"COMPARISON OF CAPILLARY BLOOD GLUCOSE VERSUS ARTERIAL BLOOD GLUCOSE IN DIABETIC PATIENTS ADMITTED IN ICU ON VASOPRESSOR SUPPORT – A ONE YEAR CROSS SECTIONAL OBSERVATIONAL STUDY"

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ENDORSEMENT

This is to certify that the dissertation entitled **"COMPARISON OF CAPILLARY BLOOD GLUCOSE VERSUS ARTERIAL BLOOD GLUCOSE IN DIABETIC PATIENTS ADMITTED IN ICU ON VASOPRESSOR SUPPORT – A ONE YEAR CROSS SECTIONAL OBSERVATIONAL STUDY"** is a bonafide research work done by **REG NO. BG0116009.**

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LIST OF ABBREVIATIONS USED

-	-	Absent
+	-	Present
CLD	-	Central laboratory devices
POC	-	Point of care
ICU	-	Intensive care unit
IIT	-	Intensive insulin therapy
EGA	-	Error grid Analysis
GOx	-	Glucose oxidase
GDH	-	Glucose-1- dehydrogenase
FAD	-	Flavin dinucleotide
PQQ	-	Pyrroloquinoline quinone
ISO	-	International Organisation for Standardisation
ADA	-	American diabetes association
CSA	-	Canadian standards association
FDA	-	US Food and drug administration
CV	-	Coefficient of variation
IMSS	-	Instituto medicano del seguro social
CLSI	-	Clinical and laboratory standards institute

HPA	-	Hypothalamic-pituitary-adrenal
TNF	-	Tumour necrosis factor
IL	-	Interleukins
GLUT	-	Glucose transporters
NADP.	-	Nicotinamide adenine dinucleotide phosphate
TLC	-	Total leucocyte count
SEG	-	Surveillance error grid
SD	-	Standard deviation
MODS	-	Multiorgan dysfunction syndrome
HTN	-	Hypertension
AKI	-	Acute kidney injury
HOCM	-	Hypertrophic obstructive cardiomyopathy
ARDS	-	Acute respiratory distress syndrome
COPD	-	Chronic obstructive pulmonary disease
HTN	-	Hypertension
LRTI	-	Lower respiratory tract infection
UTI	-	Urinary tract infections
CKD	-	Chronic kidney disease
CLD	-	Chronic liver disease

IHD	-	Ischaemic heart disease
PVD	-	Peripheral Vascular disease
ESRD	-	End stage Renal disease
Ca Cx	-	Carcinoma cervix
T2DM	-	Type 2 diabetes mellitus
mg/dL	-	Milligrams per deciliter
mm Hg	-	Millimeters of mercury
CPAP	-	Continuous positive airway pressure
SIMV	-	Synchronised intermittent mechanical ventilation
NA	-	Noradrenaline
CEG	-	Clarke’s error grid analysis

ABSTRACT

Background: Glycemic control in critically ill patients decreases infection and mortality. Patients receiving vasopressors have altered peripheral perfusion, which may affect accuracy of capillary blood glucose values measured with point of care device.

Objective: Comparison of capillary blood glucose versus arterial blood glucose in diabetic patients admitted in ICU on vasopressor support.

Methods: The accuracy of capillary and arterial blood glucose meter measurements was compared with central laboratory arterial glucose measurements; the factors associated with inaccurate measures were also determined. The accuracy of arterial and capillary samples v/s standard lab method were evaluated according to the intra class correlation coefficient, on the basis of International Organization for Standardization (ISO 15197:2013 standards). Surveillance Error Grid (SEG) analysis and Clarke's error grid was used for analysis. Agreement between the two samples was determined using the method of Bland and Altman.

RESULTS:

The level of agreement of both capillary blood glucose and arterial blood glucose measured by the glucometer with respect to standard laboratory method according to the ISO 15197 guideline 2013 was only 80.0% and 81.6% respectively. Bland–Altman plot showed a mean bias of -1.44 mg/dL in capillary POCT method whereas in arterial POCT method, mean bias was 2.37 mg/dL. Distribution of the results in Surveillance Error Grid analysis shows that, capillary blood glucose values had (10.4%) data pairs outside no risk zone while arterial blood glucose group had only 5.6% data pairs outside no risk zone. But none of the values were outside of the slight lower risk zone (light green).

As per Clarke's error grid analysis, 84% of the capillary POCT and 90.4% of arterial POCT values with respect to standard lab values were within Zone A and all other remaining values were within zone B indicating that none of the values led to erroneous treatment clinically.

CONCLUSION:

- Arterial point of care testing values had slightly better agreement in comparison to capillary point of care testing which could be deemed negligible.
- Both the values were inaccurate according to ISO standards although they had reasonably good correlation with standard lab values.
- Most studies conducted so far have shown and recommended arterial blood glucose estimation at the bedside over capillary point of care testing among critically ill patients. As per our study we choose to conclude that there is no significant variation between arterial and capillary point of care testing for blood glucose estimation. Considering arterial as a more invasive method, capillary method of glucose estimation stands as a reasonable and easy option for bedside glucose monitoring among critically ill patients on vasopressor support.

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INTRODUCTION

Glycaemic control is an essential part of caring for critically ill patients and improves outcomes. Hyperglycaemia is associated with increased morbidity and mortality, hence maintaining glucose levels below 180 mg/dL reduces mortality and morbidity, decreases the incidence of wound infections, reduces hospital length of stay, and enhances long-term survival.[1]

Blood glucose measurement can be accomplished by sending a venous or arterial sample to laboratory or by blood gas analyser. The drawback of this method is the higher cost, longer time required to obtain the results and problem of iatrogenic blood loss and resultant anaemia. Alternatively, blood glucose can also be estimated at the bedside with handheld glucometers .Although bedside glucometry using capillary blood is a well established method for ambulatory and hospital ward patients, the accuracy of this method is contentious in the critically ill patients admitted to intensive care units (ICU) with several studies reporting arterial samples to be more reliable than the capillary ones.

However, these studies had mixed ICU patients, not differentiating patients with shock on vasopressor support. Patients with shock, on vasopressor support have peripheral hypoperfusion due to low perfusion index. Peripheral vasoconstriction can lead to increased glucose extraction by the tissues because of low capillary flow and increased glucose transit time. Hence, estimation of blood glucose from capillary blood may be unreliable in patients with septic shock. This may not apply to other critically ill patients who are not in shock. Therefore, clubbing of these patients with those with septic shock may not be appropriate when assessing the efficacy of

capillary blood in monitoring blood glucose levels. [2] Therefore, we undertook this study to compare the accuracy of arterial and capillary bedside glucometry among critically ill patients in shock on vasopressor support.

OBJECTIVES

Comparison of capillary blood glucose versus arterial blood glucose in diabetic patients admitted in ICU on vasopressor support.

REVIEW OF LITERATURE

Elevated glucose levels in critically ill patients have been shown to be related to increased mortality and length of hospital stay in adults and children.[3,4] The impact of tight glycemic control on clinical outcomes of patients in the intensive care setting has recently gained recognition. Landmark studies by Van den Berghe et al. [5,6] and others demonstrated reduced mortality in patients who reached target glucose values in the range of 80–110 mg/dl and whose stay in the medical intensive care unit (ICU) was longer than 72 hours and reduced morbidity in all patients who reached these strict target glucose values. In a pooled data set analysis,[7] the same group showed that intensive insulin therapy with target blood glucose <110 mg/dl was beneficial for all medical or surgical ICU patients except those with a prior diagnosis of diabetes, yet also carried a greater risk for hypoglycaemia. Thus, it is now accepted that insulin-based treatment regimens decrease morbidity and mortality in critically ill patients,[8] yet strict glycemic control should be performed in a manner that minimises the risk of hypoglycaemia. Importantly, as the majority of ICU patients are at decreased levels of consciousness and increased stress, the detection of hypoglycaemia in these patients depends solely on glucose monitoring. The American Diabetes Association and the American Association of Clinical Endocrinologists have published guidelines that recommend a glucose target as close to 110 mg/dl for all critically ill patients.[9,10]

A cost analysis study of intensive glycemic control in critically ill adult patients revealed that strict glucose control saved an average of $1580 per patient.[11] This substantial saving was the result of shorter ICU and overall hospital length of stay, decreased ventilator-dependent days, and reduced total laboratory costs. Similarly, in mechanically ventilated patients admitted to a surgical intensive care

unit, the excess cost of hospitalisation in patients treated conventionally compared to those treated according to the intensive insulin regimen was 2638 Euro per patient.[12] These observations suggest that the cost of intensive glucose management is outweighed by the improved clinical outcomes and is worth pursuing also from an economical standpoint.

There are two options to accomplish frequent ICU blood glucose monitoring: traditional central laboratory devices (CLDs) or point-of-care (POC) meters. Blood gas analysers (BGAs), which measure glucose as part of a panel of other laboratory values, can be considered as an alternative form of a CLD. Self-monitoring of blood glucose devices, originally designed and manufactured for home use, have been redesigned for inpatient POC use at the hospital bedside. These meters have the advantage of being very portable, providing quick results, and requiring very little blood sample volume. However, there is a decrement in accuracy compared with CLDs. The CLDs are very accurate yet require larger blood volumes and may not be in close proximity to the ICU patient. Though not used primarily for glucose management, BGAs have similar accuracy and precision to CLDs. Blood gas analysers also benefit from being stationed closer to the ICU and thus could be considered as POC devices. However, for the ICU staff that must run the samples to the blood gas laboratory, they are potentially more time consuming than handheld POC meters. Also, as BGAs automatically process other blood chemistry values (e.g., pH, PaCO2, potassium, and sodium levels), the cost of using this method for IIT in the ICU may be substantial.

Accuracy

There are a number of different statistical methods used to assess accuracy. Correlation and regression analysis, Bland–Altman, and Clarke error grids all serve as accepted metrics. Although bias affects the validity of using correlation to compare two different methods of measurement,[13] regression analysis will show the deviation from the line of equality in blood glucose values between devices. This has been particularly illustrative at hypoglycaemic levels.[14] The Bland–Altman method plots the mean of paired glucose values versus the absolute difference between the paired values.[15] One value comes from the reference instrument and the other comes from the instrument under evaluation. The 95% limits of agreement is calculated from the standard deviation of the difference values × 1.96. These graphs show bias and variation between two different instruments. To assess the clinical impact of differences between glucose measuring devices, the Clarke error grid analysis (EGA) is the most often accepted tool. The EGA depicts the relative difference in values between devices, with the reference device usually on the x axis. An ideal device should have a high degree of accuracy (i.e., zone A and B values). A number of physiologic derangements in the ICU patient may affect accuracy, including poor perfusion states,[16] pH,[17] anaemia,[18] renal failure,[19] and high oxygen tension levels.[20]

Point-of-Care Devices

Unlike CLDs, which measure blood glucose levels from plasma, POC meters analyse whole blood. After the blood drops onto the test strip, plasma from the whole blood percolates into the strip layer which, in the majority of POC meters, contains one of two enzymes: GOx or glucose-1-dehydrogenase (GDH). The GOx methodology produces gluconicacid and hydrogen peroxide or ferrocyanide. The

amount of hydrogen peroxide produced results in a colour change (reflectometric), and the density of the colour change is proportional to the blood glucose value. When ferrocyanide is the byproduct, its concentration is measured by current, and the amount of the current (amperometric) is proportional to the blood glucose level. In the GDH method, NAD is converted to NADH. The concentration of NADH is proportional to the blood glucose level. Glucose is also oxidized by the enzyme glucose dehydrogenase. In older GDH meters, pyrroloquinoline quinone (PQQ) is reduced to PQQH2. The latter interacts with ferricyanide, which is reduced to ferrocyanide. The ferrocyanide donates its electron to a palladium electrode, converting the ferrocyanide back to ferricyanide.[21] There are a number of POC glucose meters that are targeted for the hospital market.[22] However, the accuracy standard that is currently applied to POC meters for the home market is the only regulatory hurdle needed for hospital use. The International Organisation for Standardisation guideline (ISO 15197) states that "ninety-five (95%) of the individual glucose results shall fall within ±15 mg/dl of the results of the manufacturer's measurement procedure at glucose concentrations ≤75 mg/dl and within ±20% at glucose concentrations >75 mg/dl." [23] Clearly, allowing up to 5% of results outside of these already loose targets seems to be inadequate for hospital use. Although these meters are marketed by various manufacturers specifically for use in the hospital environment, it is unclear if the technologies or accuracy profiles are actually any different or better than what these companies are marketing to the home glucose market.

Meter Performance Criteria for Acceptable Agreement between a Glucose Meter and Results from a Comparative Laboratory Method

Organization or society	Glucose range	Performance criteria
ADA 1987	All levels	±15%
ADA 1994	All levels	±5%
CSA	<45 mg/dl (2.5 mmol/liter)	±25% (CV < 12.5%)
	≥90 mg/dl (5.0 mmol/liter)	±15% (CV < 7.5%)
FDA (95% of data)	<100 mg/dl (5.6 mmol/liter)	±20 mg/dl (1.1 mmol/liter)
	≥100 mg/dl (5.6 mmol/liter)	±20%
ISO (95% of data)	<100 mg/dl (5.6 mmol/liter)	±10 mg/dl (1.1 mmol/liter)
	≥100 mg/dl (5.6 mmol/liter)	±20%
IMSS	<60 mg/dl (3.3 mmol/liter)	±25%
	≥60 mg/dl (3.3 mmol/liter)	±20%
CLSI (C30A)	<100 mg/dl (5.6 mmol/liter)	<15 mg/dl (0.83 mmol/liter)
	≥100 mg/dl (5.6 mmol/liter)	±20%
TNO	<117 mg/dl (6.5 mmol/liter)	±20 mg/dl (1.11 mmol/liter)
	≥117 mg/dl (6.5 mmol/liter)	±15 mg/dl (0.83 mmol/liter) (CV < 10%)

CV, coefficient of variation; CSA, Canadian Standards Association; FDA, U.S. Food and Drug Administration; ISO, International Organization for Standardization; IMSS, Instituto Mexicano del Seguro Social; CLSI, Clinical and Laboratory Standards Institute; TNO, Nederlandse Organisatie voor Toegepast Natuurwetenschappelijk Onderzoek

Figure 1. A previously published table showing meter performance criteria for acceptable agreement between a glucose meter and a CLD. Reprinted with permission from *Journal of Diabetes Science and Technology*.[42]

Factors Affecting Accuracy of Glucose Measurement:

A potential error in current practice arises from the use of blood and plasma glucose as interchangeable terms, with the consequent risk of misinterpretation.[24] The glucose concentration in plasma is approximately 11% higher than that in whole blood due to the higher water content in plasma (93%) compared with erythrocytes (73%), and therefore a multiplier of 1.11 for the conversion of glucose in blood to plasma has been recommended.[25] Use of plasma glucose concentration is suggested because the physiological activity of glucose corresponds more closely with plasma concentration than whole blood glucose concentration, which varies considerably with hematocrit.[26,27] Although POC devices measure glucose in whole blood, they almost all self-correct internally, reporting results as plasma glucose. In the United States, plasma glucose levels are reported most commonly in milligrams per deciliter, but in many other countries are reported in Système International units of millimoles per liter, with 18 mg/dl = 1 mmol/liter.

