A STUDY ON COMPARISON OF SERUM PHOSPHORUS LEVELS AS SEVERITY AND PROGNOSTIC MARKER IN ADULTS AND ELDERLY (>60 YEARS)

BY

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"A STUDY ON COMPARISON OF SERUM PHOSPHORUS LEVEL AS SEVERITY AND PROGNOSTIC MARKER IN PATIENTS ON MECHANICAL VENTILATION AMONG ADULTS AND ELDERLY (>60 YEARS)"

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ABSTRACT

Background and Objective

Mechanical ventilation(MV) is a supportive therapy for patients with acute respiratory failure. Studies have shown direct relationship between serum phosphorus on admission and risk of respiratory failure requiring MV. This study aims to determine admission serum phosphorus level's ability to predict mortality in 64 mechanically ventilated adult patients groped among young adults(<60 years) and elderly(60 years or older) admitted to an ICU.

Materials and methods- Data was collected from 64 subjects who were above 18 years of age and irrespective of sex admitted in ICU/CCU wards between December 2020 to June 2022 in BLDE (DU) Shri B M Patil Medical College and Hospital. The subjects were followed prospectively, their SOFA scores on admission was assessed to determine the morbidity of critical illness and analysis was drawn. Patients with Diabetic Ketoacidosis, Head Trauma, Renal Failure, Hyper or Hypoparathyroidism, Leukemia and Lymphoma were excluded.

Result- The study had 75% males and 25% females. The most common comorbidity among young adults was Type 2 Diabetes Mellitus (15.62%) while Hypertension (28.12%) was common in elderly. The commonest indication for mechanical ventilation was Poisoning(31.25%) in Young and Cerebrovascular Accident (34.37%) in elderly. The common complication among them was ventilator associated pneumonia (14.06%). Out of 64 patients on mechanical ventilation, nine patients had hypophosphatemia and eighteen were having hyperphosphatemia. Among eighteen patients, ten died while other eight recovered and all patients who presented with hypophosphatemia died.

Conclusion- Decreased serum phosphorus levels on admission can be a potential indicator of mortality in mechanically ventilated patients.

Keywords- Mechanical ventilation, Mortality, Serum phosphorus, SOFA score

LIST OF ABBREVIATIONS

MV	Mechanical Ventilation
iP	Inorganic Phosphorous
DNA	Deoxyribonucleic Acid
RNA	Ribonucleic Acid
ATP	Adenosine Triphosphate
NPT	Sodium dependent phosphate cotransporters
PTH	Parathyroid hormone
FGF23	Fibroblast Growth Factor 23
RDA	Recommended Daily Allowance
CKD	Chronic Kidney Disease
CVD	Coronary Vascular Disease
2,3-DPG	2,3- Diphosphoglycerate
pH	Potential of Hydrogen
PFK	Phosphofructokinase
$FEPO_4$	Fractional Excretion of filtered Phosphate
UPO_4	Urinary phosphate
PPO_4	Plasma phosphate
Cr	Creatinine
GFR	Glomerular Filtration Rate
PHP	Pseudohypoparathyroidism
BUN	Blood Urea Nitrogen
eGFR	Estimated Glomerular Filtration Rate
KDIGO	Kidney Disease Improving Global Outcome
SOFA	Sequential Organ Failure Assessment

PaO_2	Arterial Oxygen Pressure
FiO_2	Fraction of inspired Oxygen
MAP	Mean Arterial Pressure
CO_2	Carbondioxide
$PA\text{-}aO_2$	Alveolar-arterial Oxygen gradient
Vd/Vt	Total dead space ratio
TV	Tidal Volume
AC	Assist Control
SIMV	Synchronized Intermittent Mechanical Ventilation
PSV	Pressure Support Ventilation
RR	Respiratory Rate
PEEP	Positive End Expiratory Pressure
$ETCO_2$	End-Tidal Carbon Dioxide
COPD	Chronic Obstructive Pulmonary Disease
ARDS	Acute Respiratory Distress Syndrome
ILD	Interstitial Lung Disease
VAP	Ventilator Associated Pneumonia
CFU	Colony Forming Unit
BAL	Bronchoalveolar Lavage
VILI	Ventilator Induced Lung Injury
ICU	Intensive Care Unit
COVID	Corona Virus Disease
ECG	Electrocardiography
GCS	Glasgow Coma Scale

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INTRODUCTION

Hennig Brandt made the first discovery of phosphorus in Hamburg in 1669. He was a trader from Germany who enjoyed alchemy. He prepared 50 buckets of urine during this discovery, leaving them out till they putrefied and began to breed worms. He next condensed the phosphorus vapour that had been gathered by evaporating the urine into the water, after which he boiled the residue until it was red-hot. He had kept his finding a secret for a while, believing that he had found the Philosopher's Stone, which might be used to change base metals into gold due to the element's tendency to "phosphoresce," or shine in the dark. The word "phosphorous," which means "bearer of light," is taken from Greek. Brandt sold phosphorous to Daniel Kraft when he was out of money.[(1)]

It was made more commonly available after it was discovered that bone, which is composed of calcium phosphate, can be utilised to create phosphorus.

Phosphorus is one of the most important elements and is found mostly inside cells. It is naturally contained in food. It is a crucial part of RNA, DNA, bones and the teeth. Phosphorus, as phospholipids is an important part of cell membranes and ATP, which is required for cellular activity in the body.[(2)] Many elements in body exist in phosphorylated forms.

Also, it is necessary for the storage and translation of genetic information in nucleotides, intracellular signalling via the phosphorylation or dephosphorylation of important enzymes, energy transfer, and acid-base buffering.[(3)] 300 mg of phosphate enters and exits the body each day in order to maintain homeostasis.[(4)] Most of the phosphorous is present in the cells while only 1% is present extracellularly.[(3)]

Serum phosphate levels in adults typically range from 2.5 to 4 mg/dL. Hypophosphatemia is characterised as serum phosphate levels below 2.5 mg/dL, and hyperphosphatemia is defined as levels above 4.5 mg/dL.

AIMS AND OBJECTIVES

1. To study the Serum Phosphorous level as the severity and prognostic marker in patients on mechanical ventilation

2. To compare Serum Phosphorous levels among young adults(<60 years) and elderly(60 years and older) requiring mechanical ventilation.

REVIEW OF LITERATURE

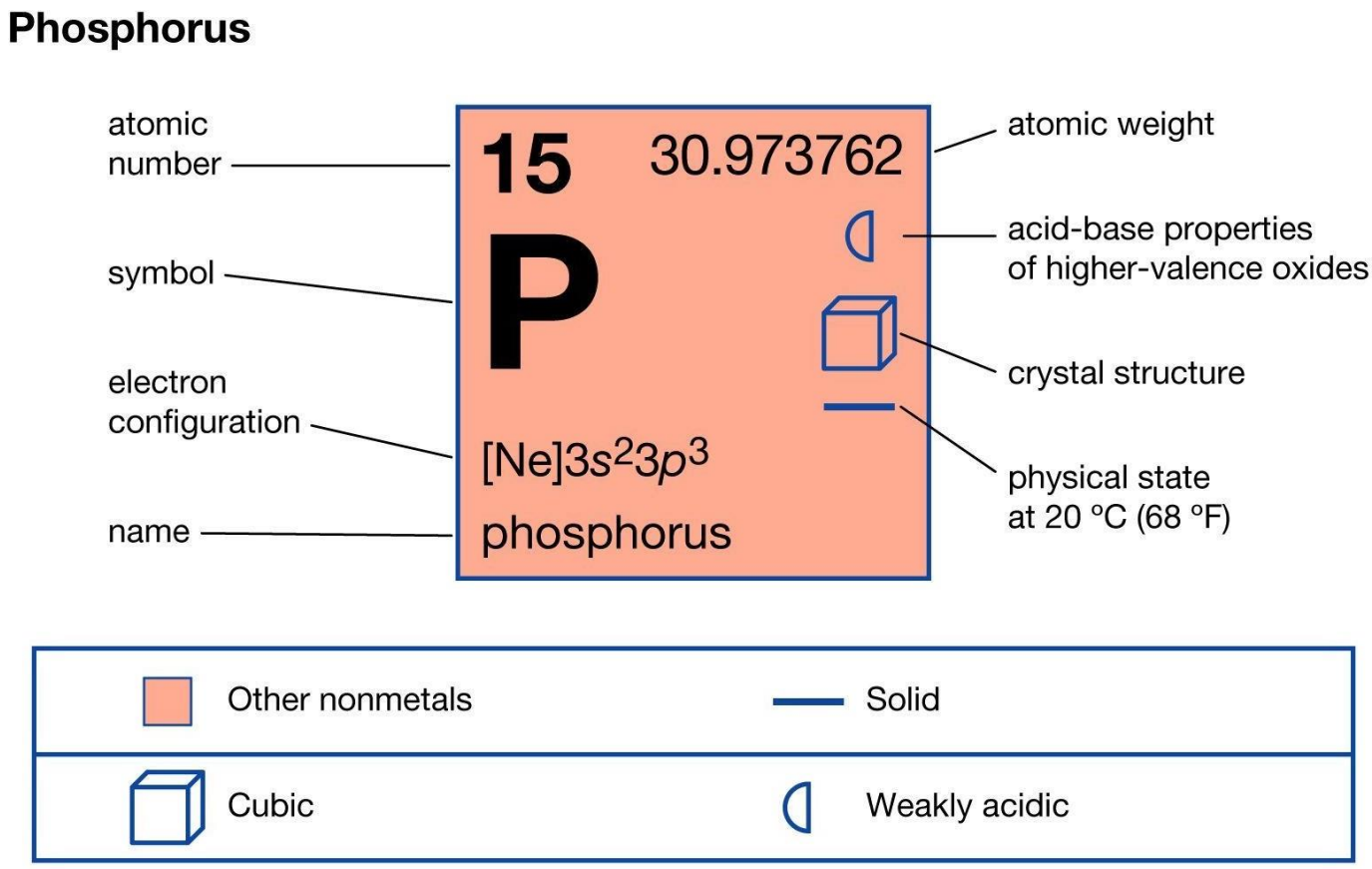


Fig.1 Chemical properties of Phosphorous

Phosphorous is the 15th element in the periodic table with an atomic weight of 30.97. It is a non-metallic chemical element with chemical symbol P.

It has two allotropes, one is white phosphorous which is poisonous, soft waxy solid which glows when placed in dark and spontaneously ignites in the air, releasing thick fumes. Sunlight or heat converts it to red phosphorous allotrope which is less reactive and soluble than phosphorous.

In the living phosphorous occurs as phosphate and is important component of DNA, RNA,ATP, and bone.

In other forms phosphorous is very toxic. White phosphorous when ingested can cause mandibular necrosis and result in “phossy jaw”. The other organic derivatives like nerve gas is also very toxic to the man.[5]

ABSORPTION OF PHOPHOROUS

Sodium Dependent Phosphate Co-transporters, [6]

The brush border cells of the kidneys have sodium-dependent phosphate co-transporters (NPT), which rely on the sodium-potassium-dependent ATPase to transport phosphate. These co-transporters exist in three types:

1. Type I Na/Pi co-transporter (NPT1): This protein is present on the proximal tubule's brush border membrane in the kidneys.

2. Type II Na/Pi co-transporters (NPT2)- It has three isoforms namely NPT2a, NPT2b, and NPT2c. NPT2b is present in small intestine and pulmonary tissue but it is absent in renal system. NPT2a and NPT2c are only found on the brush border membrane of the renal proximal tubules.

3. Type III Na/Pi co-transporters (NPT3): It regulate cellular phosphate homeostasis and are expressed in the basolateral membrane of every nephron.

FACTORS AFFECTING SERUM PHOSPHOROUS[7]

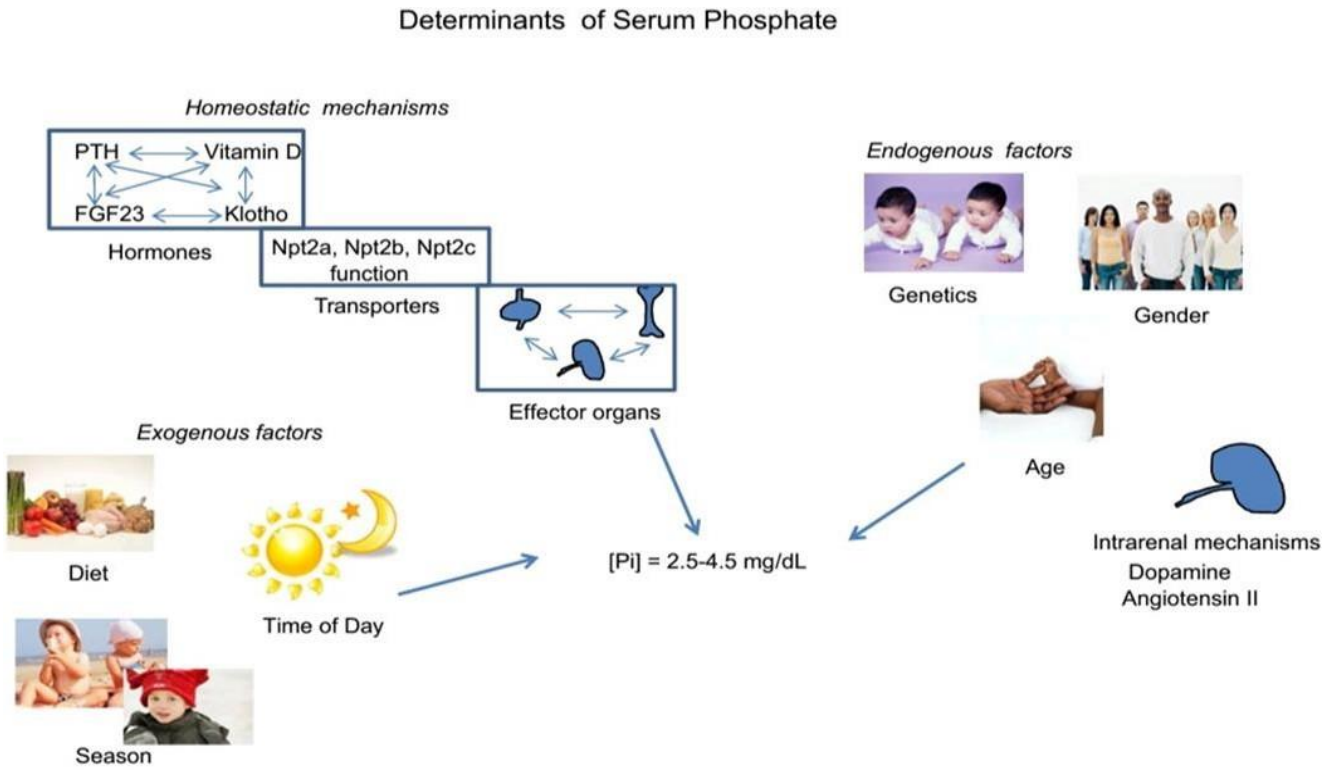


Fig. 2 Factors affecting serum phosphorous

The blood phosphorous levels are determined by multiple factors (Fig 2). Homeostasis is maintained by hormones, transporters and effective organs. The other factors involved are namely,

1. Exogenous - Season, Diet, and Time of Day

2. Endogenous- Genetics, age, and gender

Mechanisms involved to restore homeostasis has not been identified

REGULATION OF PHOSPHORUS

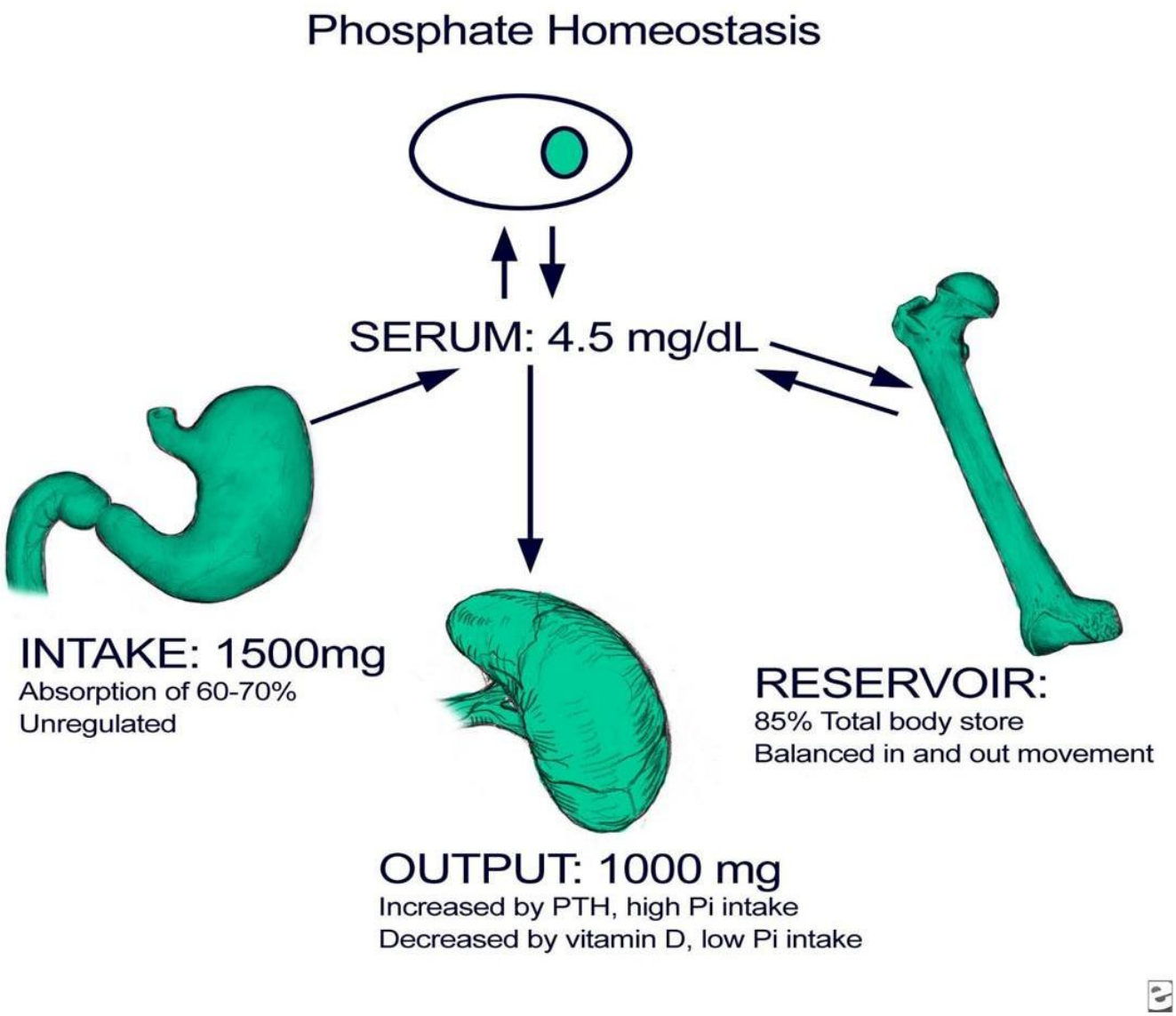


Fig.3 Phosphate regulation

The small intestine passively absorbs about 1500 mg of phosphate daily. High phosphorous intake does not impair this intestinal uptake. Remaining is absorbed actively in the body. Metabolism of phosphorous is regulated by vitamin D, Fibroblast Growth Factor(FGF-23) and parathyroid hormone (PTH). The bones act as the crucial reservoir of the phosphates. The movement of phosphorous in and out of this reservoir is important in maintaining phosphate

homeostasis in the body. Also excess of dietary phosphate is excreted by the kidneys maintaining serum phosphate levels of approximately 3-4 mg/dL.[7]

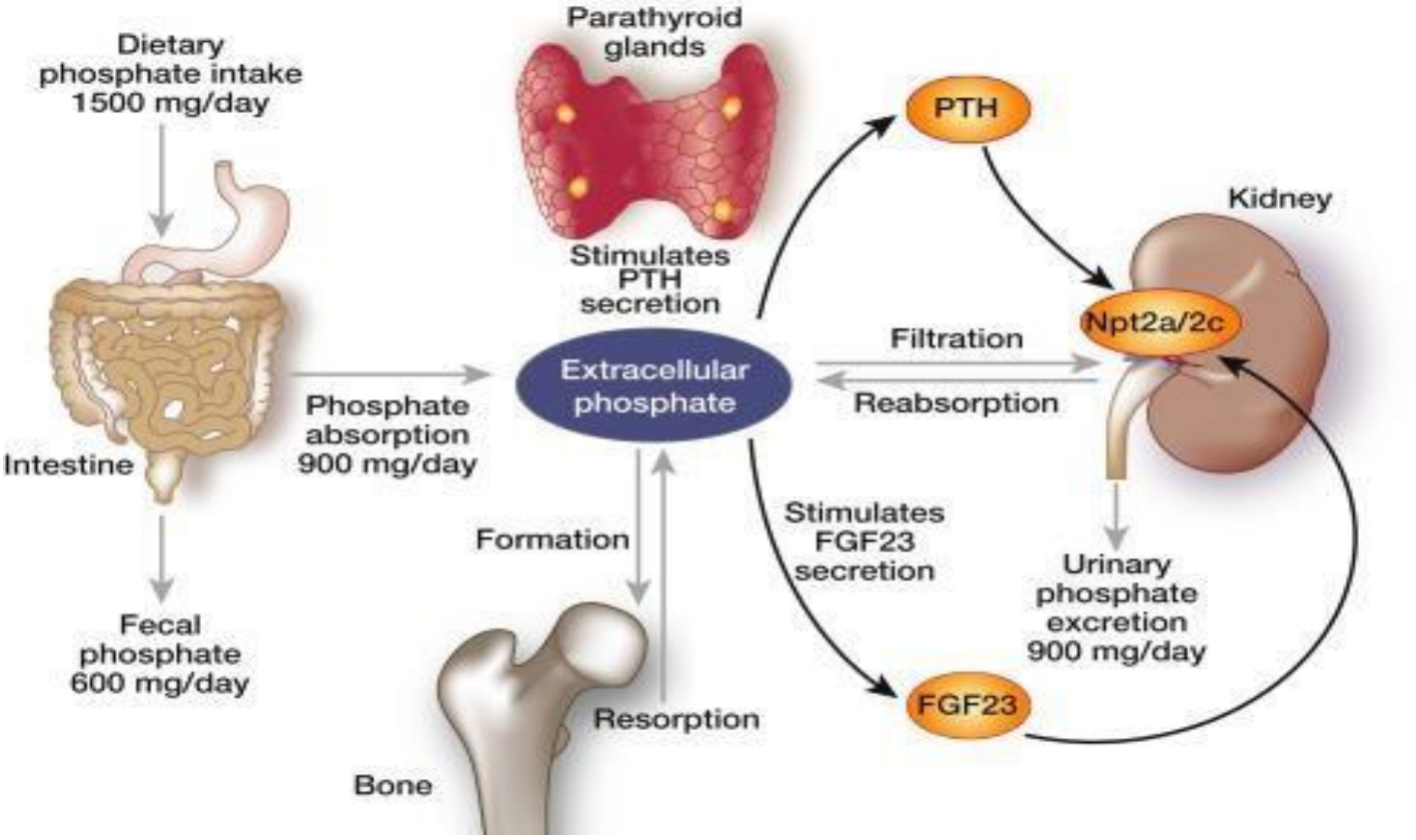


Fig. 4 Phosphate Homeostasis (Ref 8)

Phosphorus homeostasis is controlled by the kidneys, intestines, and bones, and it requires to regulate urine losses at levels that are equal to net phosphorus absorption as well as making sure that the same amounts of phosphorus are deposited in and removed from the bones.

