous os		1	1	3	9	0 vaginal	21hrs	0	8/10,9/10	0	0		0	PPH	B
53	1842-AR7	Mallamma	19	1743211	40	2	1	1	0	1	postdated	relaxed	os closed,uneffaced		1	1	0	10	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/A
54	1843-EI5	Laxmi	22	3729372	37	primi	0	0	0	1	gestational Diabetes mellitus	relaxed	os closed,uneffaced		1	1	0	8	0 vaginal	16hrs	0	7/10,8/10	0	1		1	0	D/B
55	1844-IGB	Lata	30	2948257	38	3	2	1	0	1	gestational Diabetes mellitus	relaxed	patulous os		1	1	3	10	gestational hypertension	emgtcs	8hrs	0	8/10,9/10	1	0		0	0	D/A
56	1941-TA5	Sangeta	20	2678911	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	14hrs	0	8/10,9/10	0	0		0	PPH	B
57	1942-XL9	Sugirabi	21	2435029	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	10	fetal distress	emgtcs	17hrs	1	6/10,8/10	1	0		0	0	D/A
58	1943-VT9	Durgawwa	20	395533	41	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	13	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/A
59	1944-RX3	Laxmi	20	2699006	38	2	1	1	0	1	IUGR	relaxed	patulous os		1	1	3	13	0 vaginal	21hrs	0	8/10,9/10	0	0		0	0	D/B
60	1041-GW6	Prabhavati	21	2942301	40	2	1	1	0	1	postdated	relaxed	patulous os		1	1	3	13	0 vaginal	18hrs	0	8/10,9/10	0	0		0	0	D/B
61	1042-ISE	Saraswati	22	2699953	38	2	1	1	0	1	Others	relaxed	patulous os		1	1	0	13	0 vaginal	17hrs	0	8/10,9/10	0	0		0	0	D/A
62	1043-MB9	Sneha	25	2023552	38	2	1	1	0	1	Others	relaxed	patulous os		1	1	0	10	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/A
63	1044-PNB	Sushma	20	2900117	38	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	fetal tachycardia	0	8	fetal distress	emgtcs	14hrs	1	7/10,8/10	0	1		1	0	D/B
64	1141-QMB	Sushma	24	2975514	37	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	9	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/A
65	1142-KD6	Laxmi Lamandar	24	157017	37	primi	0	0	0	0	PIH	relaxed	os closed,uneffaced		1	1	0	6	fetal distress	emgtcs	20hrs	1	7/10,8/10	1	0		1	0	D/A
66	1143-CD7	Bibijan	28	163145	39	1	1	1	0	1	Others	relaxed	os closed,uneffaced		1	1	0	8	NVDL	emgtcs	12 hrs	0	8/10,9/10	1	1		1	PPH	B
67	1144-ERL	Jyoti Nandagiri	21	2653444	38	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	10	0 vaginal	14hrs	0	8/10,9/10	0	0		0	0	D/B
68	1221-GLS	Reshma Rathod	22	2022152	40	primi	0	0	0	0	PIH	relaxed	os closed,uneffaced		1	1	0	8	fetal distress	emgtcs	8hrs	1	7/10,8/10	0	0		1	0	D/B
69	1222-VC0	Shrikrishna	25	2024/053	38	1	1	1	0	1	PIH	relaxed	os closed,uneffaced		1	1	0	11	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/A
70	1321-ON9	Sneha	22	Nurse not working	38	primi	0	0	0	0	Others	relaxed	patulous os		1	1	3	10	fetal distress	emgtcs	9hrs	0	8/10,9/10	0	0		1	0	D/B