Sampling Site: The ADA and World Health Organisation recommend the use of venous plasma glucose for measuring and reporting, but recognise the widespread use of capillary blood sampling[28] (fingertip blood samples are commonly expressed as capillary blood samples), despite evidence that this may lead to measurement error.[29] The difference between capillary and venous glucose is typically small in nonhypotensive fasting subjects, but can be up to 8% higher in capillary blood after meals or glucose challenge.[30,31] In some studies, arterial blood glucose has been shown to be significantly higher than both capillary and venous blood glucose,[32,33] whereas the difference was clinically unimportant in another study.[34]

Compared to capillary blood, arterial sampling is generally accepted to be a more accurate measurement.[35,36]

Patient and Environmental Factors: Poor peripheral perfusion (e.g., circulatory shock) results in increased tissue glucose extraction and a lower glucose value in capillary than venous blood. Capillary blood glucose specimens from severely hypotensive patients are more likely to underestimate arterial and central venous blood glucose, resulting in an incorrect diagnosis of systemic hypoglycaemia compared to normotensive patients.[37] Similar results have been obtained in intensive care patients.[36,38-40] Anaemia decreases and polycythemia increases the difference between whole blood and plasma glucose not only for the aforementioned reasons, but also impedance of plasma diffusion into the test strip by the higher viscosity (increased hematocrit) may alter results obtained by some POC devices[41,42] (see Figure 1, graph D).[43]This effect has particular relevance in critically ill neonates in whom the hematocrit may vary widely, and rapid detection of hypoglycaemia is essential. Many POC devices have been shown to be inadequate in this setting.[44-47]

Point-of-care device test strips using glucose oxidase (see later) are prone to errors caused by oxygen effects. Tang and colleagues showed that errors of 15% or more could occur with highly oxygenated blood samples (i.e., PaO2 >100 mm Hg),[48] and variances of up to 46% from reference values have been observed under hyperbaric conditions.[49-52] These devices have also been shown to underestimate blood glucose at altitude (i.e., low ambient PO2) by 1–2% per 1000 feet of elevation,[53,54] and errors of more than 15% have been shown when analysing hypoxic blood (PO2 < 44 mm Hg).[55] The severity of errors with low PO2 is highly dependent on the type of test strip (electrochemical vs photometric) and type of enzyme employed in different test

strips. The effect of temperature on the test strip reaction rate has been shown to cause clinically relevant reductions in the accuracy of some devices, with low temperatures typically causing underestimation and high temperatures causing overestimation of true blood glucose,[56,57] even within the limits specified by the manufacturers. Glucose concentrations also follow a circadian rhythm.[58] Patient and environmental factors affecting accuracy are summarised in Table 1. [42]

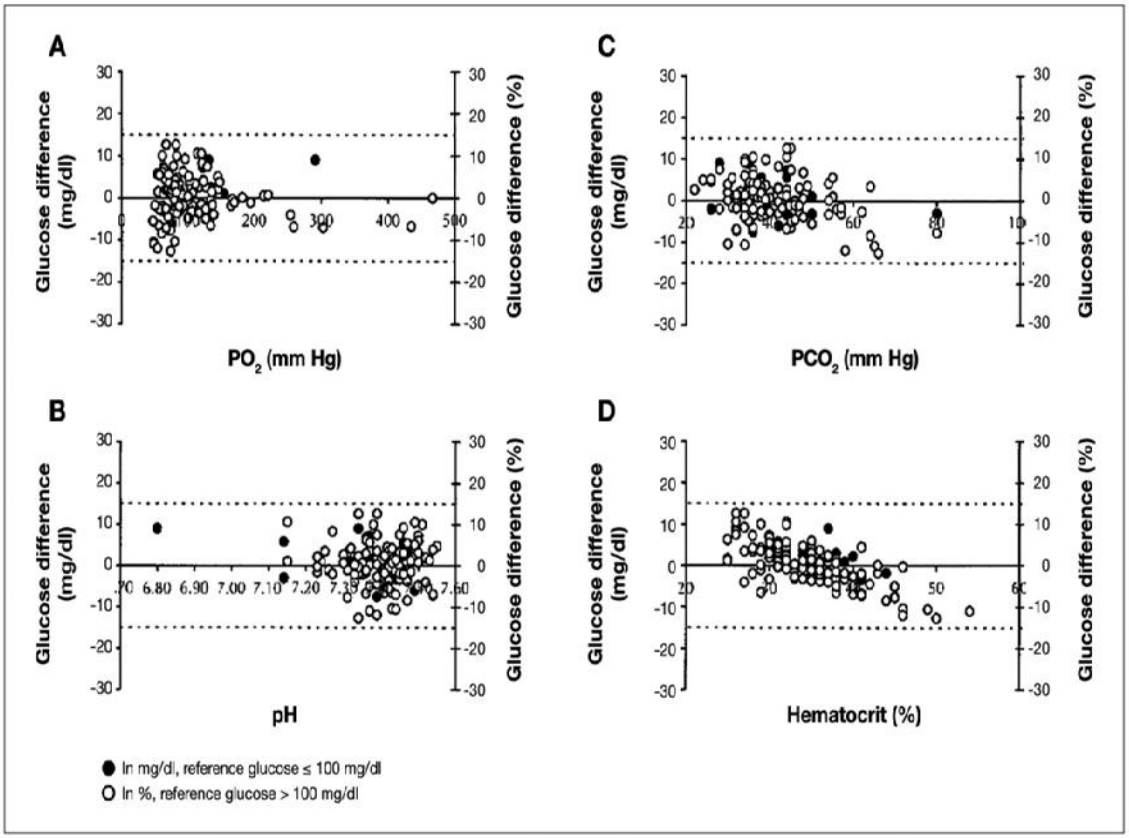


Figure 1. Plots of paired differences of SureStep Pro glucose measurements minus primary reference glucose measurements as a function of critical care variables (n = 129). Paired differences versus **(A)** PO_2, **(B)** pH, **(C)** PCO_2, and **(D)** hematocrit. The PO_2 range was 47–467 mm Hg; PCO_2, 22–80 mm Hg; pH, 6.80–7.55; and hematocrit, 25–54%. Dashed lines represent error tolerances, ± 15 mg/dl for glucose ≤100 mg/dl, and ≤15% for glucose >100 mg/dl. Reprinted with permission from *Archives of Pathology and Laboratory Medicine*.[39]

Table 1.
Confounding Variables in Glucose Measurement[a]

Variable	Methodology affected[b]	
	GO	GD
Whole blood	↓	↓
Arterial	↑	↑
Capillary	↑	↑
Postprandial state	↑	↑
Hematocrit		
Anemia	↑	↑
Polycythemia	↓	↓
Oxygen concentration		
Hypoxia	↑	–
Oxygen therapy	↓	–
pH (6.8–7.55)		
Low pH	– / ↓	–
High pH	– / ↑	–
Hypothermia	↑	↓ / ↑
Hypotension	↑	↓ / ↑
Drugs		
Ascorbic acid	↓	↓ / –
Acetaminophen	↓	↑
Dopamine	–	↓
Icodextrin	–	↑
Mannitol	↑	–

[a] Copyright © 2007 American Diabetes Association. From Diabetes Care®, Vol. 30, 2007: 403-409. Reprinted with permission from *The American Diabetes Association.*[38]
[b] Changes relative to venous plasma measured as central laboratory. GO, glucose oxidase; GD, glucose dehydrogenase.

Acute illness, the stress response and stress hyperglycemia

The stress response is mediated largely by the hypothalamic-pituitary-adrenal (HPA) axis and the sympatho-adrenal system. In general, there is a graded response to the degree of stress. Cortisol and catecholamine levels correlate with the type of surgery, the severity of injury, the Glasgow Coma Scale and the APACHE score.[59] Adrenal cortisol output increases up to ten-fold with severe stress (approximately 300 mg hydrocortisone per day).[59] In patients with shock, plasma concentrations of epinephrine increase 50-fold and norepinephrine levels increase 10-fold.[60] The adrenal medulla is the major source of these released catecholamines.[60] Adrenalectomy eliminates the epinephrine response and blunts the norepinephrine response to hemorrhagic shock .[60] The increased release of stress hormones results in multiple effects (metabolic, cardiovascular and immune) aimed at restoring homeostasis during stress. The HPA axis, sympathoadrenal system and proinflammatory cytokines (TNF-α, IL-1 and IL-6) act collectively and synergistically to induce stress hyperglycemia.

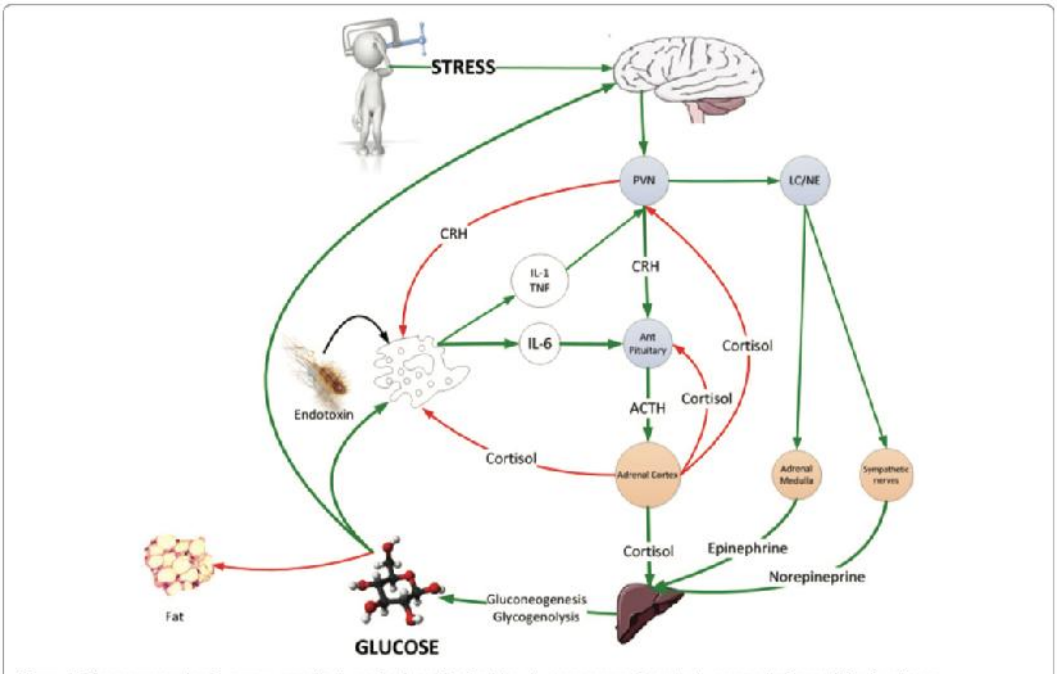


Figure 1. The neuroendocrine response to stress is characterized by gluconeogenesis and glycogenolysis resulting in stress hyperglycemia providing the immune system and brain with a ready source of fuel. ACTH, adrenocorticotrophic hormone; CRH, corticotrophin releasing hormone; LC/NE, locus ceruleus norepinephrine system; PVN, paraventricular nucleus.

The neuroendocrine response to stress is characterised by excessive gluconeogenesis, glycogenolysis and insulin resistance (Figure 1).[61]Stress hyperglycaemia, however, appears to be caused predominantly by increased hepatic output of glucose rather than impaired tissue glucose extraction. The metabolic effects of cortisol include an increase in blood glucose concentration through the activation of key enzymes involved in hepatic gluconeogenesis and inhibition of glucose uptake in peripheral tissues such as the skeletal muscles.[61] Both epinephrine and norepinephrine stimulate hepatic gluconeogenesis and glycogenolysis; norepinephrine has the added effect of increasing the supply of glycerol to the liver via lipolysis. Inflammatory mediators, specifically the cytokines TNF-α, IL-1, IL-6, and C-reactive protein, also induce peripheral insulin resistance (Figure 2).[61] In addition, the altered release of adipokines (increased zinc-alpha2 glycoprotein and decreased adiponectin) from adipose tissue during acute illness is thought to play a key role in the development of insulin resistance.[62] The degree of activation of the stress response and the severity of hyperglycaemia are related to the intensity of the stressor and the species involved. Hart and colleagues[63] demonstrated that hemorrhage, hypoxia and sepsis were amongst those stressors that resulted in the highest epinephrine and norepinephrine levels.

Mild to moderate stress hyperglycaemia is protective during stress and critical illness: Stress hyperglycaemia and insulin resistance are evolutionarily preserved responses that allow the host to survive during periods of severe stress.[64] Insects, worms and all verterbrates, including fish, develop stress hyperglycaemia when exposed to stress.[64,65] In animal models of hemorrhagic shock the administration of a hypertonic glucose solution increased cardiac output, blood pressure and improved survival.[66] In these experiments, similar osmolar doses of saline or mannitol, with

greater accompanying fluid volumes, failed to produce the sustained blood pressure changes or to improve the survival.

Glucose is largely utilised by tissues that are non- insulin dependent, and these include the central and peripheral nervous system, bone marrow, white and red blood cells and the reticuloendothelial system.[67] It has been estimated that, at rest, non-insulin mediated glucose uptake accounts for 75 to 85% of the total rate of whole glucose disposal. Glucose is the primary source of metabolic energy for the brain. Cellular glucose uptake is mediated by plasma membrane glucose transporters (GLUTs), which facilitate the movement of glucose down a concentration gradient across the non-polar lipid cell membrane.[67] These transporters are members of a family of structurally related facilitative glucose transporters that have distinct but overlapping tissue distribution. Although 14 GLUT isoforms have been identified in the human genome, glucose uptake *per se* is facilitated by GLUT-1, GLUT-3 and GLUT-4 in various tissues. Insulin increases GLUT-4-mediated glucose transport by increasing translocation of GLUT-4 from intracellular stores to the cell membrane.[67] Thermal injury and sepsis have been demonstrated to increase expression of GLUT-1 mRNA and protein levels in the brain and macrophages.[68,69] Concomitantly, stress and the inflammatory response result in decreased translocation of GLUT-4 to the cell membrane. It is likely that pro-inflammatory mediators, particularly TNF-α and IL-1, are responsible for the reciprocal effects on the surface expression of these glucose transporters (Figure 2).[61] Elevated TNF-α directly interferes with insulin signal transduction through the phosphorylation of various molecules along the insulin signaling pathway. During infection, the upregulation of GLUT-1 and downregulation of GLUT-4 may play a role in redistributing glucose away from peripheral tissues towards immune cells and the nervous system.