HORMONAL REGULATION[8]

1. PARATHYROID HORMONE (PTH)

The receptors of PTH is widely present in osteoblast, proximal and distal renal tubules. PTH facilitate breakdown of bones and increases phosphate absorption in the gut, which is carried out through the synthesis of vitamin D under the influence of PTH. This leads to increased blood

phosphate levels. This effect of parathyroid hormone is superseded by it's phosphaturic effect. As a result the net effect of PTH is low normal or frankly normal serum phosphorous levels.

Also, high PTH levels increases FGF23 production which in turn causes decreased phosphate absorption by the intestines and increased renal excretion of phosphate which can result in hypophosphatemia.

2. FGF23

It is mainly produced by the osteocytes and osteoblast. FGF23 has phosphaturic effect as it reduces NPT 2a and 2c expression. It also decreases phosphate absorption in the intestines indirectly by decreasing 1,25-dihydroxyvitamin D_3.

3. VITAMIN D

Increased Vitamin D levels directly suppresses PTH and FGF23 secretion leading to decreased phosphate secretion via kidneys thereby increasing serum phosphate levels.

EXOGENOUS FACTORS

1. TIME OF DAY

Blood phosphorous levels exhibit a diurnal cycle, peaking in the middle of the day and just after midnight, with a nadir between 8 and 10 in the morning.

2. DIET

Moe et al[9] concluded that despite the fact that both vegetarian and non-vegetarian diets had an equivalent quantity of phosphate, persons eating vegetarian diets had lower serum phosphorus levels and excreted less phosphorous than those eating diets high in animal products.

Refeeding syndrome, also known as refeeding hypophosphatemia, can develop in people who have severe protein- or calorie-deficiency. Most often, it appears 2 to 5 days after beginning enteral or parenteral nourishment. It happens as a result of the metabolism switching from a catabolic to anabolic condition.

ENDOGENOUS FACTORS

1. AGE

Keating et al mentioned that serum phosphorous levels varies with age with highest being in early neonatal period and early infancy and lower during puberty and adulthood[10]

2. SEX

Phosphorous levels also differs with aging. Following adulthood, there is decreasing levels of phosphorous in men. Women show such a trend in premenopausal period whereas in post-menopausal period there is an increase in serum phosphate level which can be attributed to loss of estrogen effect.[10]

3. GENETIC FACTORS

Mutations in sodium- phosphate cotransporters can result in altered serum phosphorous levels.[11]

Mutation in Npt2c (type IIc sodium-phosphate cotransporter) can result in syndrome of Hereditary Hypophosphatemic rickets.[11]

SOURCES OF PHOSPHORUS

DIETARY SOURCES

40%–70% of phosphorus is absorbed from the diet. Fig.5 shows the various dietary sources of phosphorous. The most amount of phosphorous is present in dairy products like eggs, meat, fish and poultry. The rate of phosphorus absorption from animal foods is higher than that from plant sources. [12]As dietary supplements, phosphorus is also available in the form of phosphate salts like dipotassium phosphate and disodium phosphate as well as phospholipids like phosphatidylcholine and phosphatidylserine. The bioavailability of phosphate salts is about 70%. The recommended dietary allowance(RDA) of phosphorous varies with age (Table 1).

Fig.5 Dietary sources of Phosphorous

DIETARY SUPPLEMENTS [12]

Table 1. RECOMMENDED DIETARY ALLOWANCES (RDAs) FOR PHOSPHOROUS

AGE	MALE	FEMALE	PREGNANCY	LACTATION
18 YEARS	1,250 mg	1,250 mg	1,250 mg	1,250 mg
19+YEARS	700 mg	700 mg	700 mg	700 mg

EFFECT OF CHRONIC KIDNEY DISEASE ON PHOSPHORUS

CHRONIC KIDNEY DISEASE (CKD)[13]

Phosphorus is excreted from kidneys. Therefore, when kidney function declines as seen in conditions like CKD, phosphate excretion decreases due to which serum phosphate concentration rises.(Fig. 6)

Increased serum phosphorous levels stimulate FGF23 from bones. Secondary to high FGF 23 levels and hyperphosphatemia, there is suppression of $1,25(OH)_2D$ and 1-alpha hydroxylase activity which leads to hypocalcemia. Hyperphosphatemia and hypocalcemia stimulate parathyroid hormone synthesis and release. But because of progressive loss of renal mass, there is impaired renal phosphate excretion. Hence, the net result is increased blood phosphorous levels.

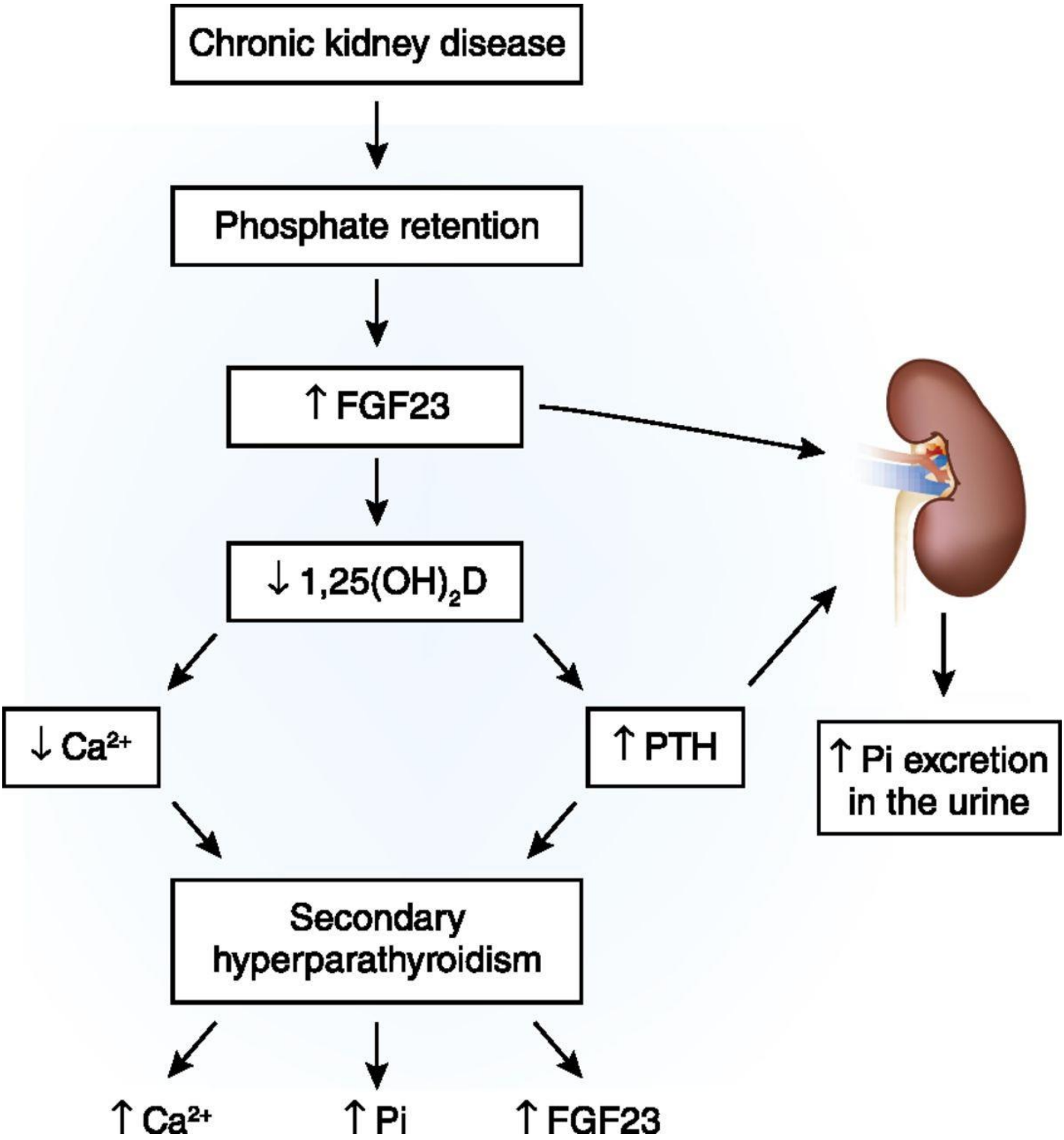


Fig.6 Relation Chronic Kidney Disease and Serum Phosphorous Levels

EFFECT OF PHOSPHORUS ON CARDIOVASCULAR DISEASES

Studies[13,14] have shown an association between high phosphate levels and risk of cardiovascular disease(CVD).

Increased dietary intake of phosphorous leads to FGF23 synthesis which subsequently results in hyperphosphatemia. This in turn leads to endothelial calcification in coronary vessels.[14]

Hyperphosphatemia in a CKD patient is associated with increased FGF23 levels and secondary hyperparathyroidism which can cause ventricular hypertrophy.[13]

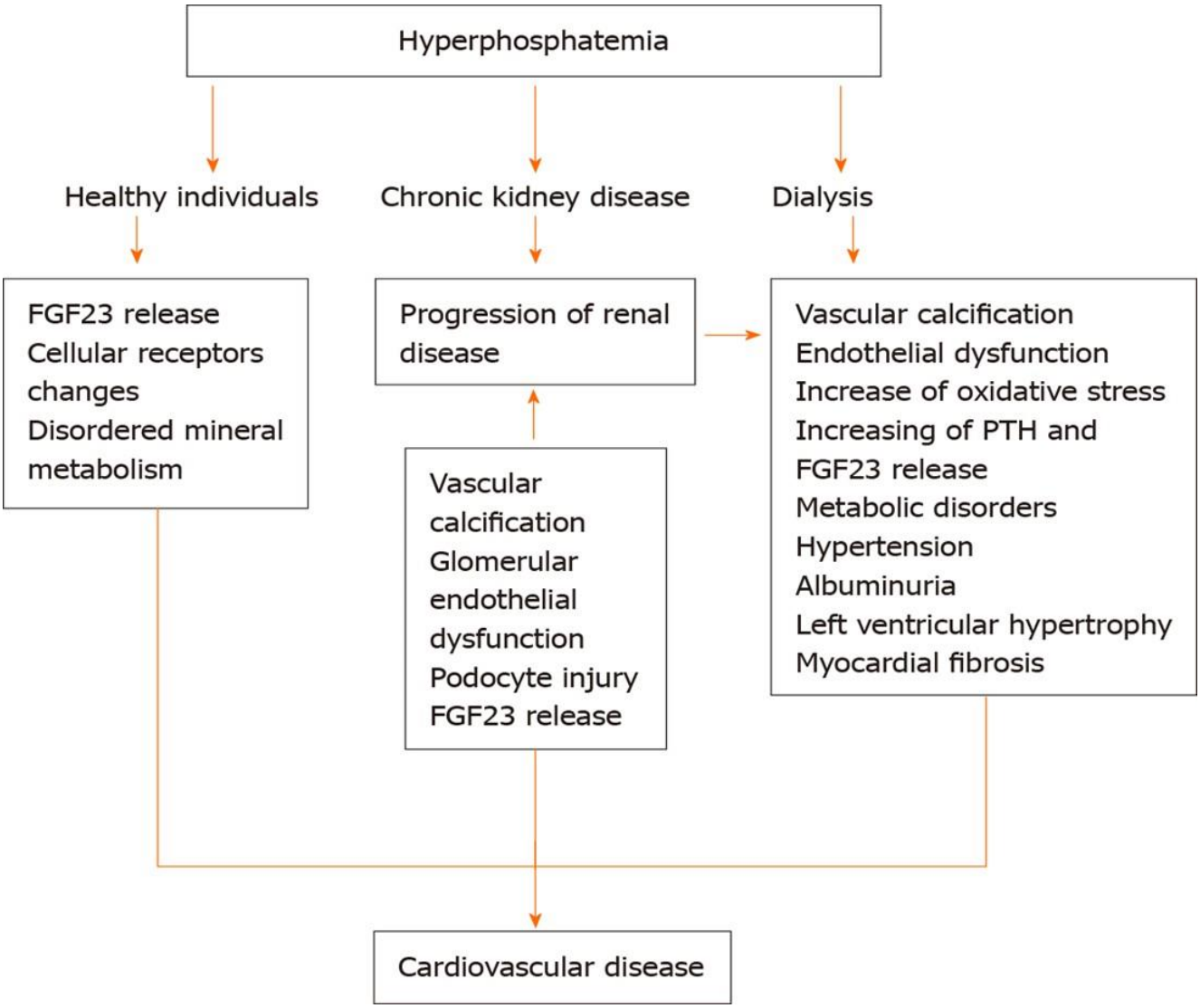


Fig.7 Relation of Cardiovascular Disease and Serum Phosphorous Levels

A study of 14,675 participants, of whom 55% were women, and who were monitored for 20 years concluded that with each 1 mg/dL increase in serum phosphate, the risk of atrial fibrillation rises by 13%.[15]

EFFECT OF PHOSPHOROUS ON RESPIRATION

It has been proposed[16] that decreased serum phosphorous levels can lead to neuromuscular syndromes by altering the concentrations of ATP(Adenosine tri-phosphate) and 2,3-DPG(2,3-diphosphoglycerate) as inorganic phosphate is required in their synthesis. Depleted ATP levels directly reduce the muscular contractility while low 2,3-DPG causes a shift of Oxygen dissociation curve to left ensuring reduced unloading of Oxygen.

According to *Aubier M et al*[17] , low blood serum phosphorous levels can result in reduced diaphragmatic contractility which can seen be improved on correcting the phosphorous levels

DRUG INTERACTIONS

ANTACIDS

Aluminium hydroxide-containing antacids bind phosphorus in the intestines, and prolonged usage of more than three months can result in hypophosphatemia. [18]

LAXATIVES

Some laxatives containing Sodium Phosphate can increase serum phosphate levels.[19]

HYPOPHOSPHATEMIA

Serum phosphate levels of less than 2.5 mg/dL is referred to as hypophosphatemia.

ETIOLOGY[20,21,22]

1. Inadequate intake of phosphate

2. Increased excretion of phosphate

3. Transfer of phosphate from the extracellular to the intracellular space

PATHOPHYSIOLOGY

1. Hypophosphatemia due to decreased intake of phosphorous

Chronically low dietary phosphate intake, intestinal malabsorption, such as chronic diarrhoea can lead to hypophosphatemia. Also, intestinal binding by exogenous substances, such as binding with aluminium and magnesium antacids, will result in the creation of aluminium or magnesium-bound phosphate salts that are non-absorbable by the body.

2. Hypophosphatemia secondary to increased phosphate excretion

i. Up to 70% of the filtered phosphate is typically reabsorption by the proximal renal tubule, and 15% is reabsorption by the distal renal tubule. Through the sodium-phosphate (NaPi) cotransporters of the proximal tubule, decreased serum phosphate concentration directly causes enhanced phosphate reabsorption. Additionally, it increases sodium-phosphate cotransporter expression and production. PTH, on the other hand, promotes phosphate excretion by inhibiting NaPi co-transporters. Consequently, hypophosphatemia can result from hyperparathyroidism.

ii. Hypophosphatemia can also result from genetic disorders such as X-linked Hypophosphatemic Rickets, which is a PHEX gene mutation that results in increased levels of Fibroblast Growth Factor 23 (FGF23), which reduces phosphate absorption.

3. Intracellular shifting of phosphate

i. When a patient who has been severely malnourished is abruptly given carbohydrates, proteins, and lipids, refeeding syndrome develops. In such a situation, insulin and glucose cause phosphate to move intracellularly, and cells absorb all free phosphate that is available in order to make ATP for energy, resulting in severe hypophosphatemia.

ii. Acute respiratory alkalosis alters cellular pH, which results in hypophosphatemia. An increase in pH activates phosphofructokinase (PFK), which in turn activates glycolysis to make ATP and take phosphate out of the cellular space. In hospitalized patients, it is the most typical cause of severe hypophosphatemia.

CLINICAL FEATURES

Typically hypophosphatemia is an incidental finding and most patients are asymptomatic.

Mild to moderate hypophosphatemia (1-2.5mg/dL) will not be clinically apparent and may present only with generalized body weakness.

Severe hypophosphatemia (<1mg/dL) may present with altered mental status, seizures, numbness, muscle pain, heart failure and muscular weakness.

MANAGEMENT

1. Measurement of Serum Phosphorous levels

2. Measurement of urinary phosphate excretion

Renal phosphate excretion can be calculated from a 24-hour urine collection or from a random urine sample using the fractional excretion of filtered phosphate (FEPO4).,

$FEPO_4 = (UPO_4 \times PCr \times 100) / (PPO_4 \times UCr)$

Where U is for urine values, and P is for plasma values, PO4 is phosphate and Cr is creatinine

Interpretation-

If the 24-hour excretion of phosphate in the urine is less than 100 mg or if FEPO4 is less than 5% means the excretion of phosphate has reduced, indicating that the cause of hypophosphatemia is either internal redistribution of phosphate or decreased intestinal absorption.

Renal phosphate wasting, which can be caused by hyperparathyroidism or a vitamin D deficit, is evidenced by 24-hour urine phosphate excretion greater than 100 mg or FEPO4 greater than 5%.

Treatment

Mild to moderate cases with serum phosphate >1mg/dL should be given phosphates orally. 1 to 3 gm (30-90 mmol) of phosphate supplements should be received by such cases every

day.[23]Patient can either be given skimmed milk (0.9mg phosphate per ml) or phosphate salts (129 mg phosphate per ml). the potential side effects include diarrhea.

Severe, symptomatic cases - Intravenous phosphate can be given to correct low serum phosphate levels of less than 1 mg/dL with 0.25 mmol/kg of phosphate, or less than 0.5 mg/dL with 0.5 mmol/kg of phosphate. The infusion rate shouldn't exceed 5 mmol/hour. Administration of intravenous phosphate might result in side effects such hypocalcemia and hyperphosphatemia.[24] It should be switched to oral replacement when the serum phosphate exceeds 1.48 mg/dL.