71	1322-YZB	Mehbubi Jalageri	34	Nurse not working	39	3	2	2	0	1	PIH	relaxed	os closed,uneffaced		1	1	0	6	imminent eclampsia	emgtcs	20hrs	1	7/10,8/10	0	0		1	0	D/A
72	1441-IN6	naushara	25	2024/869	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	early decelerations +	0	8	Thick MSL	emgtcs	21hrs	1	7/10,8/10	0	1		1	0	D/A
73	1442-PZD	Prabhavati Sajjan	31	2024/0460	40	5	3	2	1	1	postdated	relaxed	patulous os		1	1	4	12	0 vaginal	23hrs	0	8/10,9/10	0	0		0	0	D/A
74	1443-XL9	geeta	27	2024/718	38	4	2	2	1	1	PIH	relaxed	patulous os		1	1	3	10	0 vaginal	18hrs	0	8/10,9/10	0	0		0	PPH	B
75	1444-LAO	Supriya	30	1067	40	2	1	1	0	1	postdated	relaxed	os closed,uneffaced		1	1	0	10	0 vaginal	20hrs	0	8/10,9/10	0	0		0	0	D/A
76	2521-IK5	kaveri rathod	22	1899	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	1	0	9	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/A
77	2522-UL7	hajrathabi	27	4458	39	3	2	2	0	1	Others	relaxed	patulous os		1	1	3	10	0 vaginal	18hrs	1	7/10,8/10	0	1		1	0	D/B
78	2641-MK7	Jyoti Torai	20	3470	39	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	8	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/A
79	2642-UL1	Veena Ramesh	26	5230	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced		1	early decelerations +	0	10	non reassuring test	emgtcs	7hrs	0	8/10,9/10	0	0		0	0	D/B
80	2643-3J4	sahera	28	3476	39	5	4	2	0	1	postdated	relaxed	patulous os		1	1	0	9	0 vaginal	22hrs	0	8/10,9/10	0	0		0	0	D/A
81	2644-3R3	rajani	27	4567	40	3	2	2	0	1	postdated	relaxed	patulous os		1	1	3	10	failed induction with no	emgtcs	21hrs	1	6/10,8/10	0	1		1	0	D/B
82	2721-WJ4	renukashirapur	19	6995	38	primi	0	0	0	0	Others	relaxed	os closed,uneffaced		1	1	0	9	0 vaginal	16hrs	0	8/10,9/10	0	0		0	0	D/B

67	2144-EI6	Jyoti Nandageri	21	265344	38	primi	0	0	0	0	Others	relaxed	os closed,uneffaced	1	1	0	10	0	vaginal	14hrs	0	8/10,9/10	0	0	0	0	B
68	2221-GL5	Reshma Rathod	22	202152	40	primi	0	0	0	0	PIH	relaxed	os closed,uneffaced	1	1	0	8	Fetal distress	emglscs	8hrs	1	7/10,8/10	0	0	1	0	B
69	2222-JCO	Sneha rathod	25	2024/552	38	2	1	1	0	1	PIH	relaxed	os closed,uneffaced	1	1	0	11	0	vaginal	20hrs	0	8/10,9/10	0	0	0	0	A
70	2321-ON3	Savita	22	Numr not workin	38	primi	0	0	0	0	Others	relaxed	patulous os	1	1	3	10	Fetal distress	emglscs	5hrs	0	8/10,9/10	0	0	1	0	B
71	2322-VOB	Mehbubi Jalageri	34	Numr not workin	39	3	0	0	2	1	PIH	relaxed	os closed,uneffaced	1	1	0	6	imminent eclampsia	emglscs	20hrs	1	7/10,8/10	0	0	1	0	A
72	2441-EA5	naushara	25	2024/869	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	early decelerations+	0	8	Thick MSL	emglscs	21hrs	1	7/10,8/10	0	1	1	0	A