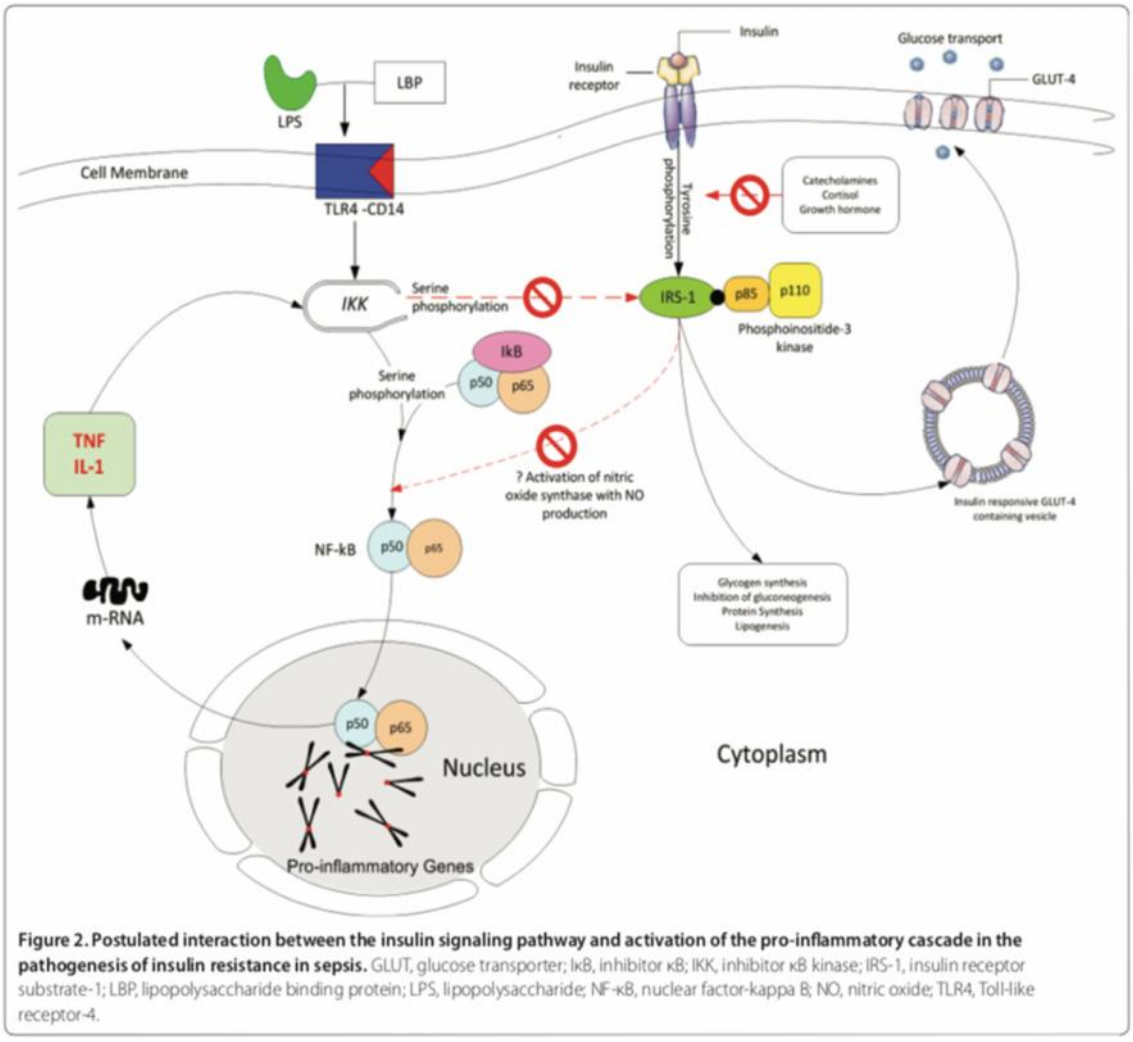


Figure 2. Postulated interaction between the insulin signaling pathway and activation of the pro-inflammatory cascade in the pathogenesis of insulin resistance in sepsis. GLUT, glucose transporter; IκB, inhibitor κB; IKK, inhibitor κB kinase; IRS-1, insulin receptor substrate-1; LBP, lipopolysaccharide binding protein; LPS, lipopolysaccharide; NF-κB, nuclear factor-kappa B; NO, nitric oxide; TLR4, Toll-like receptor-4.

For glucose to reach a cell with reduced blood flow (ischemia, sepsis), it must diffuse down a concentration gradient from the bloodstream, across the interstitial space and into the cell. Glucose movement is dependent entirely on this concentration gradient, and for adequate delivery to occur across an increased distance, the concentration at the origin (blood) must be greater. Stress hyperglycaemia results in a new glucose balance, allowing a higher blood 'glucose diffusion gradient' that maximises cellular glucose uptake in the face of maldistributed microvascular flow.[70] These data suggest that moderate hyperglycaemia (blood glucose of 140 to 220 mg/dL) maximises cellular glucose uptake while avoiding hyper- osmolarity. Furthermore, acute hyperglycaemia may protect against cell death following ischemia by promoting anti-apoptotic pathways and favouring angiogenesis. In a murine

myocardial infarction model, Malfitano and colleagues[71] demonstrated that hyperglycaemia increased cell survival factors (hypoxia inducible factor-1α, vascular endothelial growth factor), decreased apoptosis, reduced infarct size and improved systolic function. In this study, hyperglycemia resulted in increased capillary density and a reduction in fibrosis. *In vitro* and *in vivo* studies have demonstrated that cardiomyocytes exposed to an insulin-free medium supplemented with high glucose concentrations are resistant to pathological insults such as ischemia, hypoxia and calcium overload. [72]

Macrophages play a central role in the host response to injury, infection and sepsis. Macrophage activities include antigen presentation, chemotaxis, phagocytosis, bactericidal activity, cytokine secretion and wound repair. Glucose is the primary metabolic substrate for the macrophage and efficient glucose influx is essential for optimal macrophage function. Macrophages and neutrophils require NADPH for the formation of the reactive oxygen species, nitric oxide and superoxide as well as many biosynthetic pathways. Metabolism of glucose via the pentose pathway provides the metabolic intermediates required for the generation of NADPH. Following thermal injures, trauma and sepsis, non-insulin mediated glucose uptake is increased. The majority of the increased glucose uptake occurs in macrophage rich tissues.[73,74] These data suggest that the increased energy requirements of activated macrophages and neutrophils during infection and tissue injury are regulated by enhanced cellular glucose uptake related to the increased glucose diffusion gradient and increased expression of glucose transporters. In addition, these mechanisms ensure adequate glucose uptake by neuronal tissue in the face of decreased microvascular flow. Iatrogenic normalisation of blood glucose may therefore impair immune and cerebral function at a time of crises. Indeed, two independent groups of investigators using

microdialysis and brain pyruvate/lactate ratios demonstrated that attempts at blood glucose normalisation in critically ill patients with brain injury were associated with a greater risk of critical reductions in brain glucose levels and brain energy crisis. [75,76] Similarly, Duning and colleagues[77] demonstrated that hypoglycaemia worsened critical illness induced neurocognitive dysfunction. Multiple studies have demonstrated that even moderate hypoglycaemia is harmful and increases the mortality of critically ill patients.[78,79] In summary, these data suggest that stress hyperglycaemia provides a source of fuel for the immune system and brain at a time of stress and that attempts to interfere with this evolutionarily conserved adaptive response is likely to be harmful.

METHODOLOGY

The present study was conducted in the Intensive care unit of K.L.E.S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi, from January 2017 to December 2017.

Study design and duration

The study design was a hospital based observational cross-sectional study.

Study period

The present study was done for the period of one year from January 2017 to December 2017.

Place

The present study was carried out in the Intensive care unit of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belgaum.

Source of Data

Diabetic patients on vasopressor support (within 24 hrs of initiation on vasopressor support) admitted in ICU at KLES Dr. Prabhakar Kore Hospital and MRC, Belgaum

Sample size

A total of 125 diabetic patients admitted in the ICU who were on vasopressor support were included in the study.

Sampling procedure

Sample size was calculated using the formula as below.

$N = [(Z\alpha + Z\beta)\sigma/d]^2$

$Z\alpha = 1.96$ (type 1 error)

$Z\beta = 0.84$ (type 2 error)

$\sigma = 28$ (±2 Standard Deviation)

$d = 7$ (mean absolute difference)

Sampling method: Convenient sampling method

Hence the sample size of 125 was considered for this study.

Selection criteria

Inclusion Criteria

- Patients aged 18 and above.
- Diabetic and Pre-diabetic patients (HbA1C >5.7% or RBS ≥140mg/dl) admitted in ICU on vasopressor support (within 24 hrs of initiation on vasopressor support).
- Patients having an arterial line for blood pressure monitoring

Exclusion Criteria

- Patients aged < 18 years old.
- Presence of Peripheral Oedema
- Patients with haematocrit of <20% or >70%

Ethical clearance

Prior to the beginning, the study was approved by the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belagavi.

Informed consent

The patients who fulfilled the selection criteria were informed about the nature of study and a written informed consent was obtained (Annexure–I).

Data collection

The demographic data of the patients was noted along with the history of presenting illness and other comorbid conditions. Further these patients underwent clinical examination followed by systemic examination. Patients were evaluated for the following parameters on admission.

- Body temperature was measured by a medical thermometer.
- Blood pressure was measured by a sphygmomanometer on left upper arm.
- Heart rate was measured by palpatory method
- Respiratory rate.
- Oxygen saturation
- Vasopressor on flow

All these findings were noted on a predesigned and pretested proforma (Annexure-II).

Investigations

Patients were subjected to following investigations.

- Complete blood count
- Haematocrit
- Random blood sugar levels
- HbA1c
- Renal function test
- Liver function test
- Point of care capillary blood glucose estimation
- Point of care arterial blood glucose estimation

Procedure

- All the patients fulfilling the inclusion criteria and willing to participate were included in the study.
- Informed consent was obtained.
- Further they were subjected to a clinical examination and predesigned proforma.
- Glucose meter analysis of capillary blood (fingerstick), glucose meter analysis of arterial blood and central laboratory blood glucose measurement (from an arterial sample). Capillary blood samples were obtained from the patient's fingerstick sample by instillation of a drop of blood onto a test strip for glucose detection and analysed by a glucose meter (CONTOUR® TS, Bayer HealthCare; GDH/FAD chemistry). Arterial blood glucose measurements were obtained from the patient's arterial catheter: 3 mL of waste blood was first discarded, then a drop of blood was

analysed by the same glucose meter and 5 mL of blood sent to the central laboratory for biochemical analysis.

- Results of blood glucose checks from capillary and arterial sources were recorded. The type and number of intravenous vasopressor medications.

Data was analysed and tabulated

DATA ANALYSIS:

The collected data were entered into an excel sheet. The data was expressed in means and proportions and presented in the form of tables and graphs where ever necessary. The continuous variables viz., age in years, blood glucose values, haematocrit, Total Leucocyte Count (TLC), serum Creatinine were expressed in means and standard deviations. The means of continuous variables viz., capillary POCT blood glucose values v/s standard lab method and arterial POCT blood glucose values v/s standard lab method were compared using Paired t-test and the categorical variables were compared using chi-square test.

The accuracy of arterial and capillary samples v/s standard lab method were evaluated according to the intra class correlation coefficient, on the basis of International Organization for Standardization (ISO 15197:2013 standards); $<\pm$15mg/dL when POCT < 100 mg/dL & within 15% of reference value when POCT $\geq$ 100 mg/dL and Surveillance Error Grid (SEG) analysis.[80] A Pearson correlation coefficient (r) was used to evaluate the relationship between the mean arterial and capillary glucose measurement tested by POCT with standard lab method. Surveillance error grid (SEG) was plotted for clinical risk assessments of blood glucose monitor errors that assigns a unique risk score to each monitor data point

when compared to a reference value. The SEG allows the clinical accuracy of a blood glucose monitor to be portrayed as the percentages of data points falling into custom-defined risk zones.[81,82]

A Clarke error grid analysis is an internationally recognized way to evaluate and compare the accuracy of a testing device with a laboratory reference. Evaluation is based on comparing clinical consequences using the test strip method and the reference. This analysis partitions the total blood glucose range into zones, based on the effects of glucose variations on diabetes treatment and following are the zones and the respective interpretations.

- Zone A: Results to clinically correct treatment decisions either in hypoglycemia or hyperglycemia range.
- Zone B: >20% deviation from the reference; represents values that would lead to benign or no treatment error.
- Zone C: Represents values would lead to treatment decisions opposite to that called for by the blood glucose levels.
- Zone D: Represent a failure to detect and treat errors.
- Zone E: Is a clinically more serious error zone, glucose meter generated values that failed to detect hypoglycemia or hyperglycemia. Values are opposite the reference values resulting in corresponding treatment decisions opposite to those needed.

Data Analysis: Accuracy was also assessed using Clarke's error grid analysis.[111]

Agreement between the two samples was determined using the method of Bland and Altman.[4] We used the method of Bland and Altman[82] to plot the average of each laboratory standard method and capillary glucose pair against the laboratory standard method – capillary glucose difference; laboratory standard method and arterial glucose pair against the laboratory standard method and arterial glucose difference. The horizontal line in the mid of two limits of agreement (upper & lower) indicates mean of the laboratory standard method - capillary glucose pair and laboratory standard method - arterial glucose pair in the respective plots (the line of agreement). It is bounded by two parallel lines, known as the limits of agreement, which are drawn at 2 Standard Deviations (SD) above and below the line of agreement, the upper and lower limit of agreement.[83,84] Regression analysis was used to assess the statistical significance of Bland-Altman's plot. The analysis was done using SPSS version 16.0. A P- value of <0.05 was taken as statistically significant.