HYPERPHOSPHATEMIA

Hyperphosphatemia is defined as a blood phosphate level greater than 4.5 mg/dL.

ETIOLOGY

1. An abnormally high phosphorus load

2. Reduced kidney excretion of phosphate

3. Phosphate shift transcellularly

PATHOPHYSIOLOGY

1. High phosphate load:

It can be secondary to endogenous or exogenous causes,

i. Endogenous causes- Phosphate is mainly an intracellular anion therefore massive tissue breakdown from Severe Hemolysis, Rhabdomyolysis, or Tumor Lysis Syndrome can lead to the phosphate shift from intracellular to the extracellular fluid.

Tumor lysis syndrome- Cytotoxic treatment can cause tumour lysis syndrome in patients with diseases like leukaemia and lymphomas. Following treatment, the cells lyse, releasing phosphate and resulting in hyperphosphatemia. Additionally, hypocalcemia, hyperkalemia, and hyperuricemia can be associated with this.

Excessive Vitamin D - It improves the intestinal absorption of calcium and phosphate from food.

ii. Exogenous causes- This is mostly due to excessive consumption of laxatives containing phosphates.

2. Decreased renal excretion:

Reduced renal function results in less phosphate being secreted and more being retained. Renal failure is the most typical cause of hyperphosphatemia. Inorganic phosphate is much less filtered when the glomerular filtration rate (GFR) is less than 30 mL/min, which raises the serum level.

i. Hypoparathyroidism: The most common cause of hypoparathyroidism is an injury to the parathyroid gland or it's removal during anterior neck surgery.

ii. Pseudohypoparathyroidism (PHP): It is an uncommon illness that is characterised by PTH receptor resistance. On lab evaluation decreased serum calcium, elevated serum phosphate, and unreasonably high PTH levels are found.

3. Transcellular shift:

Clinical conditions like Diabetic ketoacidosis and Lactic acidosis can cause massive shifts of phosphate from intracellular to extracellular spaces.

CLINICAL FEATURES

Most of the patients are asymptomatic.

Patient can present with non-specific symptoms like Muscle cramps, tetany, joint pain, anuria, edema or can have widened pulse pressure, raised systolic blood pressure on examination.

Cataracts, conjunctivitis and Band-shaped keratopathy are examples of ocular symptoms.

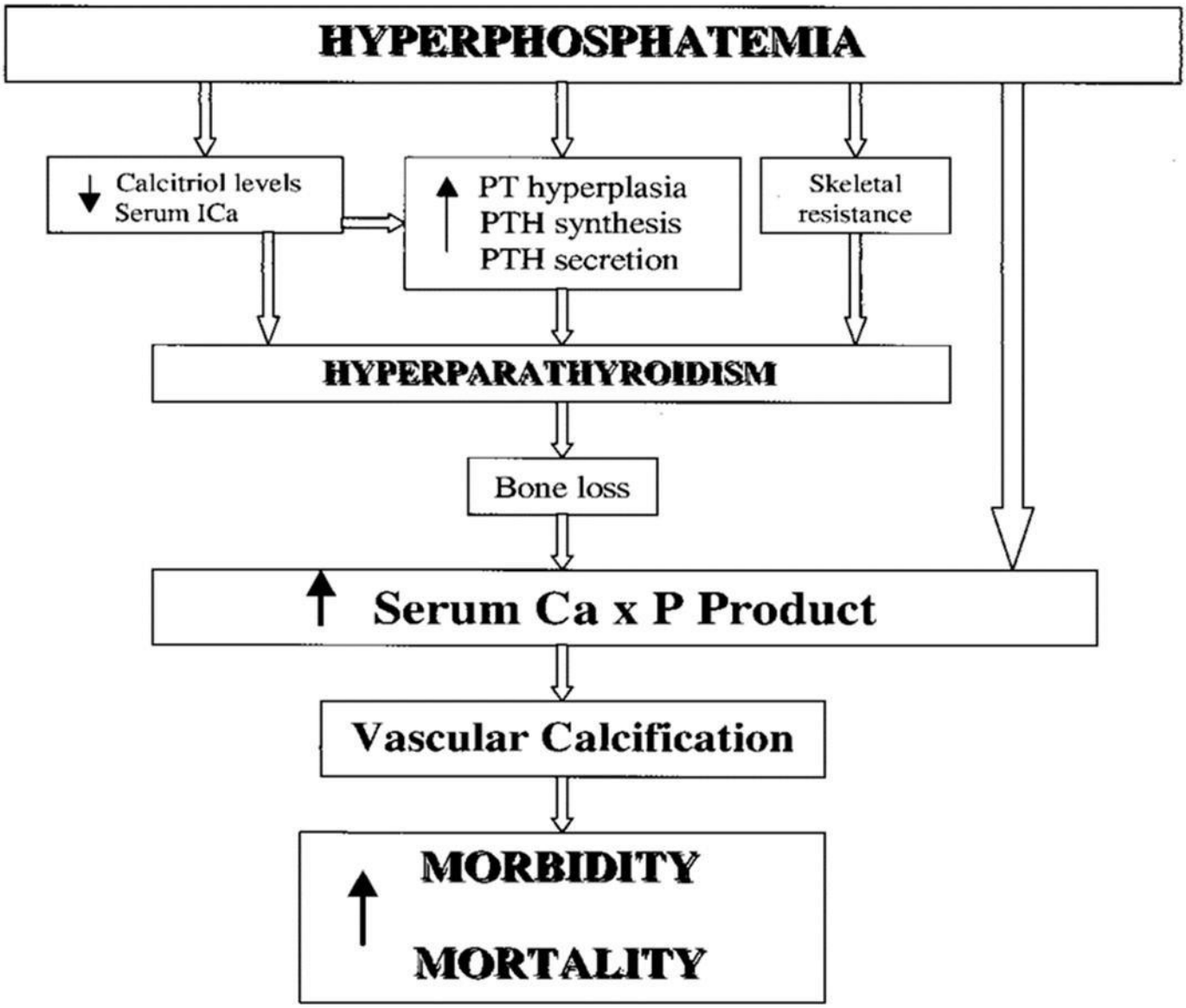


Fig.8 Hyperphosphatemia (Ref. 25)

MANAGEMENT

Investigations

Blood phosphate, vitamin D, calcium and PTH level estimation, Renal function test .Urinary phosphate and X-ray imaging

Interpretation

Raised creatinine and Blood Urea Nitrogen(BUN) with normal or elevated PTH and decreased serum calcium is suggestive of renal insufficiency.

In conditions like Vitamin D intoxication, elevated blood phosphate and calcium levels are present with raised vitamin D levels.

Tumor Lysis Syndrome can be diagnosed by elevated serum phosphorous levels with hypocalcemia ,hyperkalemia and hyperuricemia.

Rhabdomyolysis is suggested by elevated serum uric acid and creatine phosphokinase levels.

To differentiate between extra-renal and renal causes assessment of urinary phosphate should be done.

X-ray imaging can be used to look for metastatic calcifications.

MONITORING

According to KDIGO guidelines,

All patients with an estimated glomerular filtration rate (eGFR) less than 60 mL/min/1.73 m^2, serum levels of phosphorus, calcium, parathyroid hormone (PTH), and vitamin D should be measured.

Serum phosphate and calcium levels should be checked every six to twelve months if the estimated glomerular filtration rate (eGFR) is between 30 and 59 mL/min/1.73 m^2.

Serum phosphate and calcium levels should be evaluated every three to six months in patients with estimated eGFRs of 15 to 29 mL/min/1.73 m^2. [26]

TREATMENT

1. Dietary Restriction

KDIGO advises consuming between 800 and 1000 mg of phosphate per day along with 1.2 g of protein per kilogramme of body weight each day. [27]

2. Phosphate Binders[6]

When serum phosphorus levels are consistently high and do not decrease with food restriction, phosphate binders are the preferred treatment. By exchanging phosphate, an anion, with an active cation like carbonate, citrate, or acetate to generate nonabsorbable salts that can subsequently be expelled in faeces, these binders reduce the absorption of dietary phosphate in the gastrointestinal tract.

When phosphate levels are higher than 6 mg/dl, they can also be utilised in conjunction with dietary restriction.

i. Calcium-based binders

Calcium-based binders like calcium carbonate and calcium acetate are efficient and do not have the negative consequences associated with aluminum-based compounds.

Disadvantage- A positive calcium balance that results from them may cause the development of ectopic calcifications in the arteries.

ii. Sevelamer

Sevelamer e.g., Sevelamer hydrochloride and Sevelamer carbonate acts by exchanging phosphate with Hydrochloric Acid or carbonate in the gastrointestinal tract resulting in formation of non-absorbable compound and excretion through faeces.

iii. Magnesium Carbonate

Magnesium carbonate has a good tolerance and effectively lowers serum phosphate levels. Additionally, it lessens the development of hydroxyapatite, which lowers vascular calcification.

iv. Lanthanum Carbonate

It is available in chewable forms and is devoid of calcium.

Mechanism- The non-absorbable chemical lanthanum phosphate is created when lanthanum carbonate interacts with phosphate and is then expelled.

v. Ferric Citrate

Mechanism- In the digestive tract, ferric citrate converts citrate to ferric phosphate, which is nonabsorbable and hence expelled in the faeces.

COMPLICATIONS

Increased phosphates make complex with serum calcium which results in decreased ionized calcium in the body thereby leading to release of parathyroid hormone which results in secondary hyperparathyroidism.

The phenotypic transformation of the smooth muscle cells in the arteries to osteoblast-like cells caused by hyperphosphatemia, hypercalcemia, and high parathyroid concentration results in medial calcification. Large vessels, like the aorta, become more stiffened by calcification, which causes hypertension and an increase in pulse pressure..[28,29]

The 1-alpha hydroxylase, which is typically necessary for the activation of vitamin D, is inhibited by elevated phosphate levels. Reduced intestinal absorption of calcium from a decrease in active vitamin D causes subsequent bone discomfort and fractures.

Azotemia and hyperuricemia are frequent metabolic side effects of tumour lysis syndrome.

Myoglobin produced from injured tissue during rhabdomyolysis might lead to acute renal injury induced by heme pigment.

SOFA SCORE[30] **(**Sequential Organ Failure Assessment Score)

The SOFA scoring(Table 2) predicts mortality based on the degree of impairment in the functioning of six organ systems—respiratory, coagulation, hepatic, cardiovascular, central nervous system and renal.

All 6 symptoms are assessed and given a value of 0 to 4, where 0 stands for normal and 4 refers to the most abnormal.

The score ranges from 0 to 24, higher the score worse the clinical outcome.

SOFA score can be applied to all admitted critical care patients to assess their outcome.

Table 2. SOFA SCORE

RESPIRATORY SYSTEM	
PaO_2/FiO_2(mmHg)	SOFA score
>400	0
<400	1
<300	2
<200 with respiratory support	3
<100 with respiratory support	4
NERVOUS SYSTEM	
Glasgow Coma Scale	SOFA score
15	0
13-14	1
10-12	2
6-9	3
<6	4
CARDIOVASCULAR SYSTEM	
Mean arterial Pressure(MAP)or administration of vasopressors required	SOFA score
MAP>70 mmHg	0
MAP<70 mmHg	1
Dopamine 5 microgram/kg/min or lesser OR Dobutamine(any dose)	2

Dopamine >5 microgram/kg/min OR Epinephrine 0.1 microgram/kg/min or lesser OR Norepinephrine 0.1 microgram/kg/min or lesser	3
Dopamine >15 microgram/kg/min OR Epinephrine 0.1 microgram/kg/min OR Norepinephrine >0.1 microgram/kg/min	4
LIVER	
Bilirubin(mg/dL)	SOFA score
<1.2	0
1.2-1.9	1
2.0-5.9	2
6.0-11.9	3
>12	4
COAGULATION	
Platelets X 10^3/ml	SOFA score
>150	0
<150	1
<100	2
<50	3
<20	4
RENAL SYSTEM	
CREATININE(mg/dL)	SOFA score
<1.2	0

RENAL SYSTEM (CONTD.)	SOFA score
1.2-1.9	1
2.0-3.4	2
3.5-4.9 OR urine output<500ml/day	3
>5.0 or urine output <200ml/day	4

Minimum Score = 0 Maximum Score=24

Moreno et al(31) has concluded that the initial SOFA score can estimate the degree of organ dysfunction present at the time of admission. Also it can be used to evaluate any further decline in organ function during the ICU stay. They also suggested that there is a strong correlation of these parameters with outcome of the patient. Another study of 354 patients has shown that mean total SOFA score i.e. the sum of SOFA scores divided by the total days of stay in the critical care ward is correlated with the mortality of the patient.[32]

Hence, with the help of this scoring system, high risk groups of critically ill patients can be located and treatment measures can be targeted at them to reduce overall morbidity and mortality.

MECHANICAL VENTILATION

When critical illness patients develop respiratory failure, mechanical ventilation is frequently required. The standard spontaneous ventilation can be replaced or supplemented by mechanical ventilation. On the basis of clinical indicators or recommendations, mechanical ventilation can be initiated (Table 3).

Table 3. Indication of Mechanical Ventilation

Criterion	**Measurement**
Direct measurement	
Arterial oxygen tension. Arterial CO_2 tension	<50 mm Hg on room air >50 mm Hg without metabolic alkalosis
Derived indices	
PaO_2/FiO_2 PA–aO_2 ratio gradient Vd/Vt	<300 mm Hg >350 mm Hg >0.6
Clinical indices	
Respiratory rate	>35 breaths/min
Mechanical indices	
Tidal volume Vital capacity Maximum inspiratory force	<5 mL/kg <15 mL/kg >−25 cm H2 O (eg, –15 cm H_2O)

The following are the main indications for mechanical ventilation: [33]

1. The protection of the airway in a patient who has experienced trauma or an oropharyngeal infection

2. Respiratory failure with hypercapnia brought on by a reduction in minute ventilation

3. Hypoxemic respiratory failure caused by failed oxygenation

4. Cardiovascular distress where mechanical ventilation can reduce breathing's energy demands

5. Expectant course, such as expected patient deterioration or an upcoming transfer

Mechanism

Mechanical ventilator operated by giving positive pressure breaths to achieve a set tidal volume(TV) and hence is also influenced by airway's resistance..[33]

Stages of mechanical ventilation.

1. **Trigger phase**- It refers to the initiation of inhalation triggered by the set parameters by mechanical ventilation or patient's efforts.

2. **Inspiratory phase**- It includer air inhalation by the patient.

3. **Cycling phase**- This phase comprises of cessation of inhalation for few seconds before expiration resumes.

4. **Expiratory phase**- It is the passive phase of exhalation of air by the patient.

Modes of ventilation[34]

1. Assisted Control (AC)
2. Synchronized Intermittent Mechanical Ventilation (SIMV)
3. Pressure Support Ventilation (PSV)

According to the patient's clinical condition and respiratory efforts ventilator mode is adjusted. The parameters like Respiratory Rate(RR), positive end-expiratory pressure (PEEP), fraction of inspired oxygen ($Fi0_2$) and Inspiratory Flow Rate is set.[34]

FiO_2- It refers to the fraction of the inspired air and should always be set to the lowest level to achieve a blood oxygen saturation level of 92% to 96%.[35]

PEEP- It is used to raise the Functional Residual Capacity of lung and also opens the collapsible alveoli.

RR- It is adjusted such a way that patient can achieve normocapnia.

Inspiratory flow rate- its refers the rate at which inspiration can be provided.[34]

All patients who are on mechanical ventilation should have propped up position[33] with head elevation and End-Tidal Carbon Dioxide (ETCO2) should be monitored.

HEART LUNG INTERACTION DURING MECHANICAL VENTILATION[36]

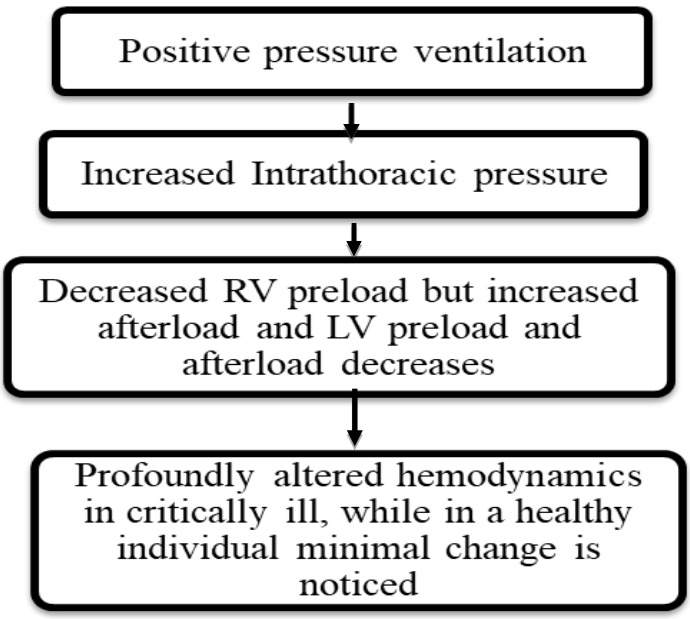


Fig. 9 Effect of Mechanical Ventilator on Cardio-Pulmonary System

To prevent the ventilator associated complications early weaning from the ventilator is required.

Weaning

It consists of two phases.

1. The first phase includes “Readiness Evaluation”. In this phase weaning and other clinical parameters are assessed to determine whether the patient can breathe effectively without assisted support.

2. The second phase is “Spontaneous Breathing Trial”. In this phase ventilatory support is removed and patient’s efforts are tested.

Patients can be considered for weaning if their pH is greater than 7.25, their arterial oxygen saturation is normal while receiving FiO_2 that is less than 0.5, they can breathe on their own, their hemodynamics are stable, and there are no recent symptoms of myocardial ischemia.

Table 4. Criteria for Weaning or Extubation:

Criterion	**Measurement**
Inspiratory pressure	<25 cm H_2O
Tidal volume	>5 mL/kg
Vital capacity	>10 mL/kg
Minute ventilation	<10 mL
Rapid shallow breathing index	<100

COMPLICATIONS OF MECHANICAL VENTILATION

Barotrauma refers to the physical damage caused to the body tissues caused by the pressure difference in the enclosed cavities in the body.

The patients with high peak inflation volume and large tidal volumes are predisposed to develop barotraumas.

The most frequent cause of barotrauma is alveolar rupture, which results in the collection of extra alveolar air. Complications such subcutaneous emphysema, pneumothorax, and pneumomediastinum may develop from this.(38)

Patients with predisposed lung pathology, such as chronic obstructive pulmonary disease (COPD), asthma,acute respiratory distress syndrome(ARDS), interstitial lung disease (ILD) and pneumocystis jiroveci pneumonia are at increased risk of experiencing barotrauma from mechanical ventilation.(39)

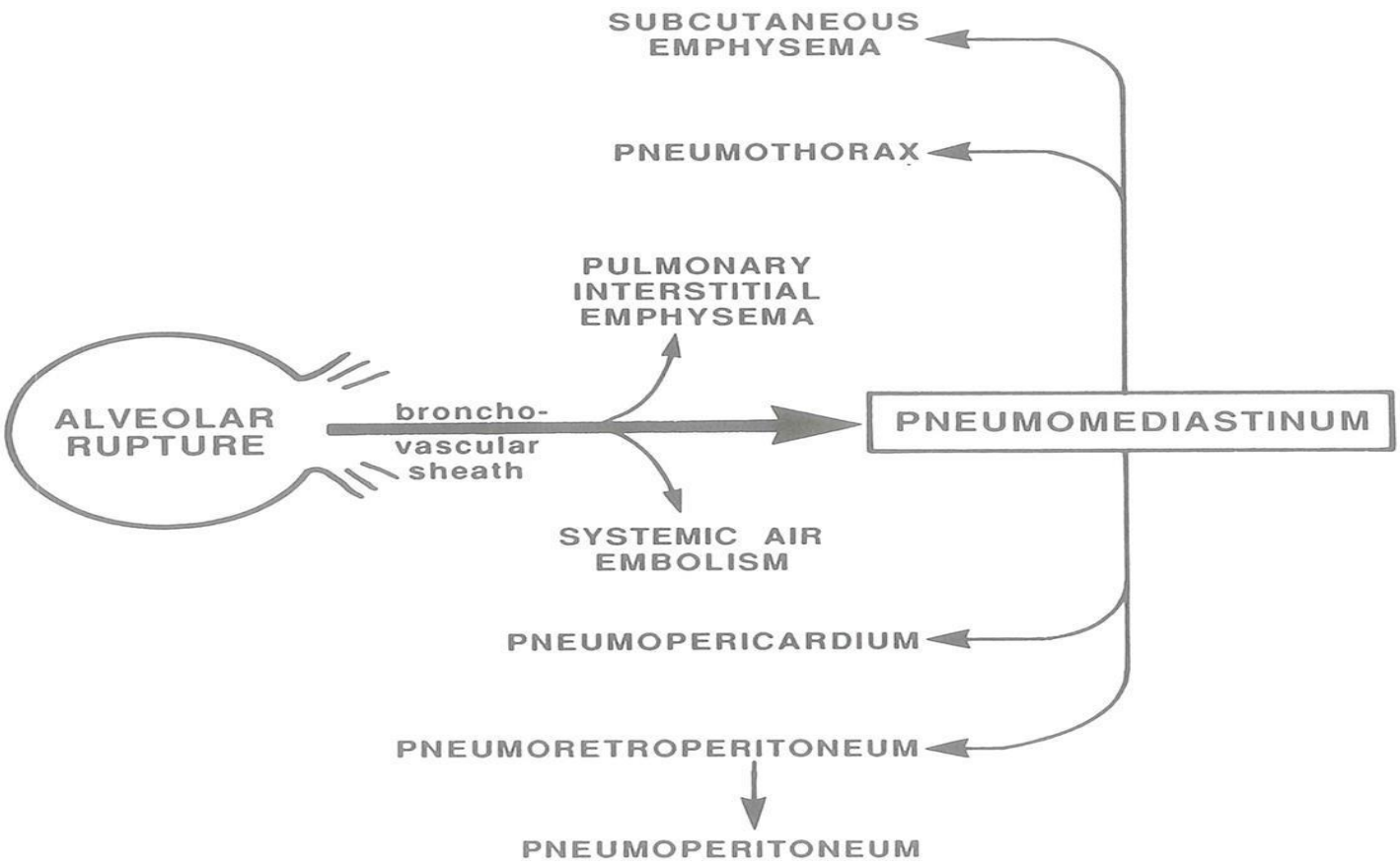


Fig.10 Pathogenesis and manifestation of Barotrauma [37]

PNEUMOTHORAX [40]

It is an early consequence of pulmonary barotrauma. Since it is an emergency, immediate action is required. Usually, the patient will complain of sudden shortness of breath, chest pain, or a sudden decline in saturation.