73	2442-PZO	Prabhavati Sajjan	31	2024/3460	40	5	3	2	1	1	postdated	relaxed	patulous os	1	1	4	12	0	vaginal	23hrs	0	8/10,9/10	0	0	0	0	A
74	2443-XJ9	geeta	27	2024/718	38	4	2	2	1	1	PIH	relaxed	patulous os	1	1	3	10	0	vaginal	18hrs	0	8/10,9/10	0	0	0	PPH	B
75	2444-UAO	Suprita	30	1067	40	2	1	1	0	1	postdated	relaxed	os closed,uneffaced	1	1	0	10	0	vaginal	20hrs	0	8/10,9/10	0	0	0	0	B
76	2521-IK5	kaveri rathod	22	1899	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	1	0	9	0	vaginal	22hrs	0	8/10,9/10	0	0	0	0	A
77	2522-UL7	hajrashaikh	27	4458	39	3	2	2	0	1	Others	relaxed	patulous os	1	1	3	10	0	vaginal	18hrs	1	7/10,8/10	0	1	1	0	B
78	2641-MA7	Jyoti Toravi	20	3470	39	primi	0	0	0	0	Others	relaxed	os closed,uneffaced	1	1	0	8	0	vaginal	22hrs	0	8/10,9/10	0	0	0	0	A
79	2642-UL1	Veena Ramesh	26	5230	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	early decelerations+	0	10	nonreasuring nst	emglscs	7hrs	0	8/10,9/10	0	0	0	0	B
80	2643-SD4	sahera	28	3476	39	5	4	2	0	1	postdated	relaxed	patulous os	1	1	0	9	0	vaginal	22hrs	0	8/10,9/10	0	0	0	0	A
81	2644-SR3	rajani	27	4567	40	3	2	2	0	1	postdated	relaxed	patulous os	1	1	3	10	failed induction with no	emglscs	21hrs	1	6/10,8/10	0	1	1	0	B
82	2721-WJ4	renukashirapur	19	6995	38	primi	0	0	0	0	Others	relaxed	os closed,uneffaced	1	1	0	9	0	vaginal	16hrs	0	8/10,9/10	0	0	0	0	B
83	2722-HM9	lalimashaikh	20	4057	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	latedecelerations	0	6	nonreasuring nst	emglscs	9hrs	0	8/10,9/10	0	1	1	placental abruption	A
84	2821-PI8	sangeeta Bodihal	23	7803	40	2	1	1	0	1	IUGR	relaxed	patulous os	1	1	3	12	0	vaginal	11hrs	0	8/10,9/10	0	0	0	PPH	B
85	2822-HM9	Ningamma	28	4281	40	5	3	2	1	1	postdated	relaxed	patulous os	1	1	3	12	0	vaginal	22hrs	0	8/10,9/10	0	0	0	0	A
86	2921-XA2	rathabai	26	5546	41	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	1	0	12	0	vaginal	16hrs	0	8/10,9/10	0	0	0	0	A
87	2922-CHO	Renukamore	24	2024/556	39	primi	0	0	0	0	Others	relaxed	os closed,uneffaced	1	1	0	8	Fetal distress	emglscs	6hrs	1	6/10,8/10	0	1	1	0	B
88	3041-LT9	sandhyathakur	32	273867	40	primi	0	0	0	0	PIH	relaxed	os closed,uneffaced	1	1	0	7	Fetal distress	emglscs	18hrs	1	7/10,8/10	0	1	1	0	B
89	3042-PN7	mahanandashirol	32	7806	38	primi	0	0	0	0	PIH	irritable	os closed,uneffaced	1	1	0	6	non resuringnst with f	emglscs	10hrs	0	8/10,9/10	0	0	0	0	B
90	3043-CLO	Shruti kollatnur	21	4425	39	primi	0	0	0	0	PIH	relaxed	os closed,uneffaced	1	1	0	6	imminent eclampsia	emglscs	12 hrs	0	8/10,9/10	0	0	0	0	A
91	3044-MX2	shalini yambhatanal	35	4776	39	6	4	3	1	1	Others	irritable	patulous os	1	1	3	10	0	vaginal	21hrs	0	8/10,9/10	0	0	0	0	A
92	3121-JT6	Mamatajatti	30	7658	40	2	1	1	0	1	postdated	relaxed	os closed,uneffaced	1	1	0	12	0	vaginal	22hrs	0	8/10,9/10	1	1	0	0	A