RESULTS

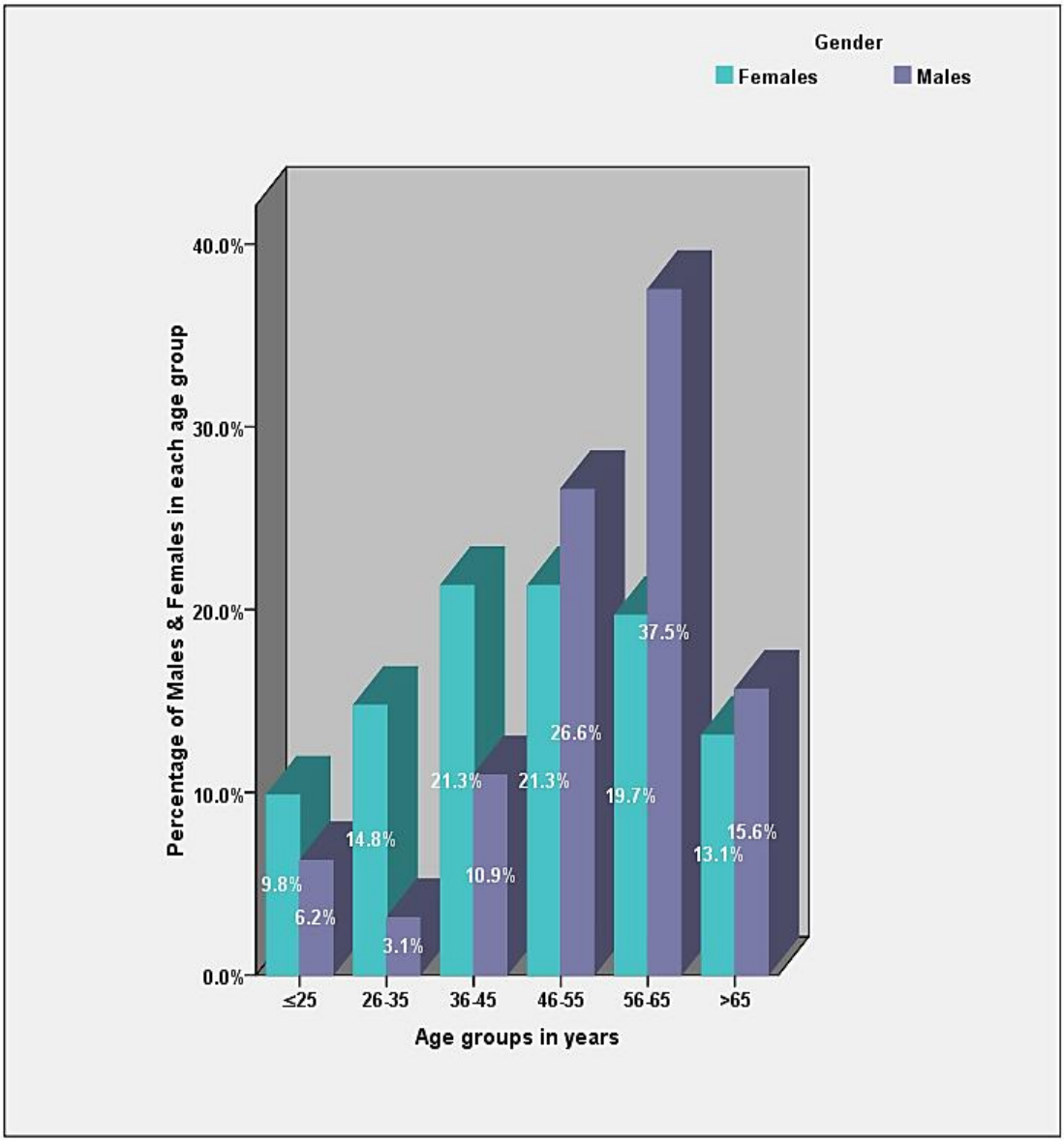
Socio-demographic characteristics of study subjects

Table 1: Distribution of the study subjects according to age and gender

Age in years	Males	Females	Total
≤ 25	04 (6.2)	06 (9.8)	**10 (8.0)**
26-35	02 (3.1)	09 (14.8)	**11 (8.8)**
36-45	07 (10.9)	13 (21.3)	**20 (16.0)**
46- 55	17 (26.6)	13 (21.3)	**30 (24.0)**
56-65	24 (37.5)	12 (19.7)	**36 (28.8)**
>65	10 (15.6)	08 (13.1)	**18 (14.4)**
Total	**61 (100.0)**	**64 (100.0)**	**125 (100.0)**

Figures in parenthesis indicate percentage

In the present study, out of 125 study subjects, 64 (51.2%) were males and 61 (48.8%) were females. Majority i.e., 36 (28.8%) of the study subjects were in the age group of 56-65 years followed by 30 (24.0 %) were in the age group of 46 - 55 years, 20 (16.0%) in 36 - 45 years, 18 (14.4%) in > 65 years, 11 (8.8%) in 26-35 years and 10 (8.0%) in the age group ≤ 25 years. The mean age was 50.59 + 14.60 years with a range from 18 to 78 years. The mean age of males and females were 53.56+13.09 years and 47.48 + 15.53 years respectively.



Graph 1: Distribution of males and females based on age

Majority of the subjects were females in the age groups ≤45 years and majority were males in the age group >45 years.

Table 2: Distribution of study subjects admitted to ICU according to different diagnosis

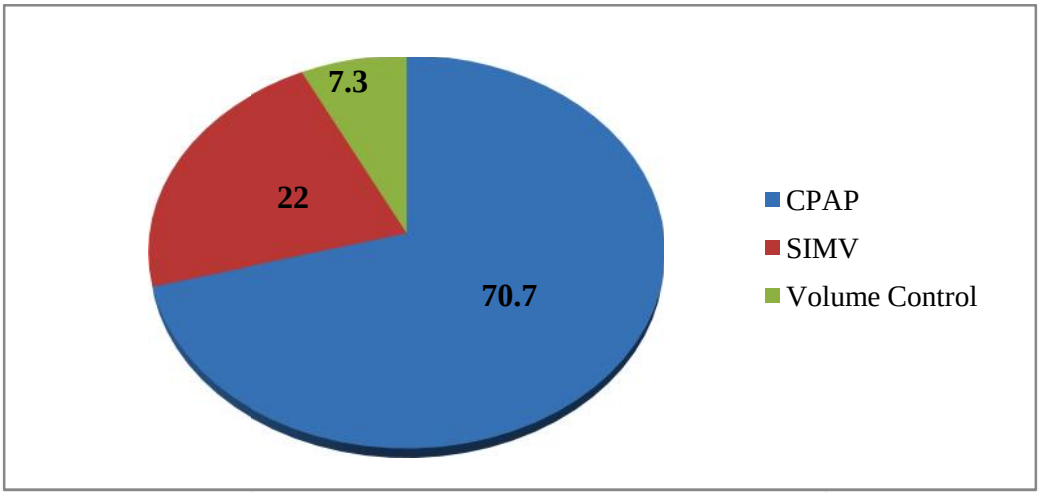
Diagnosis	Number (n=125)	Percent (%)
Gastro-intestinal System (GI)		
Peritonitis	08	6.4
Acute Gastro-Enteritis with/without MODS	06	4.8
Portal HTN with Chronic Liver Disease with/without upper GI bleed	06	4.8
	03	2.4
Cardio-vascular System		
Ischaemic heart disease	04	3.2
Dilated cardiomyopathy/HOCM[•]	03	2.4
Respiratory System		
Pneumonia with/without ARDS	32	25.6
Acute exacerbation of COPD[•]	10	8.0
Pleural Effusion (Tubercular)	04	3.2
Bronchogenic carcinoma	04	3.2
LRTI[•]	03	2.4
Interstitial Lung Disease	02	1.6
Central Nervous System		
Cerebrovascular accident with/without intra Cranial Bleed	02	1.6
Renal System		
AKI[•] with/without sepsis	03	2.4
ARF with/without Chronic kidney disease Disease[•]	02	1.6
Pyelonephritis	02	1.6
Others		
Sepsis with/without MODS	24	19.2
Dengue Shock Syndrome	03	2.4
Dyselectrolytemia	03	2.4
Poisoning (Amitraz)	01	0.8

[•] - MODS- Multi-Organ Dysfunction Syndrome; Portal HTN- Portal Hypertension; HOCM-Hypertrophic Cardiomyopathy; ARDS-Acute Respiratory Distress Syndrome; COPD-Chronic Obstructive Pulmonary Disease; LRTI-Lower Respiratory Tract Infection; AKI-Acute Kidney Injury; ARF- Acute Renal Failure

Among the patients admitted to ICU, majority were admitted with Pneumonia with/without ARDS (32, 25.6%), followed by Sepsis with/without MODS secondary to pneumonia/UTI (24, 19.2%), Acute exacerbation of COPD (10, 8.0%), Peritonitis (8, 6.4%), Acute Gastro-Enteritis with/without MODS, Portal HTN with/without chronic Liver Disease/upper GI bleed (6, 4.8%) each, Ischaemic heart disease, Tubercular Pleural Effusion, Carcinoma Bronchus (4, 3.2%) each, Upper GI bleed , HOCM, LRTI, AKI, Dengue Shock Syndrome, Dyselectrolytemia (3, 2.4%) each, Interstitial Lung Disease, CVA with/without Intra Cranial Bleed, ARF with/without Chronic kidney Disease**,** Pyelonephritis (2, 1.6%) each and one accounting for 0.8% was admitted with Amitraz poisoning.

Table 3: Distribution of study subjects according to Comorbidities existing with Type 2 Diabetes Mellitus (T2DM)

Comorbidities	Number	Percentage
Yes	**54**	**43.2**
Chronic Kidney Disease	20	37.0
HTN	16	29.6
COPD	5	9.2
Hyperthyroidism	4	7.4
Chronic Liver Disease (CLD)	3	5.6
Ischaemic Heart Disease (IHD)	2	3.7
Peripheral Vascular Disease (PVD)	2	3.7
End Stage Renal Disease (ESRD)	1	1.9
Carcinoma Cervix (Ca Cx)	1	1.9
No	**71**	**56.8**



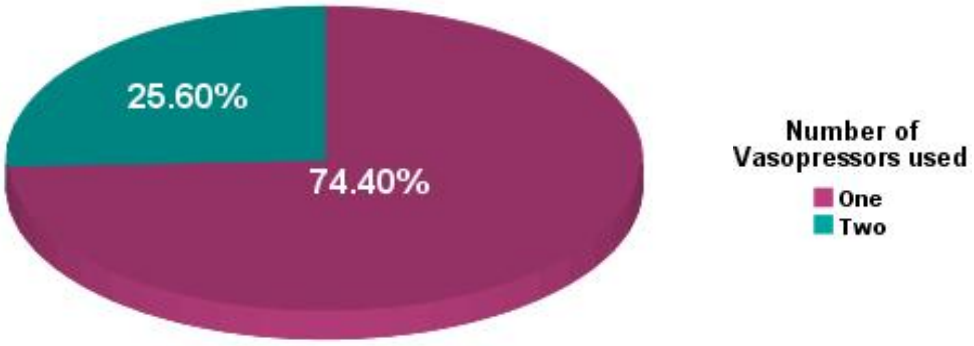
Graph-2: Percentage Distribution of Comorbidities (n=54)

In the above table and graph, majority of the study subjects i.e., 71, 56.8% of them did not have any co-morbidities apart from type 2 diabetes mellitus. Among those who had comorbidities existing with T2DM, majority had Chronic Kidney Disease (20, 37.0%) followed by Hypertension (16, 29.6%), COPD (5, 9.2%) among whom two had hypertension, hyperthyroidism (4, 7.4%), Chronic Liver Disease (3, 5.6%), Ischaemic Heart Disease, Peripheral Vascular Disease (2, 3.7%) each and ESRD, Carcinoma Cervix (1, 1.9%) each.

Table 4: Distribution of study subjects based on use of number of vasopressors

Number of Vasopressor	Number	Percentage (%)
One	93	74.4
Two	32	25.6
Total	**125**	**100.0**

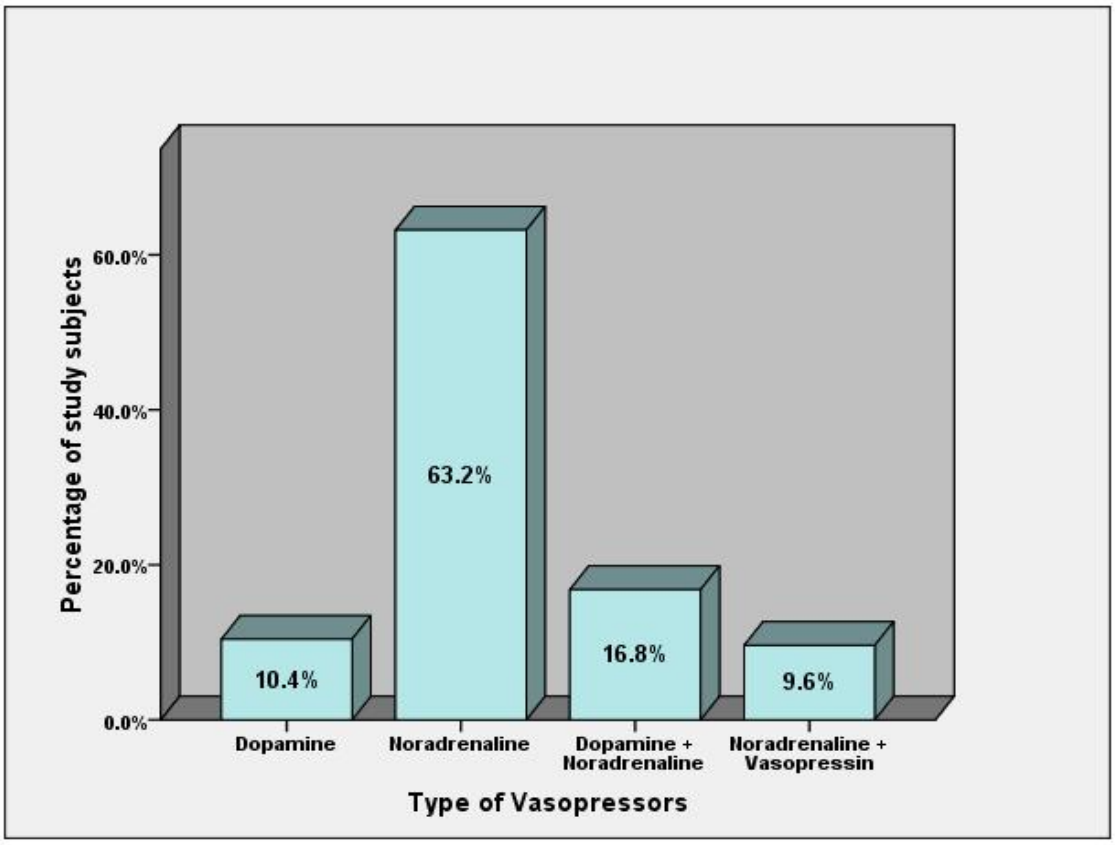
Graph-3: Percentage Distribution based on number of vasopressors used (n=125)



The above table and graph depicts that most of the study subjects i.e., 93, 74.4% were put on single vasopressor and remaining 32, 25.6% were on double vasopressors.