Clinical Findings,

Tachycardia, Tachypnea

Tracheal shift to opposite side

Hyperresonance to percussion

Absent breath sounds

The treatment is thoracotomy tube installation after an immediate needle decompression to remove the pneumothorax.

SUBCUTANEOUS EMPHYSEMA[40]

Subcutaneous emphysema is another complication of barotrauma. It can lead to compartment syndrome. Compartment syndrome should be suspected in any patient who has hemodynamic instability. Airway pressure reduction and surgical decompression are used in treatment.

BRONCHOPLEURAL FISTULA

During mechanical ventilation, persistent bronchopleural air leaks, also known as bronchopleural fistulas (BPF), may occur after either spontaneous alveolar rupture or direct laceration of the visceral pleura.

ACUTE RESPIRATORY DISTRESS SYNDROME[41]

Fighting the ventilator, as it is widely known, is a regular occurrence during mechanical breathing and could be a sign of a serious issue. Tachypnea, the involvement of supplementary respiratory muscles, thoracic cage-abdominal asynchrony, tachycardia, hypotension, and arrhythmia are among the physical indicators of anxiety or agitation.

VENTILATOR ASSOCIATED PNEUMONIA(VAP)[42]

The development of pneumonia more than 48 hours after the beginning of mechanical ventilation is referred to as VAP. A new pulmonary infiltration and at least one of the following findings are required for the diagnosis of VAP:

1. evidence of pneumonia on histopathologic examination

2. Positive pleural fluid or blood cultures that match the organisms in the tracheal aspirate

A positive culture from an endotracheal aspiration must contain at least 105 colony-forming units (CFU) per millilitre or a positive culture from a bronchoalveolar lavage (BAL) must contain at least 10 to the power 4 CFU/mL, or a positive culture from a protected brush sampling must contain at least 10 to the power 3 CFU per millilitre.

3. Leukocytosis and new-onset fever

4. A tracheal aspirate with pus

The new infiltration must appear at least 48 hours following the start of mechanical ventilation in order to be deemed ventilator-associated [43]

Given that studies have demonstrated an 88% mortality attributable directly to the infection and accompanying consequences, the combination of ARDS and pneumonia is highly harmful.

PLEURA EFFUSION

Patients who are mechanically ventilated frequently experience pleural effusions.

Systemic inflammatory response syndrome is typically associated with aggressive fluid resuscitation therapy and leaky capillaries, which together frequently result in effusion. Recovery is challenging for critically ill individuals who experience pleural effusion in the presence of respiratory failure. Additionally, it might exacerbate cardiopulmonary insufficiency.

VENTILATOR INDUCED LUNG INJURY(VILI)

A change in the alveolar-capillary barrier's permeability leads to ventilation-induced pulmonary edema.

High volume lung injury or low volume lung injury might happen depending on how long the ventilation lasts.

Increased vascular transmural pressure mechanisms

During mechanical ventilation, there can be an increase in fluid filtration by this mechanism at both extra-alveolar [44] and alveolar[45] locations.

Surfactant inactivation causes increased filtration across alveolar microvessels, while an increase in lung capacity may generate an increase in transmural pressure in extra-alveolar arteries.

Effects of surfactant inactivation

Surfactant inactivation and higher alveolar surface tension may increase alveolar epithelial permeability to tiny solutes in addition to their effects on fluid filtration. Because of the increased radial traction on the pulmonary microvessels, increasing surface tension may potentially change endothelial permeability [46]

Role of inflammatory mediators.

It has been proposed that excessive inflation during mechanical ventilation can encourage the release of cytokines or bacteria into the blood, providing mechanical ventilation with a causal role in multi-organ dysfunction [47,48,49]

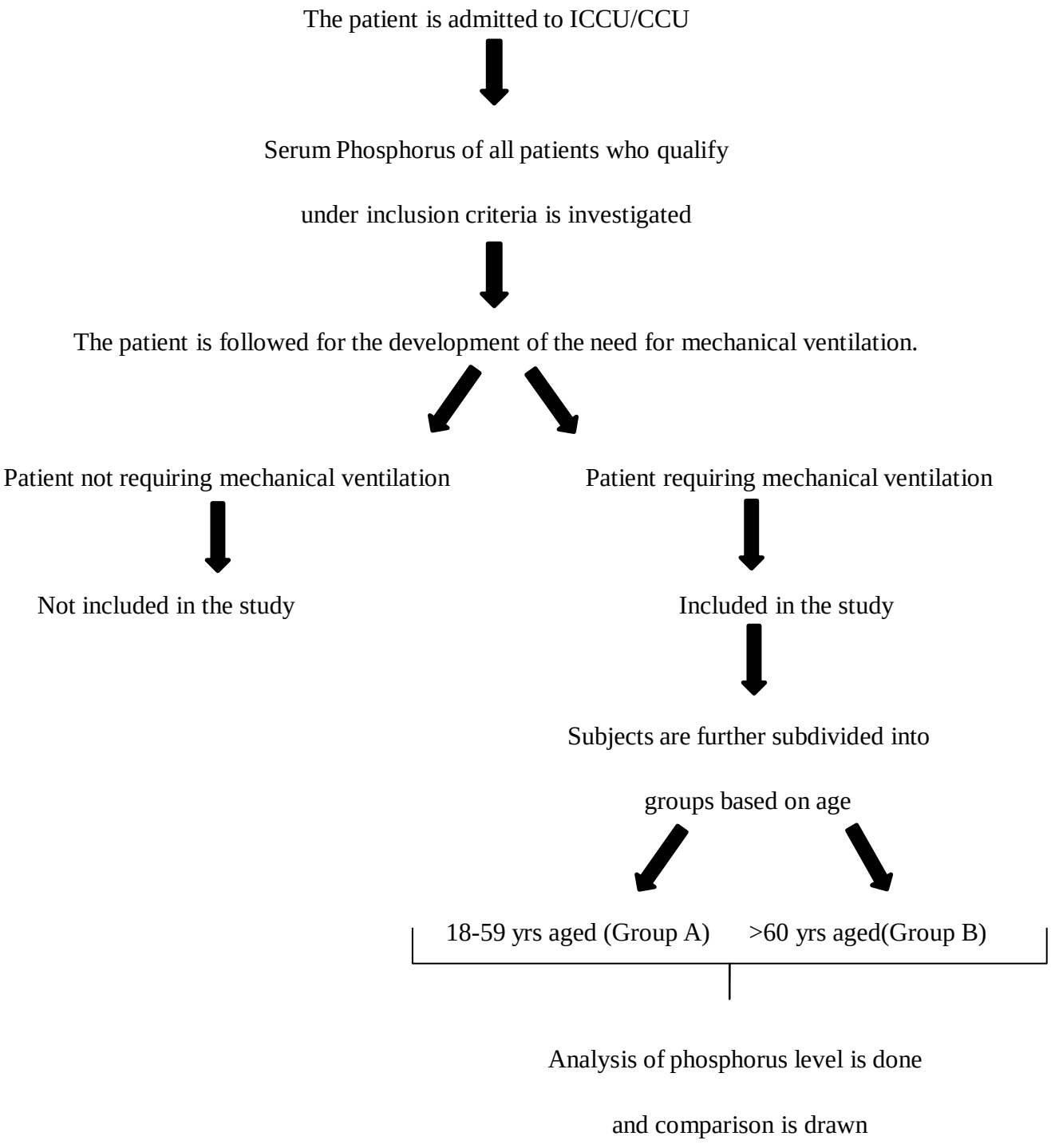
SOURCE OF DATA:

- Data will be collected from all the subjects with medical emergencies who are above 18 years and irrespective of sex admitted in the ICU/CCU wards in BLDE (DU) Shri B. M. Patil Medical College and Research Centre, Vijayapura. They will then be followed till discharge. The study assesses the serum Phosphorus level's relation to the need for mechanical ventilation among adults and the elderly. The sequential organ failure assessment (SOFA) score will be applied to all the subjects to assess the severity of illness. It also measures the outcome after correction of phosphorus abnormalities.
- The period of study is from January 2021 to June 2022.

METHOD OF DATA COLLECTION

- Adult Patients presenting with critical illnesses to BLDE (DU) Shri B. M. Patil Medical College and Research Centre, Vijayapura, were evaluated.
- SOFA scoring of the patient was done on admission and all routine and relevant investigations along with serum phosphorous was assessed.
- The patients who got admitted to the critical care wards of ICCU/CCU were followed for development of need of mechanical ventilation.
- Those patients who required mechanical ventilator were included into the study.
- Further, the patients were grouped into Group A(Young Adults) and Group B(Elderly) according to their ages.
- Patients outcome was analysed and association with admission serum phosphorous levels were evaluated.

METHODOLOGY



STUDY DESIGN:

HOSPITAL BASED CROSS-SECTIONAL STUDY

INCLUSION CRITERIA

- All adult patients aged more than 18 years and above, irrespective of sex, admitted to a critical care unit and subsequently requiring mechanical ventilation.

EXCLUSION CRITERIA

- Diabetic ketoacidosis
- Malnutrition
- Hyperparathyroidism/hypoparathyroidism
- Leukemias & Lymphomas
- Acute/Chronic Renal Failure
- Conditions requiring elective mechanical ventilation(e.g., Neuromuscular syndrome)
- The patient who does not require mechanical ventilation

INVESTIGATIONS

1. Serum Phosphorus level
2. Complete Blood Count
3. E.S.R.
4. Serum Electrolytes – Na+/K+/Ca2+
5. Liver Function Tests
6. Sugar levels(Random/ Glycated hemoglobin)
7. Chest X-ray PA View

8. E.C.G.
9. Echocardiography
10. Urine – Alb/Sugar /micro

STATISTICAL ANALYSIS

Assuming that 21% of the population subjects have the factor of interest, the study would require a sample size of **64** (32 adults and 32 elderly) for estimating the expected proportion with 10% absolute precision and 98% confidence.

SAMPLE SIZE

With the anticipated Proportion of Hypophosphatemia among mechanically ventilated patients 21.2%[18] in the population, the study would require a sample size of 64 patients with a 95%level of confidence and 10% absolute precision. Formula used

$$n=\frac{z^2\, p*q}{d^2}$$

Where z= z statistic at α level of significance

d^2= Absolute error

p= Proportion rate

q= 100-p

Statistical Analysis

- The data obtained will be put in a Microsoft Excel sheet, and statistical analysis will be done using a statistical package for the social sciences (Version 20).
- Results will be presented as Mean (Median) ±SD, counts and percentages, and diagrams.
- For normally distributed continuous variables will be compared using Independent t-test. For not normally distributed variables, the Mann-Whitney U test will be used.

- More than two subgroups will be compared using ANOVA/Kruskal Alli's test with post hoc.Categorical variables will be compared using the Chi-square test.
- ROC analysis will be used to establish a cut-off for serum phosphorus in predicting weaning success.
- The p-value of <0.05 will be considered statistically significant. All statistical tests will be performed two-tailed.

RESULTS

64 critically ill ICU patients who required mechanical ventilation over a two-year period were included in the study. The patients were divided into groups based on their ages. Thirty-two patients were in Group A (young adults) and other thirty-two were in Group B (elderly).

Table 5. AGE AND SEX DISTRIBUTION

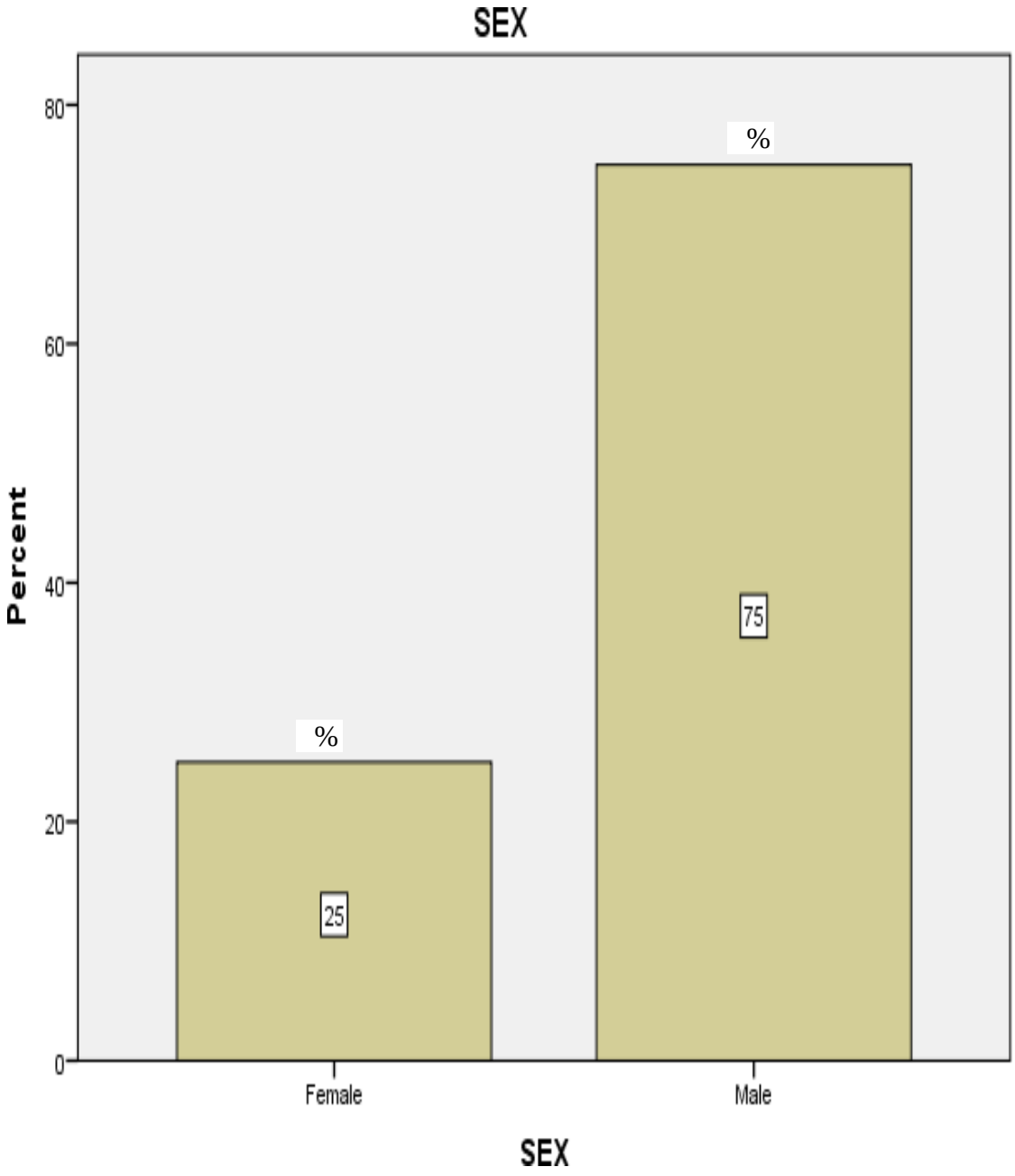
GROUP	AGE GROUP	MALE	PERCENT	FEMALE	PERCENT
YOUNG (18-59YRS) (GROUP A)	18-29	3	4.68%	3	4.68%
	30-39	9	14.06%	3	4.68%
	40-49	6	9.37%	1	1.56%
	50-59	6	9.37%	1	1.56%
ELDERLY (60YRS OR ABOVE) (GROUP B)	**60-69**	**13**	**20.31%**	**5**	**7.81%**
	70-79	7	10.93%	2	3.12%
	80-89	3	4.58%	1	1.56%
	90-99	1	1.56%	0	0.00%
TOTAL		48	75%	16	25%

Table 5 shows that the majority of the patients i.e.,28.12 % in the study belonged to the elderly age group 60-69yrs. Among these 20.31% were males while remaining 7.81% were females.

Meanwhile, in the younger group (18- 59yrs) most subjects i.e.,14.06 % males and 4.68% females belonged to the age group 30-39yrs.

The oldest subject in the study was a 90yr old male.

Graph 1 shows that the study had more males(75%) than females(25%).



Graph 1. Sex Distribution

The study also compared the comorbidities in the two groups.