93	3122-SE8	Shruti bid	29	8400	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	1	0	8	Fetal distress	emglscs	7hrs	1	6/10,9/10	0	1	1	PPH	B
94	3221-UR5	gajrabai	21	167948	40	primi	0	0	0	0	PIH	relaxed	os closed,uneffaced	1	1	0	6	persistant tachycardia wi	emglscs	12 hrs	1	7/10,8/10	0	0	1	PPH	A
95	3222-PJ3	lamiyellbai	22	272972	37	primi	0	0	0	0	PIH	relaxed	os closed,uneffaced	1	1	0	10	0	vaginal	21hrs	0	8/10,9/10	0	0	1	0	B
96	3341-DI9	Bhagyashree rachugond	21	9546	40	3	2	2	0	1	postdated	relaxed	os closed,uneffaced	1	1	0	10	0	vaginal	21hrs	0	8/10,9/10	0	0	0	0	A
97	3342-ON8	premavati rajaput	19	13344	39	2	1	1	0	1	IUGR	relaxed	patulous os	1	latedecelerations	4	9	Thick MSL	emglscs	20hrs	1	7/10,8/10	0	1	1	0	B
98	3343-PP2	savitri devakar	22	12720	38	primi	0	0	0	0	Others	irritable	os closed,uneffaced	1	1	0	8	thin MSL	emglscs	10hrs	0	8/10,9/10	0	1	1	0	B
99	3344-FCO	asmabudihal	22	9696	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	1	0	11	0	vaginal	21hrs	0	8/10,9/10	0	0	0	0	A
100	3421-UD8	jyoti jadhav	22	10399	40	2	1	1	0	1	postdated	relaxed	patulous os	1	1	3	10	0	vaginal	22hrs	0	8/10,9/10	0	0	0	0	A
101	3422-YN3	rukhsana	25	14492	41	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	1	0	6	failed induction with no	emglscs	18hrs	0	8/10,9/10	0	0	0	0	B
102	3541-UJ5	swetadeepak	25	14809	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	1	0	8	0	vaginal	22hrs	0	8/10,9/10	0	0	0	0	A
103	3542-UJ3	shyamala handral	24	13696	38	2	1	1	0	1	PIH	relaxed	patulous os	1	1	3	10	0	vaginal	18hrs	0	8/10,9/10	1	0	0		A
104	3543-BM5	shivamma	26	14245	41	2	1	1	0	1	postdated	relaxed	patulous os	1	1	3	8	Fetal distress	emglscs	8hrs	1	6/10,8/10	0	1	0	PPH	B
105	3544-UE4	Ratnabai	27	15821	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	1	0	8	Thick MSL	emglscs	10hrs	1	7/10,8/10	0	1	1	0	B
106	3621-AV9	Laxmibai ramesh	25	14562	40	primi	0	0	0	0	postdated	relaxed	os closed,uneffaced	1	1	0	10	failed induction with no	emglscs	16hrs	1	6/10,8/10	0	0	1	0	B
107	3622-NP9	Danamma	20	15888	41	2	1	1	0	1	postdated	relaxed	patulous os	1	1	3	11	0	vaginal	22hrs	0	8/10,9/10	0	0	0	0	A
108																											
109																											
110										multi-1				1- REACTIVE	1-REACTIVE						NICU admission-1	AT1 MIN-8					
111										primi-0				0-NON REACTIVE	0-NONREACTIVE						Favourable-1	AT5 MINS-8					
112																											

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"COMPARATIVE STUDY OF VAGINAL MISOPROSTOL TABLET VERSUS DINOPROSTONE INSERT IN INDUCTION OF LABOR"

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