Table 5: Distribution of study subjects based on type of vasopressors

Type of Vasopressor	Number	Percentage (%)
Noradrenaline	79	63.2
Noradrenaline + Dopamine	21	16.8
Dopamine	13	10.4
Noradrenaline + Vasopressin	12	9.6
Total	**125**	**100.0**



Graph-4: Percentage Distribution based on type of vasopressors used (n=125)

The above table and graph depicts that most of the study subjects i.e., 79, 63.2% of them used Noradrenaline alone followed by 21, 16.8% used a combination of Dopamine with Noradrenaline, 13, 10.4% used Dopamine alone and 12, 9.6% used a combination of Noradrenaline and Vasopressin.

Table 6: Mean values of blood glucose levels based on the different site and analysis of blood glucose levels

Sites and type of analysis of Blood Glucose collected	Mean±SD
Capillary blood glucose using POCT§ (mg/dL)	142.61±38.96
Arterial blood glucose using POCT§ (mg/dL)	138.80±43.58
Laboratory Arterial blood glucose (mg/dL)	141.17±40.77

§ POCT – Point of Care Testing Device

The mean values of Capillary blood glucose and arterial blood glucose recorded using the POCT device were 142.61±38.96 mg/dL and 138.80±43.58 mg/dL respectively. The mean level of arterial blood glucose levels recorded in the lab was 141.17±40.77 mg/dL which seems to be slightly higher compared to the values tested by the POCT device.

Table 7: Mean values of other blood parameters

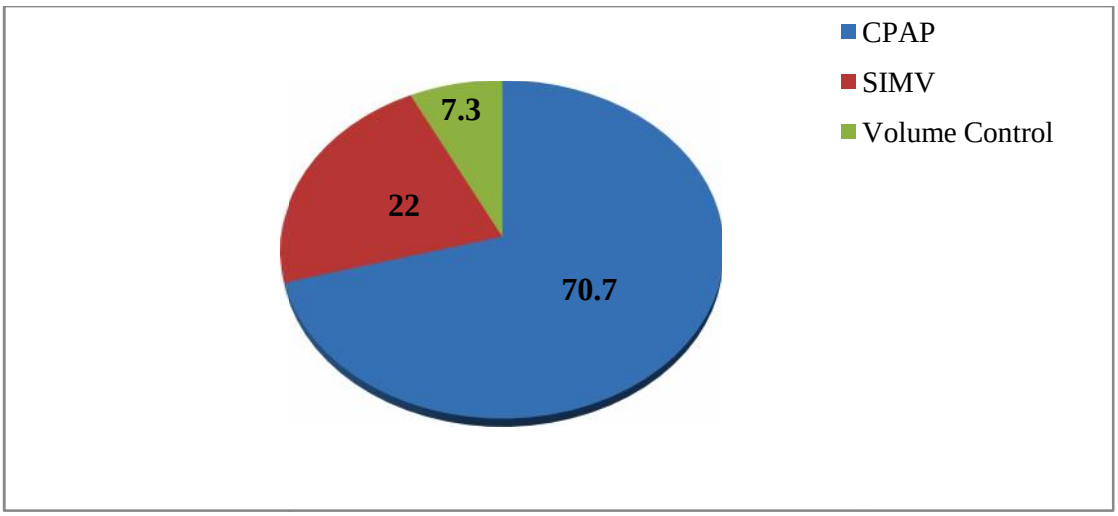
Blood Parameters	Mean±SD
Haematocrit (%)	38.66±7.21
Total Count (cells /µL)	14059±6210
Serum Creatinine§ (mg/dL)	1.4 (0.3 – 11.0)

§ - Expressed in Median (Range) because of its skewed distribution

The mean Haematocrit value was 38.66 % with a standard deviation of 7.21 %. The mean total count with a standard deviation was 14059±6210 cells/µL. The median serum creatinine was 1.4 mg/dL and it ranged from a minimum of 0.3 mg/dL to 11.0 mg/dL.

Table 8: Distribution of study subjects based on requirement and type of Ventilatory Support

Ventilatory Support	Number	Percentage
Yes	**41**	**32.8**
Continuous Positive Airway Pressure **(**CPAP) Synchronized Intermittent-Mechanical Ventilation (SIMV) Volume Control	29 9 3	70.7 22.0 7.3
No	**84**	**67.2**



Graph-5: Percentage Distribution based on type of Ventilatory support required (n=125)

The above table and graph depicts that majority i.e., 84/125, 67.2% did not require any ventilator support. Only 41/125 i.e., 32.8% required ventilator support. Among those who required ventilators majority 29/41, 70.7% required CPAP followed by 9/41, 22.0% required SIMV and remaining 3/41, 7.3% required volume control type of ventilation.

5.2: Objective-wise Table

Table 9: Absolute Mean Difference of blood glucose levels between the capillary POCT value v/s Laboratory value and arterial POCT value v/s Laboratory value

Mean Difference	Mean±SD	t-value (95% CI)	*P*-value	ICC[§] (95% CI)
Paired Difference between the capillary POCT value v/s Laboratory valuea (mg/dL)	1.44±18.67	0.86 (-1.87 to 4.75)	0.39	0.94 (0.92 -0.96)
Paired Difference between the arterial POCT value v/s Laboratory value[b] (mg/dL)	-2.37±16.56	-1.59 (-5.29 to 0.56)	0.11	0.96 (0.94-0.97)

§ - Intraclass Correlation Coefficient; a - Laboratory value minus capillary value; b - Laboratory value minus arterial value

The absolute paired mean difference between the laboratory and capillary POCT values was 1.44±18.67 mg/dL with an ICC of 0.94 [95% CI: 0.92 -0.96] and the absolute paired mean difference between the laboratory and arterial POCT values was -2.37±16.56 mg/dL with an ICC of 0.96 [95% CI: 0.94 -0.97]. The ICC value of > 0.9 indicates excellent reliability.[85]

Table 10: Comparison of capillary and arterial blood glucose levels tested by POCT device v/s arterial blood glucose levels tested by laboratory gold standard method considering ISO 15197:2013 standards

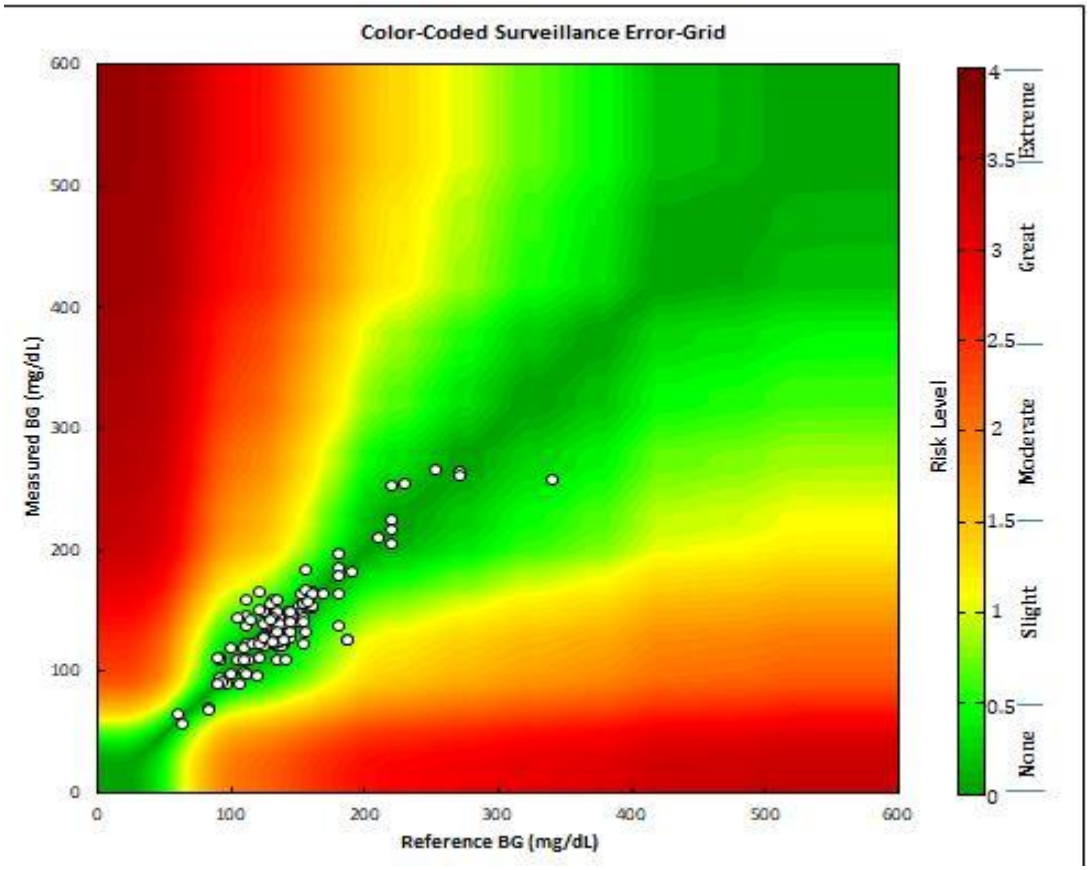
Particulars	No. of samples fitting the ISO standards (<±15mg/dL when POCT < 100 mg/dL & within 15% of reference value when POCT ≥ 100 mg/dL)	Total samples tested	Percentage	Inference
Capillary POCT v/s Lab gold standard method	**100**	125	80.0%	Inaccurate
Arterial POCT v/s Lab gold standard method	**102**	125	81.6%	Inaccurate

The level of agreement between the capillary blood glucose level measured by using the POCT and the arterial blood glucose level measured by standard lab method with respect to the ISO 15197:2013 guideline, only 80.0% were within the ISO 15197 tolerance bands. The capillary blood glucose level measured by using POCT was therefore considered inaccurate, as less than 95% of the values satisfy the criteria of the ISO clinical standards guideline.[80]

The level of agreement between the arterial blood glucose level measured by using the POCT and the arterial blood glucose level measured by standard lab method with respect to the ISO 15197 guideline, only 81.6% were within the ISO 15197 tolerance bands. The arterial blood glucose level measured by using POCT was therefore considered inaccurate, as less than 95% of the values satisfy the criteria of the ISO clinical standards guideline. However capillary POCT is more inaccurate compared to arterial POCT values.

Table 11: Degree of risk for capillary blood glucose levels tested by POCT device v/s arterial blood glucose levels tested by laboratory gold standard method as per the surveillance error grid analysis

Degree of Risk	Absolute Value	Color	No. of Hypo. Pairs (%)	No. of Hyper. Pairs (%)	Total no. of Pairs (%)
None	**0 - 0.5**	**D. Green**	57(45.6)	50(40.0)	112(89.6)
Slight, Lower	**> 0.5 - 1.0**	**L. Green**	8 (6.4)	5(4.0)	13(10.4)
Slight, Higher	**> 1.0 - 1.5**	**Yellow**	0(0.0)	0(0.0)	0(0.0)
Moderate, Lower	**> 1.5 - 2.0**	**L. Orange**	0(0.0)	0(0.0)	0(0.0)
Moderate, Higher	**> 2.0 - 2.5**	**D. Orange**	0(0.0)	0(0.0)	0(0.0)
Great, Lower	**> 2.5 - 3.0**	**L. Red**	0(0.0)	0(0.0)	0(0.0)
Great, Higher	**> 3.0 - 3.5**	**D. Red**	0(0.0)	0(0.0)	0(0.0)
Extreme	**> 3.5**	**Brown**	0(0.0)	0(0.0)	0(0.0)

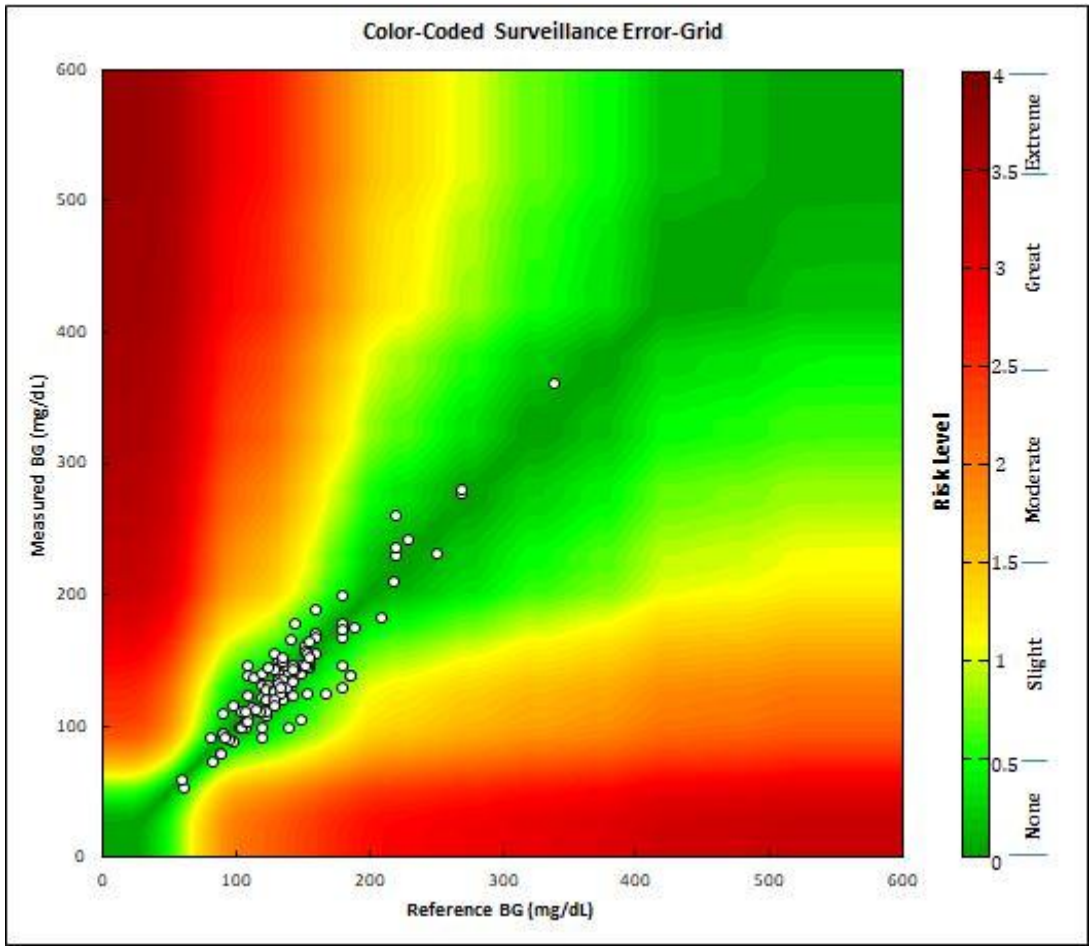


Graph-6: Surveillance error grid zones of capillary blood glucose levels tested by POCT device v/s arterial blood glucose levels tested by laboratory gold standard method