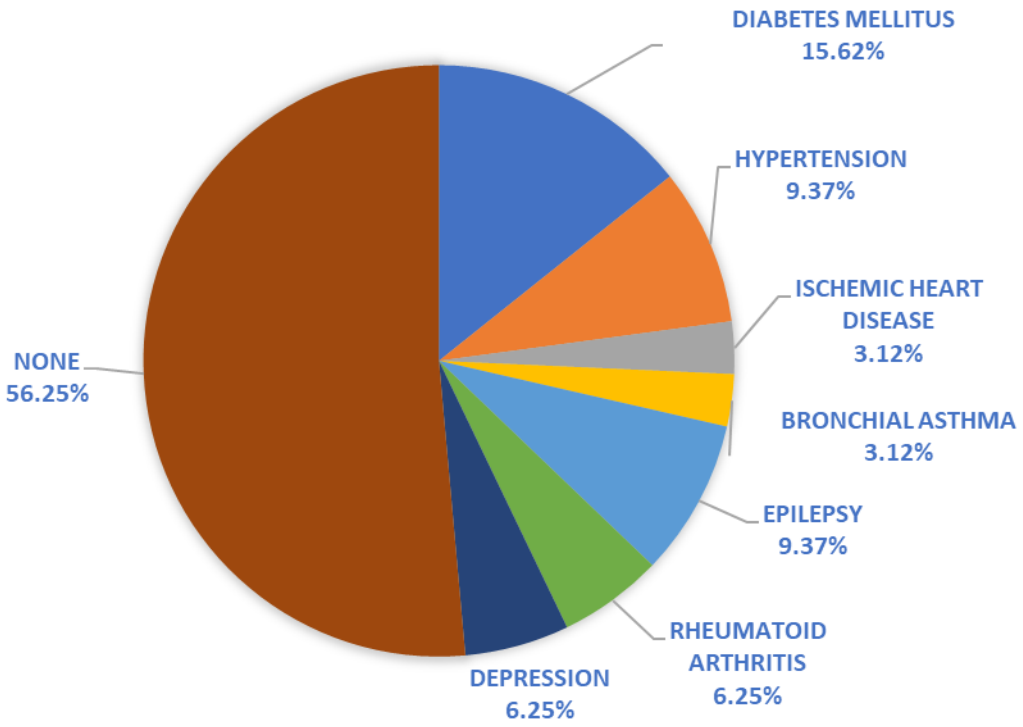


Fig. 11 Comorbidities in Group A (Young Adults)

Most of the young patients(Group A) admitted in the critical care who required mechanical ventilation had Diabetes Mellitus(15.62%), Hypertension(9.37%), Epilepsy(9.37%), Rheumatoid Arthritis(6.25%), Depression(6.25%), Ischemic Heart Disease(3.12%) and Bronchial Asthma(3.12%) while majority of the young patients(56.25%) had no accompanying illnesses.(Fig. 11)

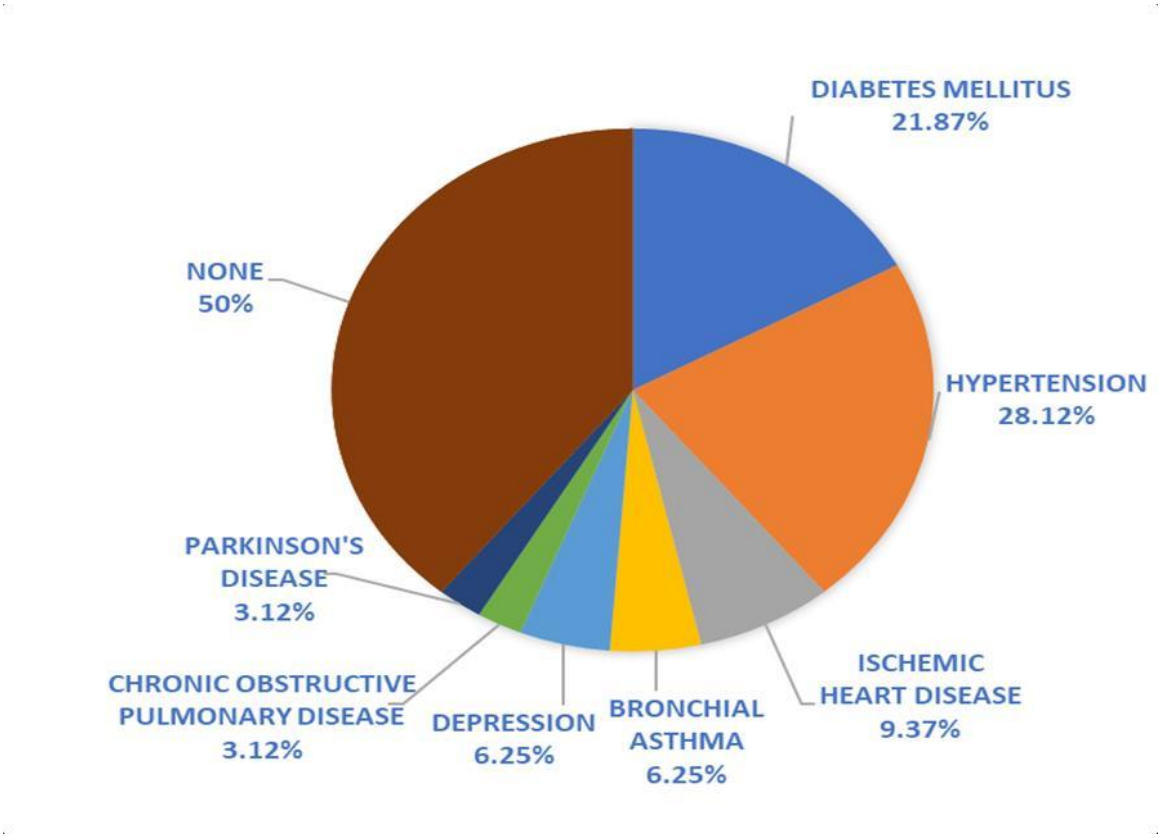


Fig.12 Comorbidities in Group B (Elderly)

On the contrary, most of the elderly(Group B) had been suffering from Hypertension(28.12%) followed by Diabetes Mellitus(21.87%) and others had Ischemic Heart Disease(9.37%), Bronchial Asthma(6.25%), Depression(6.25%), Chronic Obstructive Pulmonary Disease(3.12%) and Parkinson's Disease(3.12%) while the remaining 50% were devoid of any comorbidity.(Fig.12). Table 6 showing no relation between the age and comorbidities.

Table 6.

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	21.784[a]	17	0.193
Likelihood Ratio	29.570	17	0.030
N of Valid Cases	64		

Where 'N' is Total number of cases

TABLE 7. DIAGNOSIS AT ADMISSION IN YOUNG

DIAGNOSIS(Group A)	NUMBER (N=32)	PERCENT
POISONING	10	31.25%
CEREBROVASCULAR ACCIDENT	6	18.75%%
SEIZURE DISORDER	5	15.62%
INFECTIONS	4	12.50%
MUSCULOSKELETAL INJURIES	2	6.25%
ALCOHOLIC LIVER DISEASE	2	6.25%
CARCINOMA	2	6.25%
AUTOIMMUNE DISEASE	1	3.12%

Majority of young patients requiring mechanical ventilation in our study were provisionally diagnosed as poisoning cases(31.25%). Among others were cases of cerebrovascular

accident(15.62%), seizure disorder(15.62%), infections(15.62%), musculoskeletal disease(6.25%), alcoholic liver disease(6.25%), carcinomas(6.25%) and autoimmune disease(3.12%).(Table 7)

Among the 10 poisoning cases, most commonly encountered case was Organophosphorus Compound Poisoning seen in 4 patients, followed by Amitraz Poisoning seen in 2 patients, while 1 patient each of Pyrethroid, Emamectin Benzoate and Azathioprine Drug Poisoning. Also one case of Neurotoxic Snake Envenomation was included.

Six cerebrovascular accidents had been noted in the group A among which 5 were ischemic infarcts involving Thalamocapsular, Left Middle Cerebral Artery region, Bilateral Temporo-occipital, Right Capsulo-Ganglionic and Bilateral ponto-cerebellar infarcts. The other one case was of Right Temporal Haemorrhagic Stroke.

All 5 cases of seizure disorders were diagnosed Status Epilepticus.

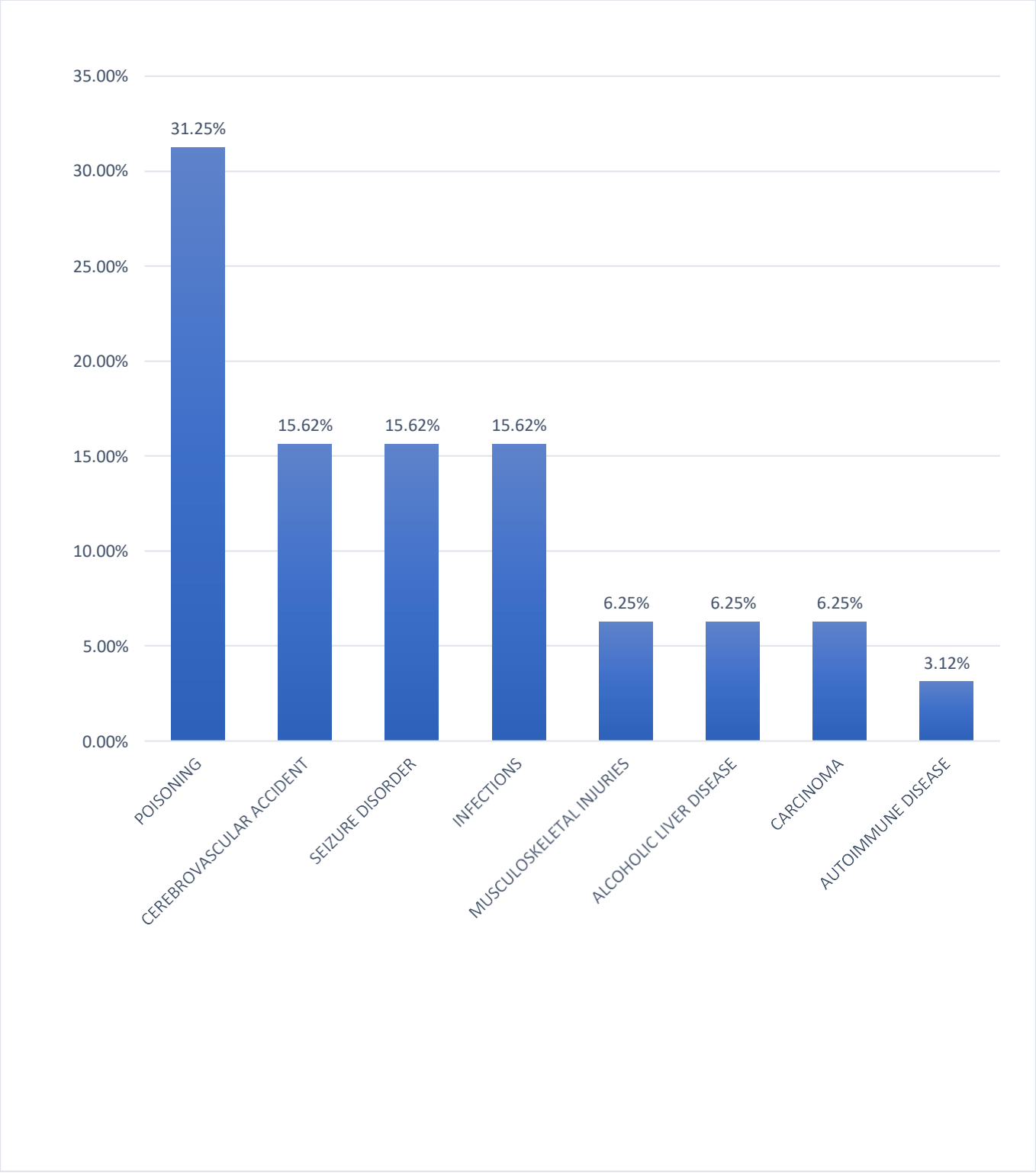
Of all 4 infectious cases, 2 were of Rhinocerebral Mucormycosis secondary to COVID 19 infection, one each with Tubercular Meningitis and Sepsis secondary to cellulitis.

Abdominal Stab Injury and Snapping Scapular Syndrome were among the two musculoskeletal injuries.

The 2 cases of Chronic Liver Disease were secondary to chronic alcoholism.

Two patients were diagnosed with Carcinoma. One was Squamous Cell Carcinoma of Buccal Mucosa, while other had Central Neurocystoma.

Of all 32 young adults, one patient had Autoimmune Encephalitis(Graph 2).



Graph 2. Diagnosis at admission (Group A)

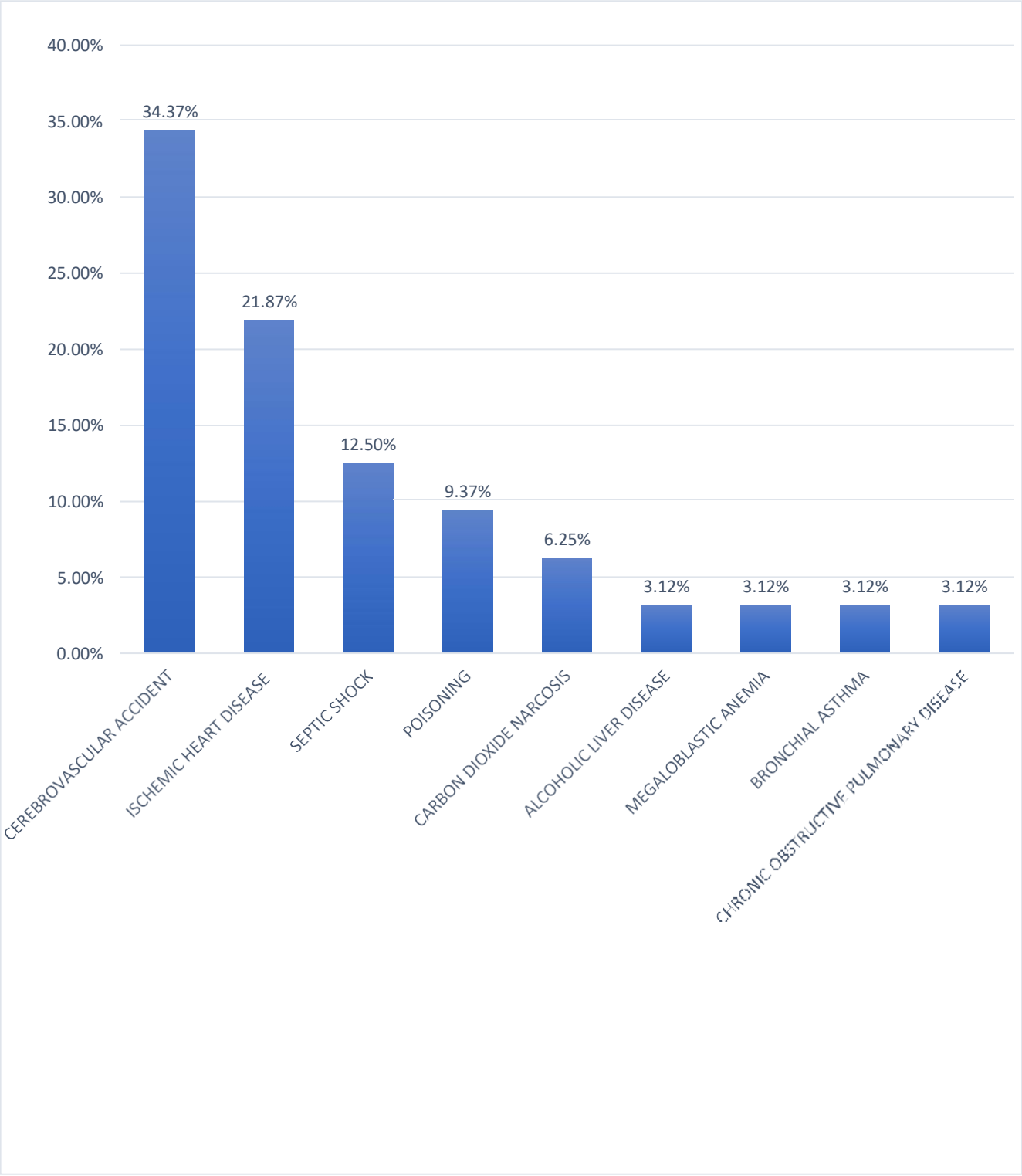
Table 8. DIAGNOSIS AT ADMISSION IN ELDERLY

DIAGNOSIS(Group B)	**NUMBER (N=32)**	**PERCENT**
CEREBROVASCULAR ACCIDENT	11	34.37%
ISCHEMIC HEART DISEASE	8	25.00%
SEPTIC SHOCK	4	12.50%
POISONING	3	9.37%
CARBON DIOXIDE NARCOSIS	2	6.25%
ALCOHOLIC LIVER DISEASE	1	3.12%
MEGALOBLASTIC ANEMIA	1	3.12%
BRONCHIAL ASTHMA	1	3.12%
CHRONIC OBSTRUCTIVE PULMONARY DISEASE	1	3.12%

Table 8 shows that the most common diagnosis among elderly was Cerebrovascular Accident (34.37%) followed by Ischemic Heart Disease(25%), Septic Shock(12.50%), Poisoning(9.37%), Carbon Dioxide Narcosis(6.25%), Alcoholic Liver Disease(3.12%), Megaloblastic Anemia(3.12%), Bronchial Asthma(3.12%), Chronic Obstructive Pulmonary Disease(3.12%).

In Group B, most elderly had Cerebrovascular Accident. Majority i.e 4 patients had Posterior Circulation Stroke. Two of them had Left Fronto-temporo-parietal infarct. Other 2 had Right Middle Cerebral Artery infarct and 1 had ischemic Stroke. Only 2 patient had Left Capsulo-Ganglionic Bleeds.

Among 8 patients with Ischemic Heart Disease, 5 developed Cardiogenic Shock, while 1 developed Pulmonary Edema. Of the other two, 1 had ST segment Elevation Myocardial Infarction(STEMI) and other one had Non-ST elevation Myocardial Infarction(NSTEMI).



Graph 3. Diagnosis at admission(Group B)

Septic Shock was diagnosed in 4 cases of whom 3 patient developed the shock secondary to pulmonary infection while the other one had gastroenteritis.

Emamectin Benzoate, Organophosphorous and Amitraz Compound Poisoning were among the three poisoning cases in group B.

2 patients had carbon dioxide narcosis secondary to obstructive sleep apnoea.

Also, one case each of Decompensated Alcoholic Liver Disease, Severe Megaloblastic Anemia, Type 2 Respiratory Failure secondary to Bronchial Asthma, and Cor pulmonale secondary to Chronic Obstructive Pulmonary Disease were included in the study (Graph 3).

CHEST X-RAY ON ADMISSION

Table 9 shows that 50% of admitted subjects had normal chest x-ray, 15.62% had pulmonary edema, 12.50% had pulmonary consolidation, 9.37% had pulmonary artery hypertension, 4.68% had pleural effusion, 3.12% had emphysematous lung and other 1.56% had tuberculosis and fibrosis.

Graph 4 compares the chest X-ray findings in the two groups.

Table 9. Chest X-Ray Findings On Admission

FINDINGS	GROUP			TOTAL (N=64)
		A(n=32)	B(n=32)	
NORMAL	Count	17	15	32
	%	53.12%	46.87%	50.00%
PULMONARY EDEMA	Count	4	6	10
	%	12.50%	18.75%	15.62%
CONSOLIDATION	Count	4	4	8
	%	12.50%	12.50%	12.50%
PULMONARY ARTERY HYPERTENSION	Count	2	4	6
	%	6.25%	12.50%	9.37%
PLEURAL EFFUSION	Count	3	0	3
	%	9.37%	0.00%	4.68%
EMPHYSEMA	Count	1	1	2
	%	3.12%	3.12%	3.12%
TUBERCULOSIS	Count	1	0	1
	%	3.12%	0.00%	1.56%
APICAL FIBROSIS	Count	0	1	1
	%	0.00%	3.12%	1.56%

Graph 4. Bar chart showing the comparison of Chest X-Ray Findings between Group A(Young Adults) and Group B(Elderly)

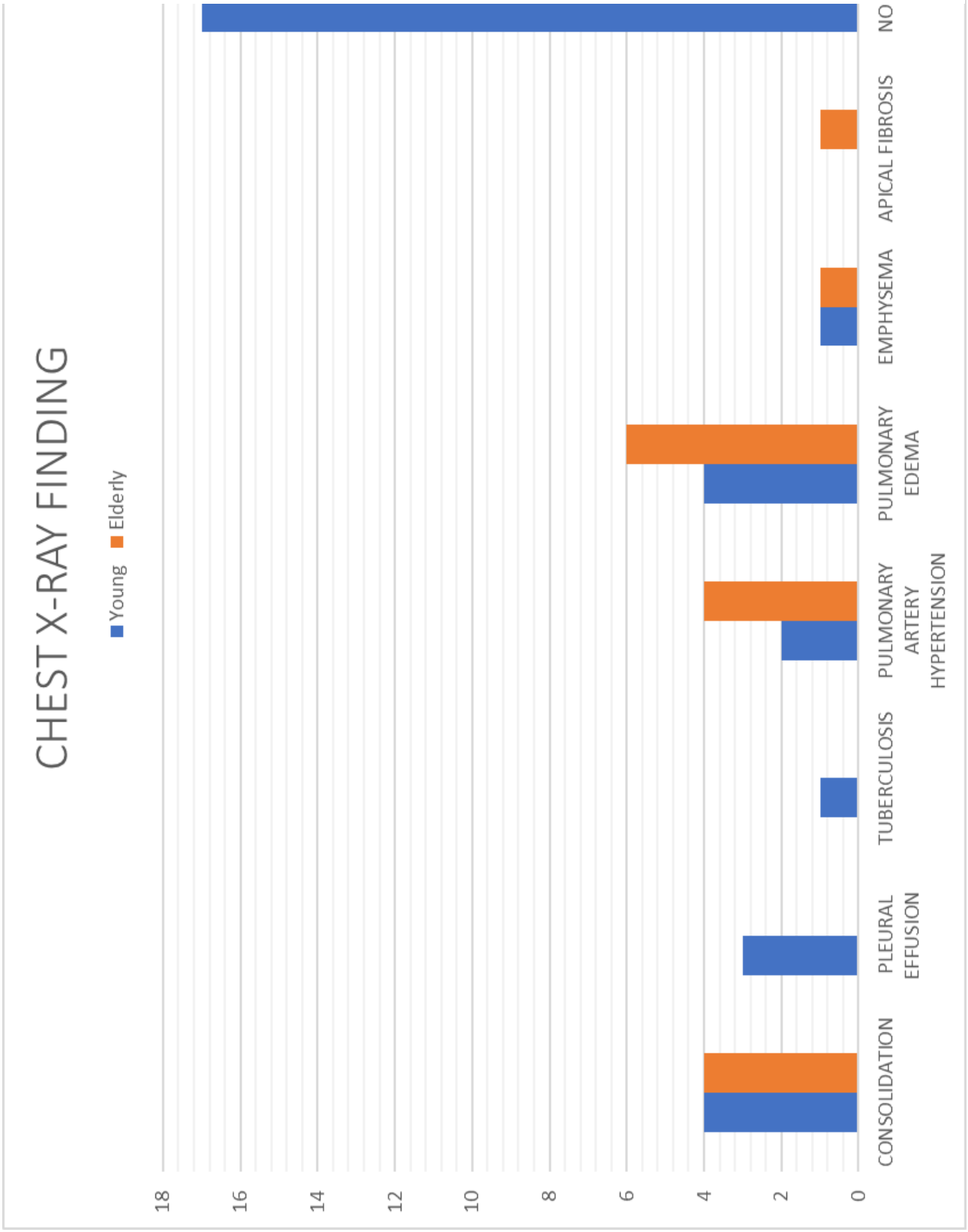


Table 10. DURATION OF HOSPITALIZATION

NUMBER OF DAYS	Group		Total
	A	B	
1	2	6	8
3	5	2	7
4	1	2	3
5	1	2	3
6	0	2	2
7	4	0	4
8	**4**	**5**	**9**
9	1	0	1
10	4	4	8
11	1	2	3
12	0	1	1
13	0	1	1
14	2	2	4
15	1	0	1
16	1	0	1
17	1	0	1
18	1	0	1
20	0	1	1
23	1	0	1
25	0	1	1
26	0	1	1
38	1	0	1
55	1	0	1

Among 64, 9 patients including 4 young and 5 elderly had 8 day stay in the hospital while the longest hospitalization of 55 days was documented by a patient with Rhinocerebral Mucormycosis with Acute Thalamocapsular Infarct.