Distribution of the results in Surveillance Error Grid analysis shows that out of 65 hypo pairs, 8 (12.3%) pairs have slight risk of hypoglycemia and out of 55 hyperglycemia pairs, 3 (5.5%) pairs have slight risk of hyperglycemia. Among 125, 112 results have no risk of hypo or hyperglycemia while 13 (10.4%) data pairs are having slight lower risk of hypo or hyperglycemia. The distribution of data pairs suggests that a device with ≤3 % errors outside of the SEG no-risk "green" zone corresponds would meet the ISO requirements of ≤5% data pairs outside the 15 mg/dL, hence indicating that nearly 10.4% data pairs are insufficiently accurate.[82,86,87]

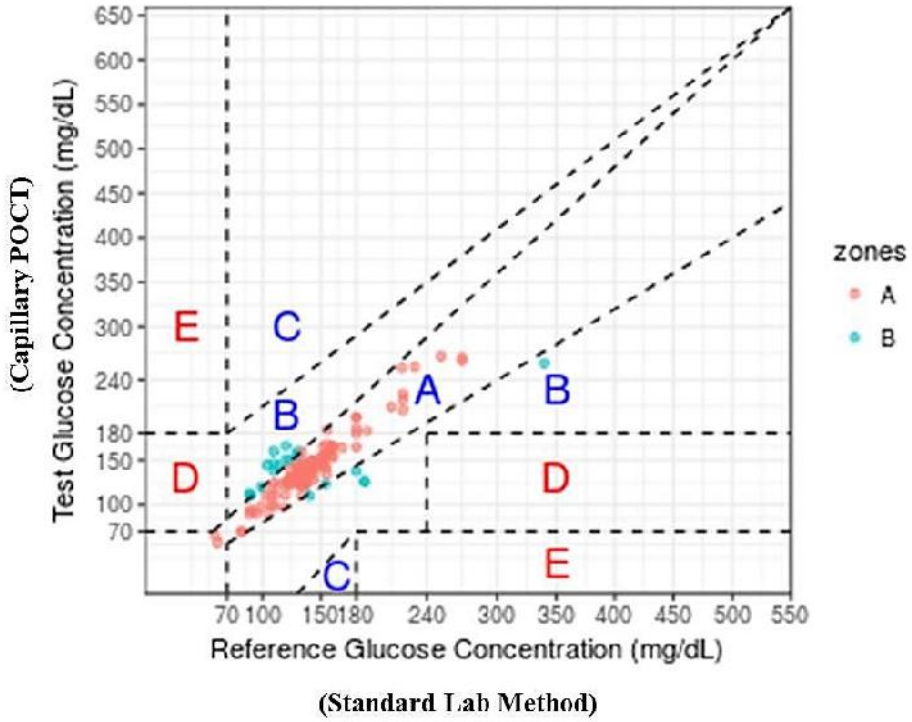
Table 12: Degree of risk for arterial blood glucose levels tested by POCT device v/s arterial blood glucose levels tested by laboratory gold standard method as per the surveillance error grid analysis

Degree of Risk	Absolute Value	Color	No. of Hypo. Pairs (%)	No. of Hyper. Pairs (%)	Total no. of Pairs (%)
None	**0 - 0.5**	**D. Green**	46 (36.8)	71 (56.8)	118 (94.4)
Slight, Lower	**> 0.5 - 1.0**	**L. Green**	3 (2.4)	4 (3.2)	7 (5.6)
Slight, Higher	**> 1.0 - 1.5**	**Yellow**	0 (0.0)	0 (0.0)	0 (0.0)
Moderate, Lower	**> 1.5 - 2.0**	**L. Orange**	0 (0.0)	0 (0.0)	0 (0.0)
Moderate, Higher	**> 2.0 - 2.5**	**D. Orange**	0 (0.0)	0 (0.0)	0 (0.0)
Great, Lower	**> 2.5 - 3.0**	**L. Red**	0 (0.0)	0 (0.0)	0 (0.0)
Great, Higher	**> 3.0 - 3.5**	**D. Red**	0 (0.0)	0 (0.0)	0 (0.0)
Extreme	**> 3.5**	**Brown**	0 (0.0)	0 (0.0)	0 (0.0)



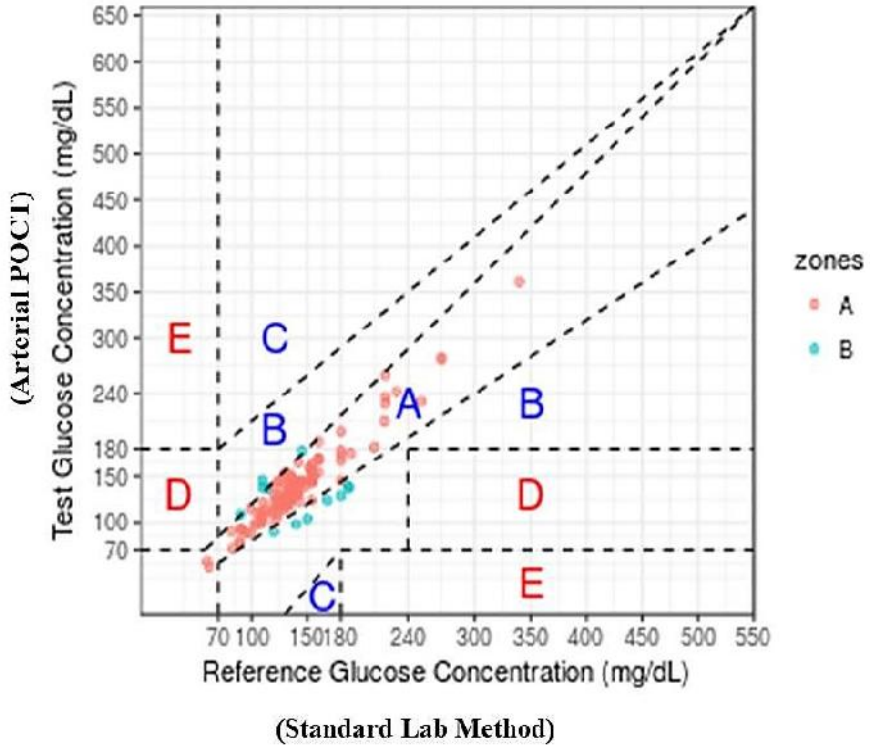
Graph-7: Surveillance error grid zones of arterial blood glucose levels tested by POCT device v/s arterial blood glucose levels tested by laboratory gold standard method

Distribution of the results in Surveillance Error Grid analysis shows that out of 49 hypo pairs, 3 (6.1%) pairs have slight risk of hypoglycemia and out of 75 hyperglycemia pairs, 4 (5.3%) pairs have slight risk of hyperglycemia. Among 125, 118 data pairs have no risk of hypo or hyperglycemia while 7 (5.6%) data pairs are having slight lower risk of hypo or hyperglycemia. The distribution of data pairs suggests that a device with ≤3 % errors outside of the SEG no-risk "green" zone corresponds would meet the ISO requirements of ≤5% data pairs outside the 15 mg/dL, hence indicating that nearly 5.6% data pairs are insufficiently accurate.[82,86,87]



Graph-8: Clarke's Error Grid Plot of capillary POCT v/s Standard Lab Method

The results showed that 84% of the paired capillary POCT and reference values fell within Zones A and remaining 16% fell within zone B indicating that none of the values led to erroneous treatment clinically.



Graph-9: Clarke's Error Grid Plot of arterial POCT v/s Standard Lab Method

The results showed that 90.4% of the paired arterial POCT and reference values fell within Zones A and remaining 9.6% fell within zone B indicating that none of the values led to erroneous treatment clinically.

Table 13: Correlation of Capillary blood glucose v/s Laboratory gold standard method

Blood Glucose estimation techniques	Capillary blood glucose	Laboratory gold standard method
Capillary blood glucose	1.00	
Laboratory gold standard method	0.89*	1.00

*Significant at $P<0.05$

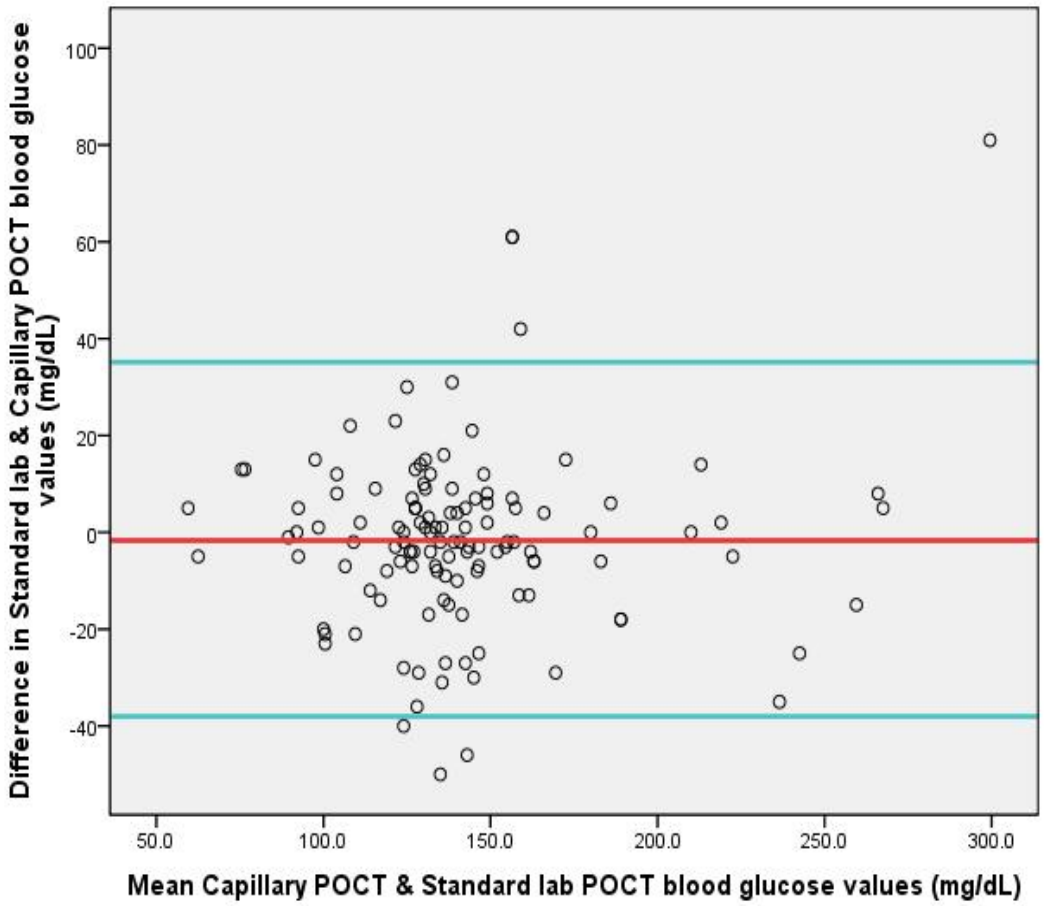
The correlation co-efficient of 0.89 ≈ 0.9 and the *P*-value indicate very high significant positive correlation between the Capillary POCT values and the standard laboratory method *(P<0.05).*[88]

Table 14: Correlation of Arterial blood glucose v/s Laboratory gold standard method

Blood Glucose estimation techniques	Arterial blood glucose	Laboratory gold standard method
Arterial blood glucose	1.00	
Laboratory gold standard method	0.93*	1.00

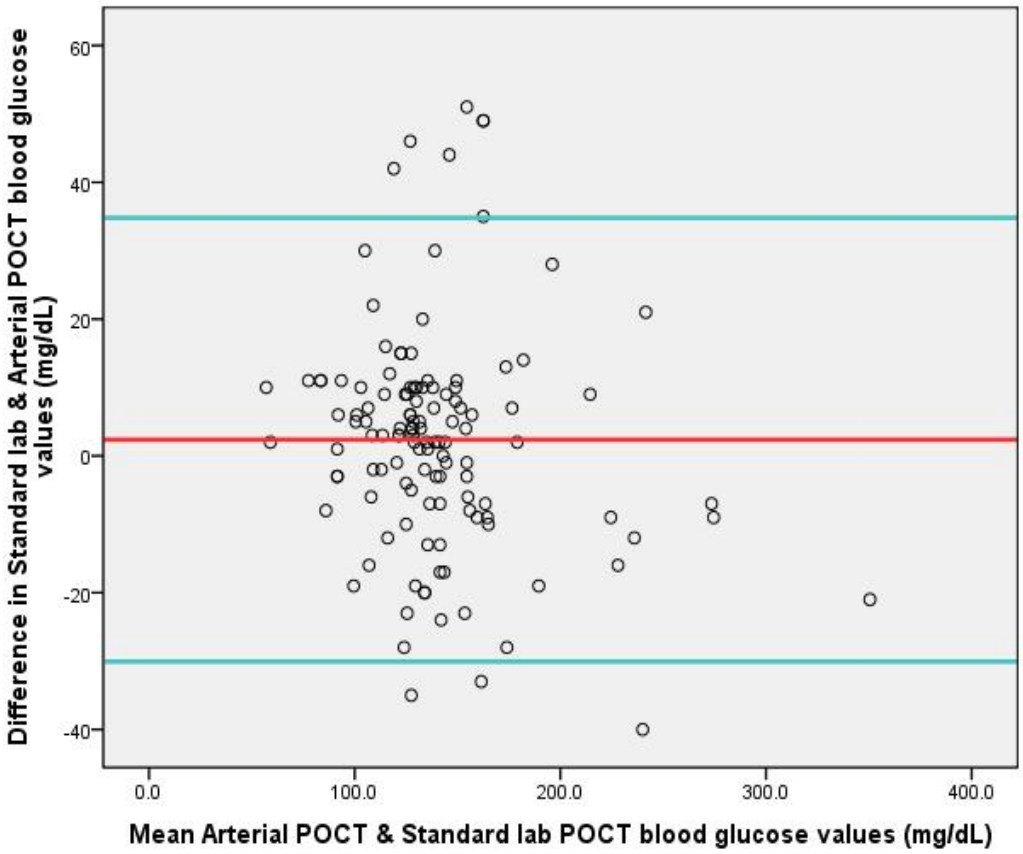
*Significant at $P<0.05$

The correlation co-efficient of 0.93 ≈ 0.9 and the *P*-value indicate very high significant positive correlation between the arterial POCT values and the standard laboratory method *(P<0.05).*[88]



Graph-10: Bland Altman Plot of capillary POCT v/s Standard Lab Method

Bias in the Bland–Altman plot was -1.44 mg/dL with an upper limit of agreement (LOA) of 35.15 mg/dL and a lower limit of agreement of −38.03 mg/dL. The beta unstandardized co-efficient was -0.048 (nearly zero) which was insignificant (P>0.05) indicating that there was no proportional bias and capillary POCT was in-agreement with the standard lab method.



Graph-11: Bland Altman Plot of arterial POCT v/s Standard Lab Method

Bias in the Bland–Altman plot was 2.37 mg/dL with an upper limit of agreement (LOA) of 34.81 mg/dL and a lower limit of agreement of −30.07 mg/dL. The beta unstandardized co-efficient was -0.069 (nearly zero) which was insignificant (P>0.05) indicating that there was no proportional bias and arterial POCT was in-agreement with the standard lab method.

Table 15: Association of various characteristics with accurate and inaccurate Capillary POCT sample values according to ISO 15197:2013 standards

Characteristics	Accurate (Row %)	Inaccurate (Row %)	χ^2 – Value (*P*-value)
Age group			
< 51 years	45 (76.3)	14 (23.7)	0.29
≥ 51 years	53 (80.3)	13 (19.7)	(0.58)
Gender			
Males	47 (73.5)	17 (31.5)	1.91
Females	51 (83.6)	10 (16.4)	(0.17)
Co-morbidities			
Yes	44 (81.5)	10 (18.5)	0.53
No	54 (76.1)	17 (23.9)	(0.47)
Number of Vasopressors used			
One	70 (75.3)	23 (24.7)	2.10
Two	28 (87.5)	04 (12.5)	(0.15)
Type of Vasopressors			
Dopamine	06 (46.2)	07 (53.8)	-
Noradrenaline (NA)	63 (79.7)	16 (20.3)	
Dopamine + NA	18 (85.7)	03 (14.3)	
Vasopressin + NA	11 (91.7)	01 (8.3)	
Requirement of Ventilators			
Yes	31 (75.6)	10 (24.4)	0.28
No	67 (79.8)	17 (20.2)	(0.59)

The proportion of those with inaccuracies were higher in <51 years age group (23.3%), among males (31.5%), among those with no co-morbidities (23.9%), among who used one vasopressor (24.7%) and among those who required ventilators (24.4%). The proportion of inaccuracies was highest among the subjects who were put on Dopamine alone (53.8%) followed by NA alone (20.3%), combination of NA and Dopamine (14.3%) and NA with vasopressin combination (8.3%). However age group, gender, co-morbidities, usage of vasopressors and requirement of ventilators were not statistically associated with the inaccuracy ($P > 0.05$).

Table 16: Association of various characteristics with accurate and inaccurate Arterial POCT sample values according to ISO 15197:2013 standards

Characteristics	Accurate (Row %)	Inaccurate (Row %)	χ^2 – Value (*P*-value)
Age group			
< 51 years	48 (81.4)	11 (18.6)	0.02 (0.88)
≥ 51 years	53 (80.3)	13 (19.7)	
Gender			
Males	54 (84.4)	10 (15.6)	1.08 (0.29)
Females	47 (77.1)	14 (22.9)	
Co-morbidities			
Yes	44 (81.5)	10 (18.5)	0.03 (0.87)
No	57 (80.3)	14 (19.7)	
Number of Vasopressors used			
One	73 (78.5)	20 (21.5)	1.25 (0.27)
Two	28 (87.5)	04 (12.5)	
Type of Vasopressors			
Dopamine	11 (84.6)	02 (15.4)	-
Noradrenaline (NA)	62 (78.5)	17 (21.5)	
Dopamine + NA	18 (85.7)	03 (14.3)	
Vasopressin + NA	10 (83.3)	02 (16.7)	
Requirement of Ventilators			
Yes	33 (80.5)	08 (21.5)	0.004 (0.95)
No	68 (80.9)	16 (19.1)	

The proportion of those with inaccuracies was higher in ≥ 51 years age group (19.7%), among females (22.9%), among those with no co-morbidities (19.7%), among who used one vasopressor (21.5%) and among those required ventilators (21.5%). The proportion of inaccuracies was highest among the subjects who were put on NA alone (21.5%) followed by those who were on vasopressin and NA combination (16.7%), dopamine alone (15.4%), and dopamine and NA (14.3%). However age group, gender, co-morbidities, usage of vasopressors and requirement of ventilators were not statistically associated with the inaccuracy ($P > 0.05$).

Table 17: Comparison of mean of various characteristics with accurate and inaccurate Capillary POCT sample values according to ISO 15197:2013 standards

Characteristics	Mean±SD		*t*-value (95% CI)	*P*-value
	Accurate (n=98)	Inaccurate (n=27)		
Haematocrit (%)	37.85±7.35	41.63±5.89	2.46 (0.74 – 6.82)	0.02*
Total Count (Cells/µL)	14294±6265	13203±6042	- 0.81 (-3766 – 1584)	0.42
Serum Creatinine (mg/dL)	2.24±2.17	1.69±1.4	- 1.24 (-1.42 – 0.32)	0.22

*indicates a significant statistical difference between the groups with $P<0.05$

The inaccurate group had significantly higher haematocrit (41.63±5.89) compared to the accurate group (37.85±7.35) [t = 2.46, $P < 0.05$]. The total count was higher in accurate group (14294±6265) compared to inaccurate group (13203±6042) [t = - 0.81, $P > 0.05$] and similarly serum creatinine also was higher in accurate group (2.24±2.17) compared to inaccurate group (1.69±1.4) [t = - 1.24, $P > 0.05$]. However, total count and serum creatinine were not significantly different in both the groups ($P > 0.05$).

Table 18: Comparison of mean of various characteristics with accurate and inaccurate Arterial POCT sample values according to ISO 15197:2013 standards

Characteristics	Mean±SD		*t*-value (95% CI)	*P*-value
	Accurate (n=101)	Inaccurate (n=24)		
Haematocrit	38.51±7.24	39.29±7.18	0.47 (-2.47 – 4.03)	0.64
Total Count	14120±6232	13800±6241	- 0.23 (-3123 – 2481)	0.82
Serum Creatinine	2.21±2.17	1.73±1.29	- 1.04 (-1.39 – 0.43)	0.30

The inaccurate group had slightly higher haematocrit (39.29±7.18) compared to the accurate group (38.51±7.24) [t =0.47, $P > 0.05$]. The total count was slightly higher in accurate group (14120±6232) compared to inaccurate group (13800±6241) [t = - 0.23, $P > 0.05$] and similarly serum creatinine also was slightly higher in accurate group (2.21±2.17) compared to inaccurate group (1.73±1.29) [t = - 1.04, $P > 0.05$]. However, haematocrit, total count and serum creatinine were not significantly different in both the groups ($P > 0.05$).

DISCUSSION

Hyperglycemia and hypoglycaemia in critically ill patients are associated with an increased risk of mortality and morbidity.[89] They also experience frequent alterations in the glucose metabolism as a result of release of counter regulatory hormones and cytokines leading to peripheral insulin resistance and an enhanced hepatic glucose production as a result of stress. [89,90,91] Achieving glycemic control through optimization of insulin therapy remains challenging. Moreover intensive insulin therapy to achieve glycemic control in hyperglycemia, lead to hypoglycaemia and severe or prolonged hypoglycemia and its complications such as seizures, coma, arrhythmia and irreversible cerebral damage.[92,93] The techniques of glucose monitoring vary across studies and also can affect the results.[15] With a purpose to elicit the accuracies of bedside glucose measuring instruments relative to standard laboratory glucose measurements the present study was taken up.

The mean age of the study participants was 51 years in the current study and similarly Lonjaret *et al*., has reported mean age of 59 years which is slightly higher with an inter-quartile range from 46 – 69 years and similarly in our study it ranges from 40 – 61 years.[13] In the present study, majority i.e., 51% were males and similarly Lonjaret *et al*., in their study also have found same findings wherein 57% were males.[92]

Looking at the pattern of admissions in ICU, respiratory (44.0%) followed by GI illness (18.4%), cardiovascular (5.6%), renal (5.6%), others (Dengue shock syndrome, dyselectrolytemia, poisoning) (5.6%) and CNS (1.6%) were the disease categories of admission. However Ashwini D *et al*., has found that cardiovascular disease (19.9%) followed by respiratory disease (18.0%) as two most common disease

categories of admission[16] and Rajathilagam *et al*., has found cardiovascular system (CVS) (41.53%, n = 54) followed by central nervous system (CNS) (22.3%, n = 29), respiratory system (13.1%, n= 17) and renal system (4.6%, n = 6) affected in their subjects. Infections and sepsis were the admitting diagnosis in 11 patients (8.46%). The remaining patients had other causes like malignancies, GIT diseases and poisoning.[95,96] Similarly, infections and sepsis were also the common reasons of admission in the current study. The differences noted may be due to different study settings.

Among those who had comorbidities existing with T2DM, majority had Chronic Kidney Disease (n=20, 37.0%) followed by Hypertension (n=16, 29.6%), COPD (n=5, 9.2%), hyperthyroidism (n=4, 7.4%), Chronic Liver Disease (n=3, 5.6%), Ischaemic Heart Disease, Peripheral Vascular Disease (n=2, 3.7%) each and Carcinoma Cervix (n=1, 1.9%). As all were diabetics, diabetes itself leads to a higher incidence of nearly all comorbidities including renal, cardiovascular, and neuropathic disease and similarly, chronic kidney disease was the elicited comorbidity.[18] Pantalone KM *et al*., have found hypertension and cardiovascular disease as common comorbidities along with T2DM in a similar study [98]

Juneja *et al*., has reported that all the patients in the study group were on noradrenaline and few were on more than one vasopressor, similarly in our study majority i.e., 90.0% were put on noradrenaline and 25% were on double vasopressors.[84]

Sonawane P *et al*., reported that majority of their subjects in ICU were put on NA (45/54, 83.3%) followed by dopamine (42/54, 77.8%), dopamine + NA combination (33/54, 61.1%), dobutamine (5/54, 9.3%), dobutamine + NA combination (3/54, 5.6%). In the current study, NA alone was used among most of

them i.e., 63.2% followed by 16.8% used a combination of Dopamine with Noradrenaline, 10.4% used Dopamine alone and 9.6% used a combination of Noradrenaline and Vasopressin. However the proportions cannot be compared as the percentage in the compared study is calculated individually for each and on addition the total percentage is more than 100.0% as the same drug is considered multiple times as single and even in combinations.[99]

Ellis MF *et al*., reported that the mean number of vasopressor medications (dopamine, epinephrine, vasopressin, norepinephrine, or phenylephrine) was 3.2 ± 1.0 and in the current study the mean number of medications used (NA, Dopamine, Vasopressin) was 1.26 ± 0.44.[21] The difference may be due to different study subjects admitted with different pattern of disease.

The mean values of Capillary blood glucose and arterial blood glucose recorded using the POCT device (glucometer) were 142.61±38.96 mg/dL and 138.80±43.58 mg/dL respectively. Similarly, Lonjaret *et al*., has reported the mean Capillary and arterial blood glucose values as 126±52 mg/dL and 133±50 mg/dL respectively. The mean level of arterial blood glucose levels recorded in our lab was 141.17±40.77 mg/dL and Lonjaret *et al*., 143±54 mg/dL which are nearly comparable except for capillary values which seems to be slightly higher compared to the study conducted by Lonjaret *et al*., and the standard lab values are slightly higher compared to the values tested by the POCT similar to the current study.[92]

The mean Haematocrit value was 38.66±7.21% in the current study and mean values of hematocrit in the study by Lacara T *et al*., was 31.7±0.8% among critically ill patients which is nearly similar to the current study.[22] Paary TTS *et al*., have reported mean total leucocyte count as 15151 ± 7798/µL and mean serum creatinine as 2.48 ± 1.97 mg/dL and are in line with the current study findings wherein, the

mean total count was 14059±6210 cells/µL and mean serum creatinine was 2.13±2.04 mg/dL.[23] However the compared study is not specific to diabetics but majority of them had diabetes as the comorbidity.

Majority did not require any ventilator support in the current study indicating the non-impaired ventilatory mechanism, however in a study by Garingarao CJ *et al*., majority of the hypotensive group required ventilatory support which may be due to inclusion of subjects with other types of shock.[103]

The absolute mean difference between the laboratory and capillary values were 16±22 mg/dL with an ICC of 0.91 [95% CI: 0.89 - 0.93] and between the laboratory and arterial values was 10±21 mg/dL with an ICC of 0.92 [95% CI: 0.90 - 0.94] according to Lonjaret *et al*. However in the present study, absolute mean difference between the laboratory and capillary values was -1.44±18.67 mg/dL with an ICC of 0.94 [95% CI: 0.92 - 0.96] and the absolute mean difference between the laboratory and arterial values was 2.37±16.56 mg/dL with an ICC of 0.96 [95% CI: 0.94 -0.97]. Though the difference between the two methods are lesser compared to the findings by Lonjaret *et al*., the ICC value indicates excellent reliability in the present study also.[85,92]

In this study, the level of agreement between the capillary blood glucose level measured and the arterial blood glucose level measured by standard lab method with respect to the ISO 15197 guideline 2003 was considered inaccurate as only 84.0% were within the ISO 15197 tolerance bands which is in concordance to the findings of Ellis MF et al , where 88.3% were within the ISO 15197 (2003) tolerance bands indicating inaccuracy of capillary method of measurement. The level of agreement between the arterial blood glucose level and the blood glucose level measured by standard lab method with respect to the ISO 15197 guidelines (2003), only 90.4%

were within the ISO 15197 tolerance bands which is in concordance to the findings of Ellis MF et al., where 94.4% were within the ISO 15197 (2003) tolerance bands.

In this study, the level of agreement between the capillary blood glucose level and the arterial blood glucose level measured by standard lab method with respect to the ISO 15197 guideline 2013 was considered inaccurate as only 80.0% were within the ISO 15197 tolerance bands. The level of agreement between the arterial blood glucose level and the blood glucose level measured by standard lab method with respect to the ISO 15197 guidelines (2013), only 81.6% were within the ISO 15197 tolerance bands.

The proportion of subjects within the tolerance bands for both capillary and arterial blood glucose measurements were slightly lower in comparison to the study by Ellis MF et al was because of the updated guidelines of 2013 which includes a margin of 15% compared to margin of 20% in 2003.24 However, when compared to 2003 ISO guidelines, the proportion of those within the tolerance bands were in concordance with the findings by Ellis MF et al.24 This indicates that capillary POCT values of blood glucose are more inaccurate compared to arterial POCT.

In the current study, Surveillance Error Grid analysis in capillary blood glucose measurement showed 10.4% data pairs are having slight lower risk of hypo or hyperglycemia and in arterial blood glucose measurement, 5.6% data pairs are having slight lower risk of hypo or hyperglycemia. Ancona P *et al*., in their study to compare flash glucose monitoring and capillary and arterial blood glucose measurements found that, 24.7% in capillary group and 16.2% in arterial group were having data pairs in slight lower risk of hypo or hyperglycemia.[25] According to Rojekar MV *et al*., Surveillance Error Grid analysis (SEG) showed that 20% had very negligible risk on analyzing plasma samples of paediatric subjects in ICU settings by comparing

glucometers with laboratory findings.[7] Because of the paucity of literature in comparing the accuracy of POCT vs standard lab methods among adults in ICU settings, the results of SEG in our study has been compared with the studies with different glucose monitoring techniques, study settings and subjects.