Table 11 shows a total of 13 patients from 64 included in the study had developed complications secondary to the invasive ventilation. Ventilator associated pneumonia(VAP) (9 i.e., 14.06%) was the most common complication. This was followed by pleural effusion and pulmonary edema which was seen only in the young(Graph 5).

Table 11. Complications secondary to Mechanical Ventilation

COMPLICATION		**GROUP**		**TOTAL**
		A	**B**	
VENTILATOR ASSOCIATED PNEUMONIA (VAP)	*Count*	3	6	9
	%	9.37%	18.75%	14.06%
PLEURAL EFFUSION	*Count*	2	0	2
	%	6.25%	0.00%	3.12%
PULMONARY EDEMA	*Count*	2	0	2
	%	6.25%	0.00%	3.12%
NONE	*Count*	25	26	51
	%	78.12%	81.25%	79.68%

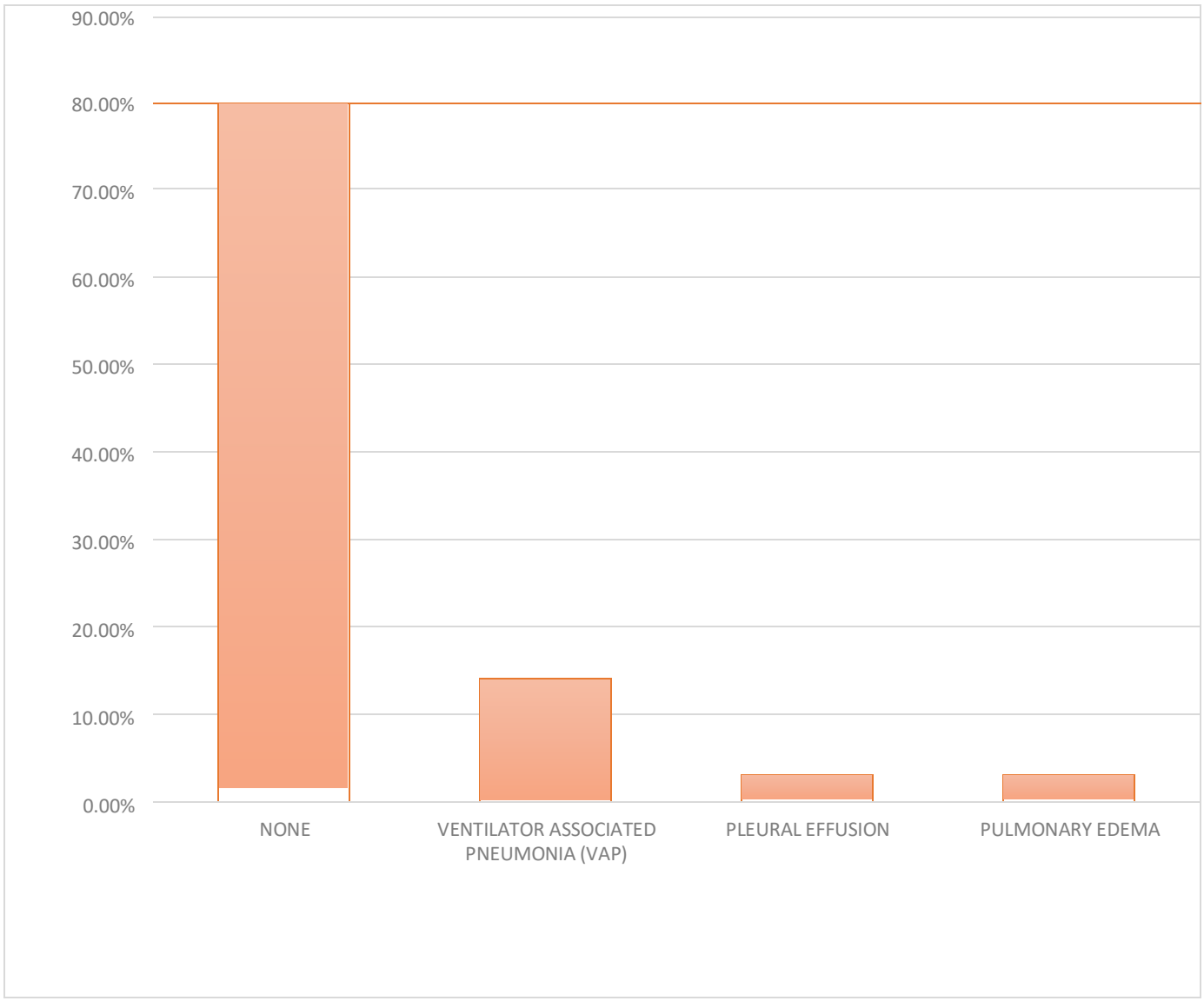
Though VAP was more common in elderly there was no significant relation between the age group and the complications encountered (Table 12).

Table 12. Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	5.020[a]	3	0.170
Likelihood Ratio	6.584	3	0.086
N of Valid Cases	64		

a. 6 cells (75.0%) have expected count less than 5. The minimum expected count is 1.00.

Fig.17 shows that about 80% of the critically ill mechanically ventilated had no complications.



Graph 5. Bar Chart showing Ventilator associated complications

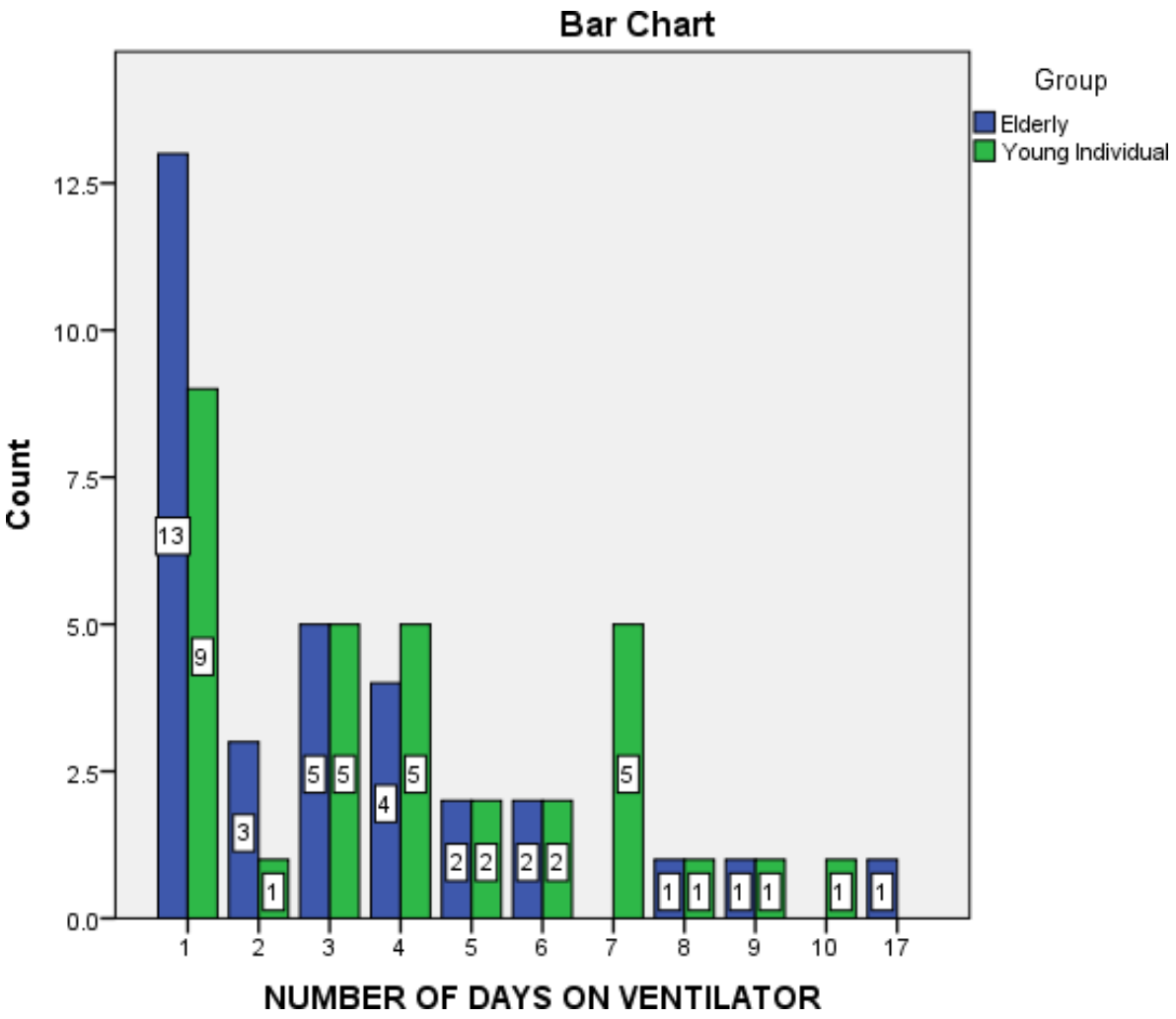
NUMBER OF DAYS ON VENTILATOR

Most patients(i.e.,22) were on ventilator for one day(Table 13). Maximum days a patient had been on ventilator was 17 days by an elderly who had Ischemic Stroke.

Table 13. Total Days of Mechanical Ventilation in hospitalized patients

DAYS	**GROUP**		**TOTAL**	**Chi square value**	**P value**
	A	**B**			
1	**9**	**13**	**22**		
2	1	3	4		
3	5	5	10		
4	5	4	9		
5	2	2	4		
6	2	2	4	8.38	0.548
7	5	0	5		
8	1	1	2		
9	1	1	2		
10	1	0	1		
17	0	1	1		

No relation has been established in our study between age and days on ventilator.



Graph 6. Bar chart comparing total days of mechanical ventilation of the two groups

Of most people who were on ventilator for a day, 13 were elderly while 9 were young. Most days any young patient spent on ventilator was 10 days by a patient diagnosed with Organophosphorus Compound Poisoning (Graph 6).

Table 14. Electrocardiographic findings(ECG) in the two groups

ECG FINDING	GROUP		TOTAL
	A	B	
VENTRICULAR ECTOPIC BEATS	0	1	1
LEFT BUNDLE BRANCH BLOCK, TACHYCARDIA	0	1	1
LEAD 2,3,aVF T WAVE INVERSION	1	0	1
LEAD 2,3,aVF T WAVE INVERSION, ST DEPRESSION V5,V6	1	0	1
P-PULMONALE, V1-V4 T WAVE INVERSION V5-V6 ST DEPRESSION, TACHYCARDIA	0	1	1
P-PULMONALE, V1-V6 T WAVE INVERSION	0	1	1
P-PULMONALE	0	1	1
POOR R WAVE PROGRESSION	0	2	2
POOR R WAVE PROGRESSION, V4-V6 T WAVE INVERSIONS,TACHYCARDIA	0	1	1
LEFT VENTRICULAR HYPERTROPHY	0	1	1
RIGHT BUNDLE BRANCH BLOCK	1	0	1
RIGHT BUNDLE BRANCH BLOCK, TACHYCARDIA	0	1	1
RIGHT VENTRICULAR STRAIN PATTERN	0	1	1
LEAD 2,3,aVF T WAVE INVERSION	1	0	1
P-PULMONALE, V1-V4 T WAVE INVERSION V5-V6 ST DEPRESSION, TACHYCARDIA	0	1	1
P-PULMONALE	0	1	1
POOR R WAVE PROGRESSION, V4-V6 T WAVE INVERSIONS,TACHYCARDIA	0	1	1
RIGHT BUNDLE BRANCH BLOCK	1	0	1
RIGHT VENTRICULAR STRAIN PATTERN	0	1	1
SINUS TACHYCARDIA	10	4	14
ST DEPRESSION LEAD 2,3, aVF WITH T WAVE INVERSION WITH RECIPROCAL CHANGES IN CHEST LEADS	0	1	1

ECG FINDING	GROUP		TOTAL
	A	B	
ST ELEVATION IN V1,V2,V3 AND RIGHT BUNDLE BRANCH BLOCK	0	1	1
ST ELEVATION V1-V3 WITH T WAVE INVERSION,P-PULMONALE	1	0	1
ST ELEVATION V2-V4 WITH T WAVE INVERSION IN V2-V5	1	0	1
T WAVE INVERSION IN LEAD 2,3,AVF,V3-V6	0	1	1
TACHYCARDIA, V2-V6 T WAVE INVERSION	1	0	1
ST DEPRESSION V2-V6, LEAD 2,3,aVF WITH T WAVE INVERSION	1	0	1
ST DEPRESSION V4-V6	0	1	1
V1-V2 T WAVE INVERSION	1	0	1
ST DEPRESSION V1-V6 T WAVE INVERSION	0	1	1
ST DEPRESSION V1-V6, T WAVE INVERSION LEAD 2,3,aVF	0	1	1
NORMAL	14	11	25
Total	32	32	64

Table 15. Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	27.931[a]	25	0.311
Likelihood Ratio	37.675	25	0.050
N of Valid Cases	64		

a. 48 cells (92.3%) have expected count less than 5. The minimum expected count is .50.

39 patients had normal ECG among them 14 had sinus tachycardia(Table 14). There was no relation in the ECG findings and the age group(Table 15).

SOFA SCORE

All the patients admitted to the critical care ward and subsequently mechanically ventilated were evaluated for the SOFA score at admission(Table 16). 14 patients presented with a SOFA score of 8, of these 8 were young and 6 were elderly. Out of these 8 young, 5 died and 8 recovered while among elderly only 1 died and other 5 recovered. Among the 64 patients in our study, 2 elderly had the highest SOFA score of 13 on admission. Of these 2, one elderly was 78 year old male diagnosed with Septic Shock secondary to lower respiratory tract infection with Ischemic Heart Disease with an ejection fraction of 40-45%. He was found to have high serum phosphorous levels of 6.3mg/dL. During his period on mechanical ventilator he developed ventilator associated pneumonia and subsequently died. The other patient was also a case of septic shock and later died.

There was no significant association between the SOFA score on admission and the outcome in our study.(Table 17)

Table 16. SOFA Score on admission

SOFA SCORE		**Group**		**Total**
		A	**B**	
2	Count	0	1	1
	%	0.00%	3.12%	1.56%
3	Count	4	2	6
	%	12.50%	6.25%	9.37%
4	Count	2	2	4
	%	6.25%	6.25%	6.25%
5	Count	4	6	10
	%	12.50%	18.75%	15.62%
6	Count	6	3	9
	%	18.75%	9.37%	14.06%
7	Count	4	5	9
	%	12.50%	15.62%	14.06%
8	Count	**8**	**6**	**14**

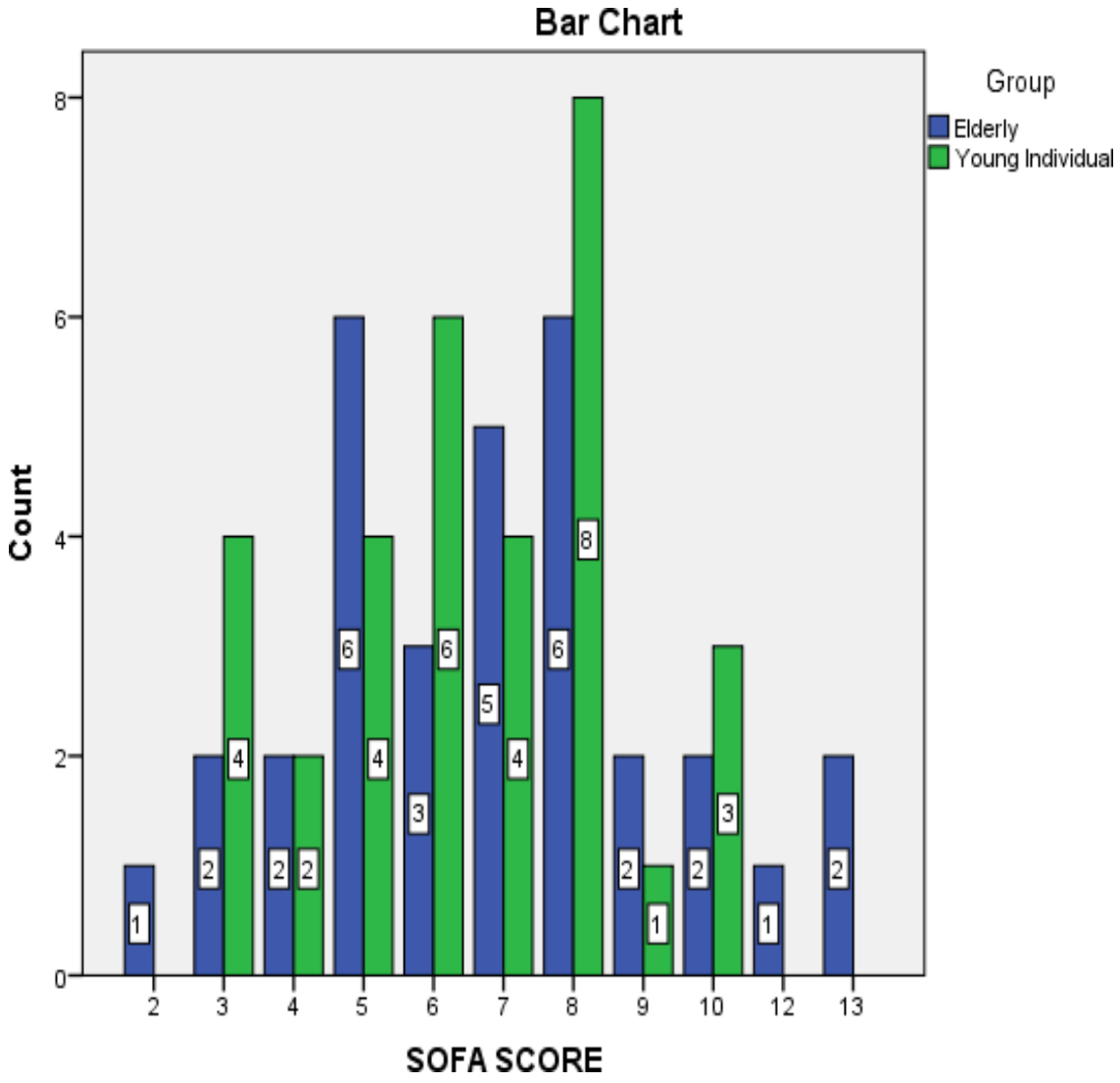
	%	25.00%	18.75%	21.87%
9	Count	1	2	3
	%	3.12%	6.25%	4.68%
10	Count	3	2	5
	%	9.37%	6.25%	7.81%
12	Count	0	1	1
	%	0.00%	3.12%	1.56%
13	Count	0	2	2
	%	0.00%	6.25%	3.12%

None of the patients had a SOFA score of 1, 11 or more than or equal to 14.

Table 17. Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.997[a]	10	0.726
Likelihood Ratio	8.586	10	0.572
N of Valid Cases	64		

a. 18 cells (81.8%) have expected count less than 5. The minimum expected count is .50.



Graph 7. Bar chart comparing SOFA Score at admission of the two groups

OUTCOME

18 of the 32 young included in our study recovered and 14 died while in the group B, 17 recovered and 15 died (Table18).

Table 18. Outcome of the study

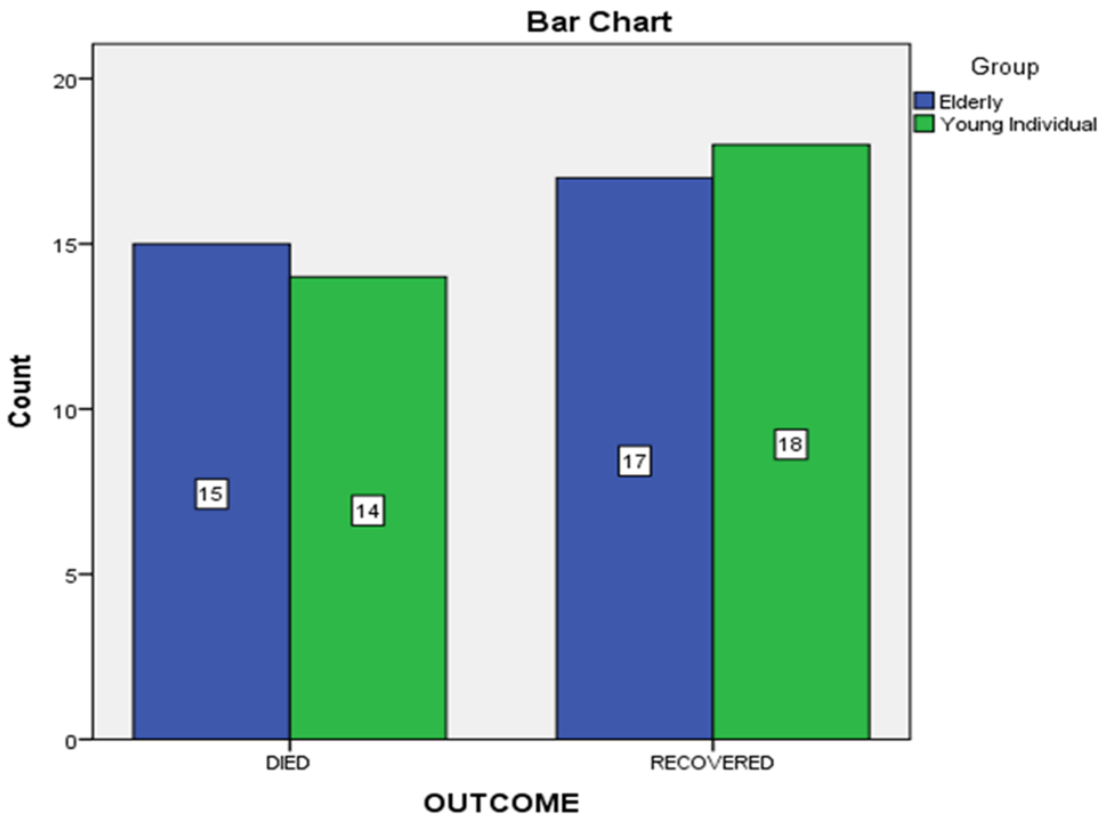
OUTCOME		Group		Total N= 64	Chi-square value	P-value
		Young (GROUP A) n=32	Elderly (GROUP B) n=32			
RECOVERED	Count	18	17	35	0.063	0.802
	%	56.25%	53.12%	54.68%		
DIED	Count	14	15	29		
	%	43.75%	46.87%	45.31%		
TOTAL	Count	32	32	64		
	%	100.00%	100.00%	100.00%		

GROUP A

22 patients of the young group had normal serum phosphorous levels on admission of which 15 recovered and 7 died. Among those with high blood phosphorous levels, 3 survived and 1 died while all 6 patients who were admitted with low serum phosphorous levels died.

Table 19. Serum phosphorous and outcome comparison in Group A

SERUM PHOSPHORUS	NUMBER OF PATIENTS (n=32)	RECOVERD	DIED
<2.5	6	0	6
2.5- 4.5	22	15	7
>4.5	4	3	1



Graph 8. Bar Chart depicting the outcome of the two groups

GROUP B

Among elderly, 17 admitted had normal blood phosphorous, of those 12 recovered and 5 died. Of patients admitted with hyperphosphatemia on admission, 7 died and 5 recovered while all 3 admitted with hypophosphatemia i.e., <2.5mg/dL died and none recovered.

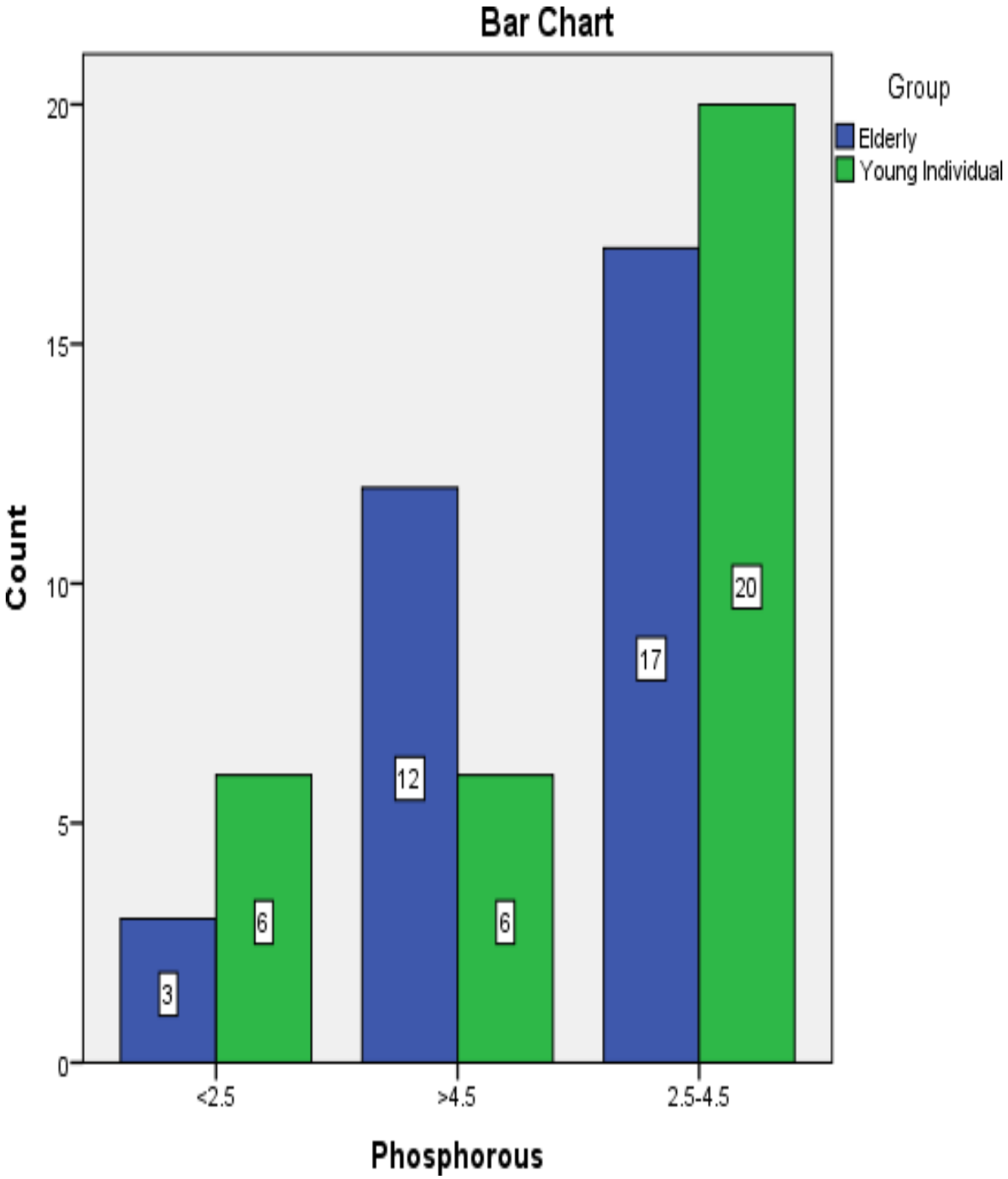
Table 20. Serum phosphorous and outcome comparison in Group B

SERUM PHOSPHORUS	NUMBER OF PATIENTS (n=32)	RECOVERED	DIED
<2.5	3	0	3
2.5- 4.5	17	12	5
>4.5	12	5	7

The serum phosphorous levels were categorized as normal, hypophosphatemia and hyperphosphatemia. The levels were compared to the two groups(Graph 9) and no association was found between the age groups and admission serum phosphorous.

Table 21. Serum Phosphorous and Age groups Comparison

Serum Phosphorous (mg/dL)		GROUP		Total	Chi-square value	p-value
		Young	Elderly			
<2.5	Count	6	3	9	3.243	0.198
	% within Group	18.80%	9.40%	14.10%		
2.5-4.5	Count	20	17	37		
	% within Group	62.50%	53.10%	57.80%		
>4.5	Count	6	12	18		
	% within Group	18.80%	37.50%	28.10%		

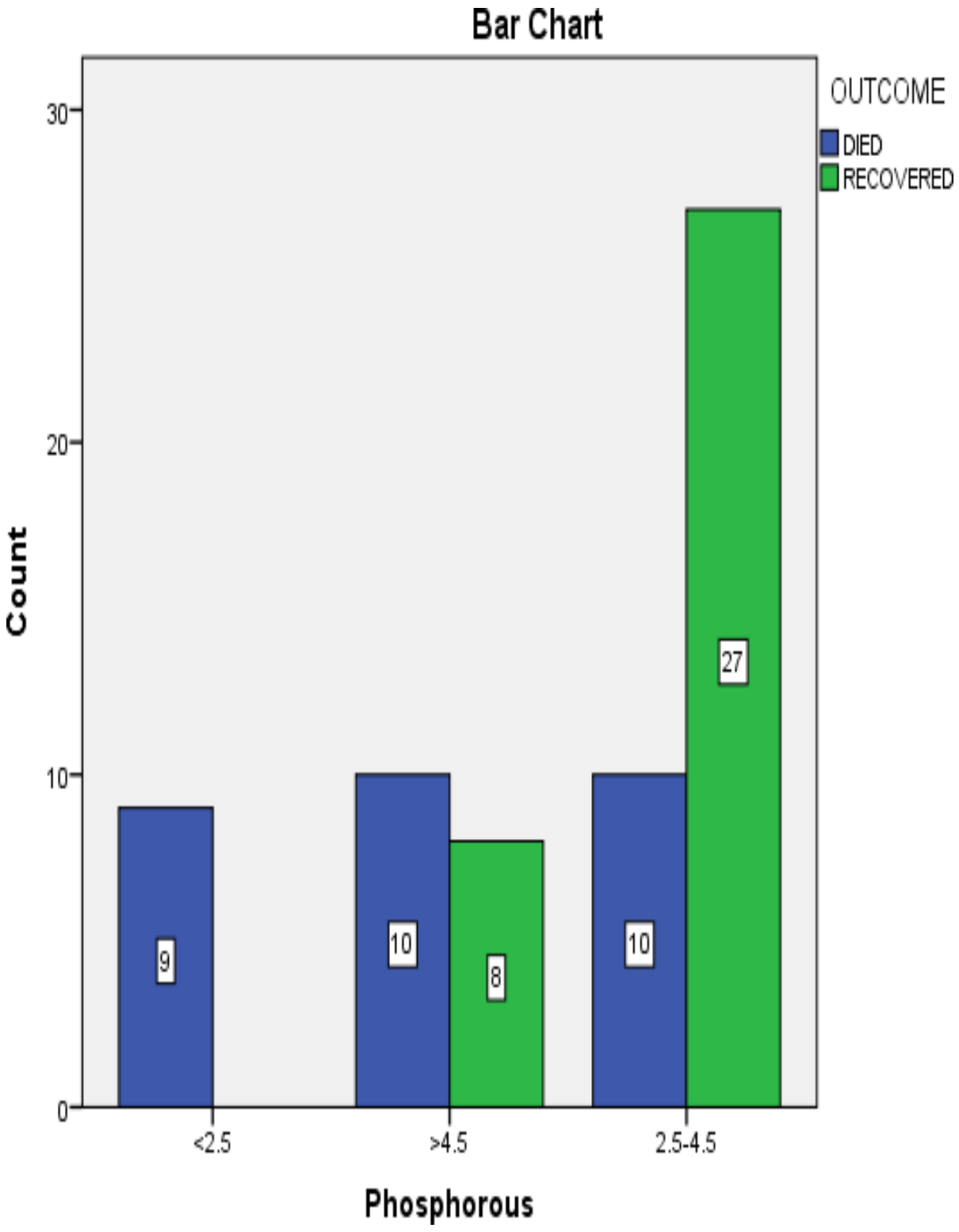


Graph 9. Bar chart comparing Serum Phosphorous levels with age groups

Blood phosphorous levels at admission were also compared with the outcome of the patients and it was noticed in our study(Graph 10) that there was a significant relation (p-value=0.000) between the two(Table 22).

Table 22 Association of Serum Phosphorous levels with outcome of the patient

Serum Phosphorous		OUTCOME		Total	Chi-square value	p-value
		RECOVERED	DIED			
<2.5	Count	0	9	9	16.617	0.000
	% within outcome	0.00%	31.00%	14.10%		
>4.5	Count	8	10	18		
	% within outcome	22.90%	34.50%	28.10%		
2.5-4.5	Count	27	10	37		
	% within outcome	77.10%	34.50%	57.80%		



Graph 10. Bar chart comparing Serum Phosphorous levels and outcome of the patient

DISCUSSION

Our study's objective was to assess the severity and prognostic value of the admission serum phosphorus level in mechanically ventilated patients among young adults (<60 years) and elderly (60 years or older).

Critically ill patients who develop acute respiratory failure are managed by supportive therapy like mechanical ventilation, but prolonged mechanical ventilation is itself associated with many serious complications like tracheal stenosis, barotraumas, pneumothorax and sepsis.[50] Hence, the key to survival in such cases is early weaning from the ventilatory support.[51]

Removal of the endotracheal tube and withdrawal of ventilator support are both steps in the process of weaning from invasive ventilation. It can only be started once the underlying issue or respiratory failure has been addressed.[52]

Boles JM et al[53] has mentioned in his study that one of the crucial cause of respiratory failure is hypophosphatemia and other metabolic derangements. According to *Dooley et al* [54] ,the inability to wean from a mechanical ventilator may be caused by insufficient 2,3 diphosphoglycerate synthesis, which shifts the haemoglobin dissociation curve to the left and reduces oxygen supply to the tissues. Also the phosphorylated intermediates like ATP are not produced adequately in hypophosphatemic states which hampers diaphragmatic contractility.

Gravelyn and colleagues[55] studied 23 patients and concluded that respiratory muscle weakness is common among the patients with hypophosphatemia which can be improved with phosphate repletion.

Another study by *Alsumrain MH et al* [56]in 2010 concluded that adequate serum phosphorus levels are associated with successful weaning from ventilation.

Furthermore, a number of studies[57-63] have demonstrated that, in the absence of any underlying lung pathology, a correlation exists between respiratory muscle weakness and low serum phosphorus levels.

Hypophosphatemia is difficult to diagnose as it presents with nonspecific symptoms. Therefore it is crucial to get the baseline serum phosphorous levels for all patients admitted in a critical care unit.

Our study had recruited all patients admitted to the ICU who subsequently required mechanical ventilation. On admission serum phosphorous levels and SOFA scoring was done to evaluate the condition of the patient and assess the need for assisted ventilation. Also the patients were grouped according to their age group into young adults and elderly. Our study was the first to compare serum phosphorous levels with the age group. No significant association of blood phosphorous and age was seen in our study.

The study predominantly had males which was also seen in the study conducted in the year 2017 by *Talakoub et al.*[2]Majority of patients in our study belonged to the age group 60-69 yrs. The oldest patient was a 90 yr old male. Most of the young adults were found to have diabetes mellitus whereas commonest comorbidity in elderly was hypertension. Our results were similar to *Alsumrain MH et al*[56].

Most of the young were admitted with Poisoning while Cerebro-Vascular Accident was the common diagnosis on admission among the elderly. 50% of the mechanically ventilated patients had no underlying lung pathology. Most of the patients were hospitalized for 8 days while the maximum hospitalization was for 55 days. 22 patients were ventilated for only one day while an elderly had spent 17 days on mechanical ventilator. Most common complication was Ventilator Associated Pneumonia seen in 14.06% patients similar to *Driks M et al*. 21.87% patients had a SOFA sore of 8 on admission. In our study no significant association has been established between SOFA score and the outcome. 9 patients were found to have low phosphorous. All 9 hypophosphatemic patients (6 young and 3 elderly) in the study could not be weaned of the mechanical ventilator and later died. 16 patients(4young and 12 elderly) had hyperphosphatemia on admission of whom 8 survived and other 8 died. Remaining 39 patients had normal phosphorous levels and of which 27 recovered and 12 died due to critical illness.

Table 23. Descriptive Data

	Group	Mean	SD	Mann-Whitney U Value	p-value
AGE	Young	38.68	10.28	1024	< 0.001
	Elderly	70	8.31		
DAYS OF HOSPITALIZATION	Young	11.125	10.874	448	0.392
	Elderly	8.37	6.47		
NUMBER OF DAYS ON VENTILATOR	Young	4.063	2.675	399.5	0.123
	Elderly	3.313	3.306		
S. PHOSPHORUS	Young	3.656	2.116	648.5	0.068
	Elderly	4.528	2.116		

The only restrictions in our study was that the GCS is used by SOFA for neurological evaluation, which is exceedingly challenging or nearly impossible in patients who are under the influence of sedative drugs, it increases the risk of data collecting errors.

CONCLUSION

Every hour is a golden moment in critical care. Patients admitted in critical care unit are at risk of respiratory failure or sudden cardiac death.

The assessment of Serum Phosphorous levels at the time of admission in critical care setup helps us to identify group of the patient who are at risk for respiratory failure and may require artificial ventilation. Both hypophosphatemia and hyperphosphatemia are considered risk factors for the development of respiratory failure.

Though our study did not recognize the levels of serum phosphorous and need for ventilator support on admission with statistical significance, we recommend serial measurements of serum phosphorous levels in critically ill patients for accurate prediction of ventilator support need.

Serum Phosphorous levels across all the age groups does not remain same both in health and diseased state hence can be a good predictor for respiratory failure. The care for people on ventilator needs to be multidisciplinary and should aim to prevent the complications.

Elderly patients need to be treated aggressively to prevent mortality. These are the set of patients who have multidisciplinary comorbidities and are on polypharmacy which also poses challenges in the management.

SUMMARY

From December 2020 to June 2022, 64 patients with critical illness requiring mechanical ventilation were studied at BLDE (DU) Shri B. M. Patil Medical College and Research Centre in a hospital based cross-sectional study.

All the patients were examined in detail at the time of admission and SOFA score was applied to predict the outcome.

The admission serum level of all patients was estimated and those requiring mechanical ventilation were included in the study.

Patients were grouped into Group A and Group B according to their ages.

Significant association between serum phosphorous at admission and outcome was established as all the patient who had presented with low blood phosphorous needed mechanical ventilation and subsequently died.

No relation between serum phosphorous levels and age group was found during the study.

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ANNEXURE I (ETHICAL CLEARANCE CERTIFICATE)

IEC/NO-9/2021
Date- 22/01/2021

B.L.D.E. (DEEMED TO BE UNIVERSITY)
(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)
The Constituent College
SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

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

Title: Comparison of serum phosphorus level as severity and prognostic marker in patients on mechanical ventilation among adults and older people

Name of PG student: Dr Priyanka Tomar, Department of Medicine

Name of Guide/Co-investigator: Dr Anand.P.Ambali, Professor of Medicine

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

Following documents were placed before Ethical Committee for Scrutinization:

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

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ANNEXURE –II

INFORMED CONSENT FORM

BLDEDU'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA- 586103

TITLE OF THE PROJECT - "COMPARISON OF SERUM PHOSPHORUS LEVEL AS SEVERITY & PROGNOSTIC MARKER IN PATIENTS ON MECHANICAL VENTILATION AMONG ADULTS AND ELDERLY(>60 YRS)"

PRINCIPAL INVESTIGATOR - DR. PRIYANKA TOMAR

P.G.GUIDE NAME - DR. ANAND P. AMBALI
PROFESSOR OF MEDICINE

CO-GUIDE - DR. SHIVANAND L.K.
ASSOCIATE PROFESSOR OF ANAESTHESIA

CHAIRMAN ETHICAL COMMITTEE

All aspects of this consent form are explained to the patient in the language understood by him/her.

1) PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

2) PROCEDURE:

I am aware that in addition to routine care received, I will be asked a series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

3) RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition, and the procedure of this study isnot expected to exaggerate these feelings that are associated with the usual course of treatment.

4) BENEFITS:

I understand that my participation in this study will help patient's survival and a better outcome.

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to confidentiality and privacy regulations. Information of a sensitive personal nature will not be a part of the medical records but will be stored in the investigator's research file and identified only by a code number. The code-key connecting name to numbers will be kept in a separate location.

If the data are used for publication in the medical literature or for teaching purposes, no name will be used, and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr. Priyanka Tomar is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

7) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or

future care at this hospital. I also understand that Dr. Priyanka Tomar may terminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist if this is appropriate.

8) INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to_____the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

DR PRIYANKA TOMAR
(Investigator)

Date :

II) STUDY SUBJECT CONSENT STATEMENT:

I confirm that DR PRIYANKA TOMAR has explained to me the purpose of the research, the study procedures that I will undergo, the possible risks and discomforts, and benefits that I may experience in my own language. I have read, and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

_____

_____

Participant / Guardian Date:

_____

_____

Witness to signature Date:

ANNEXURE III- PROFORMA

BLDEDU'S S.H.R.I. B.M.PATIL MEDICAL COLLEGE

HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA

SCHEME OF CASE TAKING

Informant :

Name: CASE NO:

Age: IP NO:

Sex:

D.O.A.: D.O.D.:

Past Occupation:

Present Occupation:

Residence:

Chief complaints:

Chief Complaints

History of presenting illness

Past Medical History- Diabetes Mellitus /HTN /TB /Bronchial Asthma /Epilepsy /Heart Disease /Malnutrition /Hyperparathyroidism /Hypoparathyroidism /Leukemia /Lymphoma /Chronic Renal Failure /Neuromuscular Syndrome

Drug & other treatment History

Personal History- Diet /Appetite / Sleep / Bowel /Bladder

Alcohol / Smoking / Tobacco /Others addictions

Family History

GENERAL PHYSICAL EXAMINATION

Appearance-Well /Unwell /Severely Ill

Nutrition & Built

Cyanosis /Jaundice /Anemia /Skin Lesions /Clubbing

Vital Signs- Pulse Rate /Blood Pressure /Temperature /Respiratory Rate

Head to toe examination: Mouth & Pharynx- Lips /Tongue /Teeth /Gums /Buccal Mucosa

Hands- Clubbing /Tremors /Koilonychia /Leuconychia /Deformity

Neck - Thyroid /Pulsations /Lymphadenopathy

Upper & Lower Limbs- Edema /Pigmentation /Lymphadenopathy

SYSTEMIC EXAMINATION.

C.N.S		C.V.S	Respiratory	Abdomen
Higher Functions Cranial Nerves	INSPECTION	Anemia/Cyanosis/clubbing/Arterial pulse/edema/cold extremities J.V.P	Appearance/ Movement	Shape Umbilicus Movement Surface
Motor System Coordination	PALPATION	Apical Impulse/Other pulsations	Lymph Nodes/Swelling/Tenderness/Trachea/Heart/Chest expansion	Organomegaly
Reflexes	PERCUSSION		Resonance /Dullness	Note
Sensory System	AUSCULTATION	S1/S2/S3/S4	Breath sounds-	Bowel Sounds

VENTILATOR NOTES
INTUBATION, Date-
EXTUBATION, Date-
Total duration of MV-
OUTCOME- Died /Recovered
Complications of ventilation-

INVESTIGATIONS

Serum Phosphorus Levels	
Complete Blood Count	Hb- TLC- Plt Count- MCV /MCH /MCHC /MPV N /L /E /M /B
Serum Electrolytes	Na^+ /K^+ /Ca^{2+}
Liver function tests	S. Blb-(T)- (D)- (ID) SGPT- SGOT- ALP- Alb-
Sugar levels	RBS- / HbA1c -
Urine	Alb /Sugar /micro
E.C.G.	
Chest X-ray PA View (Findings)	
Echocardiography	

SOFA SCORE (FOR THE ASSESSMENT OF SEVERITY & PROGNOSIS)

VARIABLES		SCORE
RESPIRATION PaO2/FiO2(mmHg)		
COAGULATION (Platelets)		
LIVER, Bilirubin (mg/dL)		
CARDIOVASCULAR		
NERVOUS SYSTEM G.C.S. Score		
RENAL Creatinine (mg/dL) Urine Output		

CONCLUSION:

Date:-

Signature:-

KEY TO MASTERCHART

S. Phosphorous	Serum Phosphorous
VAP	Ventilator Associated Pneumonia
IHD	Ischemic Heart Disease
HTN	Hypertension
DM	Diabetes Mellitus
NR	Not Recordable
MCA	Middle Cerebral Artery
EF	Ejection Fraction
PAH	Pulmonary Artery Hypertension
CVA	Cerebrovascular Accident
TB	Tuberculosis

MASTER CHART

S. No.	NAME	AGE	SEX	COMORBIDITIES	DIAGNOSIS	DAYS OF HOSPITAL STAY	NUMBER OF DAYS ON VENTILATOR	COMPLICATIONS	PULSE RATE (bpm)	BLOOD PRESSURE (mm of Hg)	RESPIRATORY RATE (cpm)	CHEST XRAY	S. PHOSPHORUS(mg/dL)	SOFA SCORE	OUTCOME
1	ANJALI KAMBALE	19	F	DIABETES MELLITUS	LEFT EYE MUCORMYCOSIS SECONDARY TO COVID 19 INFECTION WITH TYPE 2 DIABETES MELLITUS	3	3	NONE	110	90/60	16	LEFT LUNG PATCHY CONSOLIDATION	1.2	8	DIED
2	JAKKAPPA DODDINI	22	M	NONE	ORGANOPHOSPHORUS COMPOUND POISONING	7	3	NONE	72	110/60	18	NORMAL	2.7	7	RECOVERED
3	PRIYANKA RANGU CHAVAN	24	F	NONE	LEFT HYPOCHONDRIAC STAB INJURY	10	1	NONE	112	130/70	14	NORMAL	3.8	3	RECOVERED
4	BHARATI B TELI	24	F	NONE	PYRETHROID POISONING TO INFLICT SELF HARM	4	4	VAP	108	130/90	18	RIGHT LUNG PATCHY CONSOLIDATION	4.7	5	DIED
5	MAHESH PUJARI	26	M	NONE	SNAPPING SCAPULAR SYNDROME WITH SCAPULOTHORACIC BURSITIS	10	1	NONE	84	120/70	17	PULMONARY ARTERY HTN,PULMONARY EDEMA	4.2	6	RECOVERED
6	MANJUNATH KOLI	29	M	EPILEPSY	BREAKTHROUGH SEIZURES	10	6	NONE	92	120/80	18	NORMAL	3	4	RECOVERED
7	SWALIYA SAJIN KOTAMBRI	30	F	RHEUMATOID ARTHRITIS	TUBERCULAR MENINGITIS (RIFAMPICIN RESISTANCE)	8	7	NONE	100	110/60	22	NORMAL	1.5	6	DIED
8	MUTTU SIDDAPPA YARANAL	30	M	NONE	POISONING UNDER ALCOHOL INFLUENCE	1	1	NONE	130	90/60	43	NORMAL	2.9	10	DIED
9	SANTOSH SOUDAGAR	33	M	DIABETES MELLITUS	RHINOCEREBRAL MUCORMYCOSIS WITH ACUTE THALAMOCAPSULAR INFARCT WITH TYPE 2 DM	55	6	NONE	82	110/80	18	NORMAL	3.5	6	RECOVERED
10	SHRIDEVI DYAMGOL	35	F	NONE	REFRACTORY STATUS EPILEPTICUS	7	7	NONE	151	110/50	40	NORMAL	0.7	8	DIED
11	ANIL NATU RATHOD	35	M	NONE	STATUS EPILEPTICUS	1	1	NONE	108	110/70	20	NORMAL	4.3	8	DIED
12	PRASHANT DALAWAI	35	M	NONE	ORGANOPHOSPHORUS COMPOUND POISONING	14	10	NONE	136	110/70	38	NORMAL	2.8	8	RECOVERED
13	SANTOSH MANUR	35	M	NONE	EMAMECTIN BENZOATE POISOINING	8	5	NONE	80	120/80	19	NORMAL	8.4	7	RECOVERED
14	SHANTESH BIRADAR	36	M	NONE	AMITRAZ COMPOUND CONSUMPTION	7	2	NONE	90	110/70	16	NORMAL	3.3	6	RECOVERED
15	VEERESH BHIMANA GOUDA KULAGERI	37	M	EPILEPSY	STATUS EPILEPTICUS	8	1	VAP	120	128/60	30	RIGHT UPPER LOBE CONSOLIDATION	3.5	8	RECOVERED

16	MABOOBI SHIVAPUR	37	F	DEPRESSION	ORGANOPHOSPHORUS COMPOUND POISONING	5	5	NONE	74	170/90	22	LEFT LOWER LOBE CONSOLIDATION	4.1	4	DIED
17	MANAPPA SHARANAPPA NAIKODI	38	M	NONE	NEUROTOXIC SNAKE BITE	14	7	NONE	60	130/90	28	NORMAL	6.1	8	RECOVERED
18	IRANNA ANGADI	38	M	NONE	SEPSIS SECONDARY TO CELLULITIS	3	1	NONE	130	70/60	20	NORMAL	1	10	DIED
19	SHANKAR KOGANUR	40	M	NONE	CARCINOMA RIGHT BUCCAL MUCOSA WITH SQUAMOUS CELL CARCINOMA	23	1	NONE	80	110/70	25	NORMAL	4.1	3	RECOVERED
20	SAIDABBI BILAGI	40	F	RHEUMATOID ARTHRITIS	AZATHIOPRINE INDUCED PANCYTOPENIA	3	1	PULMONARY EDEMA	70	90/70	30	PULMONARY EDEMA	6.7	5	DIED
21	BASAVARAJ KHALAWADI	40	M	NONE	LEFT MCA INFARCT	9	4	NONE	100	120/80	17	NORMAL	2.5	5	RECOVERED
22	RAMAKRISHNA KAREPPA BIRADAR	45	M	NONE	SEIZURES SECONDARY TO B/L TEMPORO OCCIPITAL INFARCT	7	7	NONE	140	110/70	22	PAN-EMPHYSEMA	4.1	8	DIED
23	ANAND CHANDRASHEKHAR	45	M	BRONCHIAL ASTHMA	ALCOHOLIC LIVER DISEASE	11	4	NONE	92	122/80	15	PLEURAL EFFUSION	4.5	5	RECOVERED
24	ASHOK BASAPPA BABALESHWAR	48	M	EPILEPSY	BREAKTHROUGH SEIZURES,STATUS EPILEPTICUS	10	3	PULMONARY EDEMA	146	130/90	26	PULMONARY EDEMA	3.9	7	RECOVERED
25	SOMASHEKHAR KANABUR	48	M	NONE	TUBERCULAR MENINGITIS	15	4	NONE	80	100/70	34	MILIARY TB	3.7	7	RECOVERED
26	MAMATAJ BEGAM NAZEERAHEMAD KHAN	50	F	HTN,DIABETES MELLITUS	ISCHEMIC STROKE	38	7	LEFT PLEURAL EFFUSION	88	170/90	24	LEFT PLEURAL EFFUSION	3.9	3	RECOVERED
27	RAMZANSAB DASTGIRSAB SAINIK	50	M	DIABETES MELLITUS	CENTRAL NEUROCYSTOMA	17	3	VAP	114	140/80	18	LEFT LOWER LOBE CONSOLIDATION	2.1	3	DIED
28	HANAMANTH NAYAK	52	M	DEPRESSION	AMITRAZ INSECTICIDE CONSUMPTION	16	4	NONE	68	80/60	32	PULMONARY ARTERY HTN	6.9	10	RECOVERED
29	HANAMANTH SHAHAPUR	52	M	NONE	AUTOIMMUNE ENCEPHALITIS	18	9	NONE	103	140/90	36	NORMAL	3	6	RECOVERED
	VISHAWANATGIRIMALLAPPA MADIKESHWAR	53	M	NONE	TYPE 1 RESPIRATORY FAILURE WITH MULTIPLE ACUTE PONTINE AND CEREBELLAR INFARCTS WITH IHD AND ALCOHOL WITHDRAWAL	8	8	NONE	130	150/90	48	PULMONARY EDEMA	4.9	9	DIED
31	SHANKERGOUDA POLICEPATIL	56	M	DIABETES MELLITUS,HTN,IHD	DECOMPENSATED ALCOHOLIC LIVER DISEASE WITH TYPE 2 DIABETES, HTN & OLD IHD	3	3	LEFT PLEURAL EFFUSION	90	110/80	28	LEFT PLEURAL EFFUSION	2.7	8	DIED
32	PRAHLAD YALLAPPA JOGI	56	M	HTN	RIGHT TEMPORAL LOBE HAEMORRHAGE WITH HTN	3	1	NONE	146	250/120	28	NORMAL	2.3	6	DIED
33	GANAPATHU SANTAPPA PRASAD	60	M	NONE	SEPTIC SHOCK WITH DYSELECTROLYTEMIA SECONDARY TO ACUTE GASTROENTERITIS	14	1	NONE	120	140/80	40	NORMAL	4.2	9	RECOVERED

34	SIDDAGOND MALLAPPA MALLAD	60	M	BRONCHIAL ASTHMA	CARBON DIOXIDE NARCOSIS SECONDARY TO MORBID OBESITY	8	3	NONE	86	130/80	30	NORMAL	4.1	3	DIED
35	KAVERI PUNDALIK HADAPAD	60	F	DIABETES,HTN	CARDIOGENIC SHOCK WITH PULMONARY EDEMA SECONDARY TO IHD WITH DM & HTN	8	1	NONE	150	NR(IONOTROPIC SUPPORT)	30	PULMONARY EDEMA	8.8	8	RECOVERED
36	SIDDAGOWDA BIRADAR	60	M	NONE	EMAMECTIN BENZOATE COMPOUND POISONING	6	1	NONE	160	130/80	18	NORMAL	2.6	8	RECOVERED
37	SHIVNINGAPPA	61	M	NONE	ORGANOPHOSPHORUS COMPOUND POISONING	1	1	NONE	102	180/110	32	NORMAL	3.1	7	DIED
38	TUKARAM MANKAR	62	M	NONE	POSTERIOR CIRCULATION STROKE	1	1	NONE	120	210/110	22	RIGHT UPPER LOBE CONSOLIDATION	2.3	7	DIED
39	VEERBHADRAPPA KORE	64	M	DIABETES,HTN	RIGHT MCA TERRITORY INFARCT WITH TYPE 2 DM AND HTN	1	1	NONE	130	140/90	24	NORMAL	2.1	7	DIED
40	MALLU KUMAR	64	M	DIABETES MELLITUS,HTN	POSTERIOR CIRCULATION STROKE	6	3	NONE	85	160/80	18	NORMAL	5	5	RECOVERED
41	SUDHINDRA	64	M	DIABETES MELLITUS,HTN	DRCOMPENSATED ALCOHOLIC LIVER DISEASE	3	2	NONE	140	70/60	33	NORMAL	4.6	12	DIED
42	CHANDIBAI K RATHOD	65	M	TYPE 2 DIABETES MELLITUS	IHD WITH EF 25-30% WITH APICAL CLOT WITH CARDIOGENIC SHOCK	4	1	NONE	132	120/80	25	PULMONARY EDEMA	3.7	2	DIED
43	LAXMI KARANDE	65	F	IHD,HTN,DM	LEFT FRONTOTEMPOROPARIETAL INFARCT WITH RHD AND PAH	12	3	NONE	70	100/60	16	PULMONARY ARTERY HTN	4	5	RECOVERED
44	ASHOK H PATANGE	65	M	NONE	SEPTIC SHOCK SECONDARY TO RIGHT UL PNEUMONIA	5	1	NONE	120	90/60	22	RUL CONSOLIDATION	2.7	13	DIED
45	RAMU TULAJU RATHOD	65	M	NONE	CVA(RIGHT MCA INFARCT WITH COMPRESSSION ON LATERAL VENTRICLE)	4	2	NONE	90	140/80	18	NORMAL	1.9	7	DIED
46	ANNAKKA MELLIGERI	66	F	HTN	CARBON DIOXIDE NARCOSIS SECONDARY TO SLEEP APNOEA	20	4	VAP	54	130/70	19	PULMONARY ARTERY HTN,LEFT LL CONSOLIDATION	5.2	5	RECOVERED
47	GURUBAI S HOSAMANI	68	F	NONE	ST ELEVATION MYOCARDIAL INFARCTION	8	1	VAP	110	90/60	32	NORMAL	3.5	10	RECOVERED
48	TUKARAM S PATIL	68	M	NONE	ISCHEMIC STROKE-CAPSULOGANGLIONIC INFARCT	25	17	NONE	98	110/80	14	NORMAL	2.6	6	RECOVERED
49	HONNABAI KUSAPPA INGALAGI	68	F	NONE	SEVERE MEGALOBLASTIC ANEMIA WITH SHOCK	5	4	NONE	100	90/60	20	PAN-EMPHYSEMA	5.5	10	DIED
50	SHARANAPPA BIJARAGI	69	M	NONE	ISCHEMICA HEART DISEASE WITH PULMONARY EDEMA	1	1	NONE	130	120/80	30	PULMONARY EDEMA	12.7	4	DIED

51	SATIRAVVA BHIMASHYA WALIKAR	70	F	NONE	POSTERIOR CIRCULATION STROKE	1	1	VAP	80	90/60	18	NORMAL	4.8	8	DIED
52	ASHOK P MAHAINDRAK AR	70	M	HTN	IHD WITH CARDIOGENIC PULMONARY EDEMA WITH CARDIOGENIC SHOCK	8	4	NONE	120	150/90	30	PULMONARY EDEMA	3.2	5	RECOVE RED
53	MAHADEV	70	M	COPD,OLD TB	ACUTE EXACERBATION OF COPD WITH SEVERE PAH WITH IHD AND OLD TB	10	1	VAP	103	140/90	24	RIGHT APICAL FIBROSIS	6.7	6	RECOVE RED
54	HANUMANTH SHIVAPPA BHAJANTRI	72	M	HTN	LEFT CAPSULOGANGLIONI C BLEED WITH HYPERTENSION	11	5	NONE	120	170/110	14	NORMAL	4.4	8	RECOVE RED
55	NINGANAGOU DA	75	M	DIABETES MELLITUS,IHD ,CVA	SEPTIC SHOCK SECONDARY TO LEFT LL PNEUMONIA WITH UTI WITH IHD, TYPE 2 DM AND OLD CVA	14	4	NONE	68	110/60	30	LEFT LOWER LOBE CONSOLIDAT ION	5.2	7	DIED
56	LAKKAPPA LAGAMANNA PUJARI	75	M	NONE	AMITRAZ INSECTICIDE CONSUMPTION	8	2	NONE	96	90/60	24	NORMAL	6.6	8	RECOVE RED
57	SHANKERGOU DA PATIL	75	M	NONE	CVA(LEFT CAPSULOGANGLIONI C HAEMORRHAGE) WITH NEWLY DIAGNOSED HTN	10	8	NONE	84	240/100	24	NORMAL	4.3	9	RECOVE RED
58	BASAVVA B HADAPAD	76	F	IHD,HTN	ANTERIOR WALL MYOCARDIAL INFARCTION WITH CARDIOGENIC SHOCK AND ARRHYTHMIA	1	1	NONE	80	110/70	16	PULMONARY ARTERY HTN	5.7	4	DIED
59	BAPURAYA RAMAGOND	78	M	NONE	SEVERE SEPTIC SHOCK SECONDARY TO LRTI WITH IHD WITH EF 40-45% WITH POST HERPETIC NEURALGIA	3	3	VAP	102	130/80(IONOTR OPIC SUPPORT)	30	B/L LOWER LOBE CONSOLIDAT ION	6.3	13	DIED
60	CHANDRASHE KHAR	80	M	NONE	LEFT FRONTOTEMPOROPA RIETAL INFARCT	26	9	VAP	76	110/70	18	RIGHT LUNG CONSOLIDAT ION	4	3	RECOVE RED
61	VISHWANATH SHALAWALI	83	M	PARKINSONS	CARDIOGENIC SHOCK SECONDARY TO ISCHEMIC HEART DISEASE	10	6	NONE	110	100/60	18	PULMONARY EDEMA	4.2	5	DIED
62	KASHIBAI BHIMARAO SAVVAGE	85	F	BRONCHIAL ASTHMA	ACUTE EXACERBATION OF BRONCHIAL ASTHMA WITH TYPE 2 RESP. FAILURE	13	3	NONE	120	130/70	38	PULMONARY ARTERY HTN	4.2	6	RECOVE RED
63	ABDUL	87	M	OLD CVA	POSTERIOR CIRCULATION STROKE	10	5	NONE	104	140/100	30	NORMAL	3.1	8	RECOVE RED
64	MALLAPPA	90	M	NONE	NSTEMI WITH OLD CVA	11	6	NONE	86	170/100	28	PULMONARY EDEMA	3.6	5	RECOVE RED