In our study, according to Clarke's error grid analysis, 84% of the paired capillary POCT with respect to standard lab values were within Zone A and remaining 16% were within zone B indicating that none of the values led to erroneous treatment clinically, which is in concordance to the findings of Ellis MF et al, where 88.3% of paired capillary POCT values were within zone A.

Similarly, 90.4% of the paired arterial POCT with respect to standard lab values were within Zone A and remaining 9.6% were within zone B indicating that none of the values led to erroneous treatment clinically, which is in concordance to the findings of Ellis MF et al , where 94.4% of paired arterial POCT values were within zone A.

Correlation of Capillary and arterial blood glucose v/s Laboratory gold standard method showed a positive correlation with correlation co-efficient of 0.89 and 0.93 respectively which is comparable to the study findings of Petersen JR *et al*., who also found correlation co-efficient of r=0.87-0.89 and r= 0.97-0.99 for capillary and arterial blood glucose measurements indicating capillary sampling to have a significantly lower correlations to laboratory values compared to arterial methods in ICU settings.[26] Boyd R *et al*., in their study also found positive correlation of 0.97 for capillary blood glucose monitoring.[27] According to Li X *et al*., in their study, Correlation between capillary values measured by glucometer and arterial values measured by blood gas analyzer was also less in the study group (r = 0.897, P = 0.001

and $r = 0.964$, $P < 0.001$).[28] Majority of the studies conclude that Capillary blood glucose monitoring is reliable only in a selected group of ICU patients.

In the current study, Bland–Altman plot showed a mean bias of -1.44 mg/dL (LOA: 35.15, −38.03 mg/dL) in capillary POCT method whereas in arterial POCT method, mean bias was 2.37 mg/dL (LOA: 34.81 mg/dL, −30.07 mg/dL). The beta unstandardized co-efficient was -0.048 and – 0.069 (P>0.05) in capillary and arterial groups indicating no significant proportional bias with the standard lab method .In a study by Lacara T *et al*, mean bias +/- precision and root-mean-square differences were 2.1 +/- 12.3 and 12.35, respectively, for fingerstick blood and 0.6 +/- 10.6 and 10.46 for catheter blood, concluding that the values for point-of-care and laboratory tests did not differ significantly which is comparable to this study. [22] Similarly, Garingarao CJ *et al*., found that in hypotensive groups who were on vasopressors, the mean bias was –34.9 mg/dL (LOA: –207.1, 137.4 mg/dL) with lower mean POC glucose compared to venous glucose values inferring hypotensive critically ill patients on vasopressor support, POC glucose meter values had lower accuracy in terms of ISO 2003:15197 criteria and Bland–Altman agreement analysis.[24] Salacinski AJ *et al*, compared glucometer with a standard reference instrument for analyzing blood glucose, wherein on regression analysis the fixed bias was −7.148 mg/dL, an underestimation of plasma glucose indicating a significant (+13%) proportional bias as indicated by the slope of the regression line being greater than 1.0 suggesting that the glucometer provided poor validity and reliability results compared to the results provided by the reference laboratory analyzer.[108]

In the Capillary POCT sample, the proportion of those with inaccuracies were higher in <51 years age group (51.9%) and among males (63.0%) whereas in arterial POCT sample, proportion of inaccuracies were higher in ≥ 51 years age group

(54.2%), among females (58.3%) which is comparable to the findings of Lonjaret L *et al*., who noted age and gender had no influence on any differences between methods..[13]

The number of vasopressors did not influence the accuracy of the POCT results as the degree of vasoconstriction does not depend on the number of vasopressors used, as it just optimizes the required mean arterial pressures in shock states.[109]

In this study, the proportion of inaccuracies was highest among the subjects who were put on Dopamine alone in capillary samples and NA alone in arterial samples. Kotwal N and Pandit A has mentioned that dopamine can increase glucose values on GDH-based meters, primarily at high drug concentrations in the capillary samples which might have been indicated in our study too in the capillary samples.[110]

According to Lonjaret L *et al,* who studied various factors contributing to inaccuracy found that Norepinephrine as a predictive factor for inaccuracy for arterial samples.[92]

In the current study, both capillary and arterial blood glucose measurements, showed that inaccurate group had higher haematocrit compared to the accurate group. In a study by Lacara T *et al*., noted that haematocrit values were a significant contributors to difference scores between the laboratory and the catheter POC methods ($F3,45 = 8.17$, $P < .001$).[101]

LIMITATIONS

Our study is limited by the fact that there is a lack of control group i.e., without vasopressors as the role of vasopressor on blood glucose levels could better be established in the presence of a control group. The study lacks the generalisability as it was a purposive sampling. Some of the naturally occurring substances in the body viz., High triglyceride levels, bilirubin, drugs – Dopamine can act as confounders as they tend to interfere in the blood glucose readings which needs to be addressed. High triglyceride levels cause falsely low blood glucose values as they tend to take up volume reducing the glucose levels. Also, bilirubin has been noted to cause pseudo hypoglycemia. Some drugs also affect the capillary glucose readings - Dopamine increased glucose values on GDH-based meters, primarily at high drug concentrations.[110]

CONCLUSION

- Arterial point of care testing values had slightly better agreement in comparison to capillary point of care testing which could be deemed negligible.
- Both the values were inaccurate according to ISO standards although they had reasonably good correlation with standard lab values.
- According to surveillance error grid analysis none of the data pairs featured outside the green zone (none- slight lower risk) which suggests that there was no significant change in diabetic management for any pair.
- We used Clarke's error grid analysis, which is a tool to evaluate and compare the accuracy of a testing device with a laboratory reference for our study to look for agreement of both capillary and arterial POCT values with respect to standard lab values and found that all values were within zone A and zone B which suggests that there was benign or no treatment error. Thus reinstating that the source of blood nor mode of blood glucose estimation altered clinical management
- Most studies conducted so far have shown and recommended arterial blood glucose estimation at the bedside over capillary point of care testing among critically ill patients. As per our study we choose to conclude that there is no significant variation between arterial and capillary point of care testing for blood glucose estimation among critically ill patients on vasopressor support. Considering arterial as a more invasive method, capillary method of glucose estimation stands as a reasonable and easy option for bedside glucose monitoring of critically ill patients on vasopressor support.
- Our study also suggests that, number of vasopressors did not influence the accuracy of point of care testing results.

RECOMMENDATIONS:

Based on the conclusions and limitations we would recommend on the following:

- Conducting additional studies, with larger samples and among different clinical statuses of critically ill patients'.

- To assess the clinical outcome of blood glucose monitoring from various blood sources who were on their respective insulin protocols.

SUMMARY

Hyperglycemia and hypoglycaemia in critically ill patients are associated with an increased risk of mortality and morbidity. Achieving glycemic control through optimization of insulin therapy remains challenging. Moreover intensive insulin therapy to achieve glycemic control in hyperglycemia, often leads to severe or prolonged hypoglycemia and its complications such as seizures, coma, arrhythmia and irreversible cerebral damage. This warrants frequent bedside gulcose monitoring. The techniques of glucose monitoring vary across studies and can also affect the results. With a purpose to elicit the accuracies of bedside glucose measurement relative to standard laboratory glucose measurements the present study was taken up.

Through our study we compared the accuracy of capillary blood glucose estimation and arterial point of care glucose estimation with standard laboratory blood glucose estimation among critically ill patients admitted in ICU on vasopressor support

Arterial point of care testing values had slightly better agreement in comparison to capillary point of care testing which could be deemed negligible. Both the values were inaccurate according to ISO standards although they had reasonably good correlation with standard lab values.

According to surveillance error grid analysis none of the data pairs featured outside the green zone (none- slight lower risk) and similarly in Clarke's error grid analysis none of the values featured outside of zones A,B which suggests that there was no significant change in diabetic management.

Most studies conducted so far have shown and recommended arterial blood glucose estimation at the bedside over capillary point of care testing among critically ill patients. As per our study we choose to conclude that there is no significant variation between arterial and capillary point of care testing for blood glucose estimation among critically ill patients on vasopressor support.

Considering arterial as a more invasive method, capillary method of glucose estimation stands as a reasonable and easy option for bedside glucose monitoring of critically ill patients on vasopressor support.

Our study also suggests that, number of vasopressors did not influence the accuracy of point of care testing results.

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ANNEXURE I – CONSENT FORM

TITLE OF RESEARCH STUDY: "COMPARISON OF CAPILLARY BLOOD GLUCOSE VERSUS ARTERIAL BLOOD GLUCOSE IN DIABETIC PATIENTS ADMITTED IN ICU ON VASOPRESSOR SUPPORT – A ONE YEAR CROSS SECTIONAL OBSERVATIONAL STUDY"

Objective and purpose of the study

This research is intended to compare the accuracy of capillary blood glucose with arterial blood glucose in diabetic patients admitted in the ICU on vasopressor support. The principal investigator of the study is Dr._____ under the guidance of Dr. _____.

Need For Study

Glycemic control in critically ill patients decreases infection and mortality. Patients receiving vasopressors have altered peripheral perfusion, which may affect accuracy of capillary blood glucose values measured with point-of-care devices.

We aimed to compare the accuracy of capillary bedside glucometry with arterial samples in critically ill patients with shock

Procedure

If you agree to be part of the research study you will be asked history and will be subjected to clinical examination and investigations.

Risk and Benefits

There is as if no risk involved in the study except from the only risk and possible discomfort you might get is while taking capillary blood from the finger tip for bed side glucose estimation, which is a routine procedure done for all diabetic/ICU

patients. It may cause swelling, pain, redness, bruising or infection (rarely happens) at the site from where the blood is drawn

Alternatives

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part you can later change my mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsored may stop your participation in this study any time. If you choose not to take part in the study you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy

In the event of injury, related to the study, treatment will be made available at KLES Dr. Prabhakar Kore Hospital and Medical Research Center, Belgaum. There is no compensation or payment for such medical treatment by law.

Voluntary participation / withdrawal

Your participation in this study is entirely voluntary and you may withdraw from the study at any time.

Financial incentives for participation

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

If you have any questions about your rights as a participant and regarding the study you may call

DR. _____
Investigator,
PG in General Medicine,
Jawaharlal Nehru Medical College,
Belgaum - 590 010
Ph. No.: 9743759910

DR. _____
Associate Professor,
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DR. GANGA PILLI,
Chairman,
J.N.M.C., Ethical Committee for Human Research,
Professor and Head Department of Pathology,
Jawaharlal Nehru Medical College,
Belgaum – 590 010
Ph. No. 0831-2471350

CONSENT FORM

I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this entire consent form or it has been read to me, and has been explained to me in my own vernacular language and has all my questions answered. I will be given a copy of this consent form.

Signature / Left Thumb print of the Participant or legally authorized representative.

Participant's Name : …….

Signature / Left Thumb

Impression of the participant's : …….

Name of the legally

Authorized representative/ Guardian : …….

Signature/ Left Thumb Impression : …….

Witness's Name : …….

Signature/ Left Thumb Impression. : …….

Investigators Name and Signature : ……

Date:

Place:

ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಗಾಗಿನ ಸಮ್ಮತಿ ಪತ್ರ

ನಾವು ನಿಮ್ಮನ್ನು ಸಂಶೋಧನೆಯಲ್ಲಿ ತೊಡಗಿಸಿಕೊಳ್ಳಲು ವಿನಂತಿಸುತ್ತಿದ್ದೇವೆ **ಕಂಪಾರೀಸನ್ ಆಫ್ ಕಂಪೀಲಿಯರ್ ಬ್ಲಡ್ ಗ್ಲೋಕೋಸ್ ಇನ್ ಡೈಬೀಟಿಕ್ ಪೆಸೆಂಟ್ಸ್ ಅಡ್ಮಿಟೇಡ್ ಇನ ಆಯ್.ಸಿ.ಯು. ಆನ್ ವಾಸೋಪ್ರೇಸರ್ ಸಪೋರ್ಟ್ ಆಟ್ ಕೆ.ಎಲ್.ಇ.ಎಸ್. ಡಾ॥ ಪ್ರಭಾಕರ ಕೋರೆ ಹಾಸ್ಪೀಟಲ್ ಮತ್ತು ಎಮ್.ಆರ್.ಸಿ. ಬೆಳಗಾವಿಯಲ್ಲಿ ಮಾಡುವ ಒಂದು ಕ್ರಾಸ್ ಸೆಕ್ಷನಲ್ ಅಧ್ಯಯನ" ಡಾ॥ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ ಜೆ.ಎನ್.ಮೆಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ ಇವರು ಡಾ॥ ಪ್ರೊಫೇಸರ, ವೈದ್ಯಕೀಯ ವಿಭಾಗ, ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ,** ಇವರ ಮಾರ್ಗದರ್ಶದಲ್ಲಿ ನಡೆಸುತ್ತಿದ್ದೇವೆ.

ಗೌರವಾನ್ವಿತರೇ ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಅರ್ಹರಿದ್ದಿರಿ.

ಸಂಶೋಧನೆಯಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆ ವೈಯಕ್ತಿಕವಾಗಿದ್ದು, ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ನಿಮ್ಮ ನಿರ್ಧಾರವಾಗಿರುತ್ತದೆ. ಇದರಿಂದ ಜೆ.ಎನ್ ಮೇಡಿಕಲ್ ಕಾಲೇಜಿಗೆ ನಿಮಗಿರುವ ಸಂಬಂಧಕ್ಕೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು.

ನೀವು ನನ್ನ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮನ್ನು ತೊಡಗಿಸಿಕೊಳ್ಳಲು ಒಪ್ಪಿದ ನಂತರ ಪ್ರಾಯೋಗಿಕವಾಗಿ ವಿಚಾರಣೆ ಹಾಗೂ ಅದಕ್ಕೆ ತಕ್ಕಂತೆ ತನಿಖೆ ನಡೆಸಲಾಗುವುದು. ನಿಮ್ಮ ಹಾಗೂ ಕುಟುಂಬದ ಇತಿಹಾಸದ ಬಗ್ಗೆ ಸಂಧರ್ಶನ ನಡೆಯಿಸಲಾಗುವುದು.

ತಾವುಗಳು ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಾಗ ಮುನ್ನೆಚ್ಚರಿಕೆ ಕ್ರಮಗಳನ್ನು ಅನುಸರಿಸಲಾಗುವುದು. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದರಿಂದ ಖಾಯಿಲೆಯ ಸಂಪೂರ್ಣ ಮಾಹಿತಿ ಪಡೆಯಬಹುದು. ಇದರಿಂದ ಇದೇ ಖಾಯಿಲೆಯಿಂದ ಬಳಲುತ್ತಿರುವ ಇತರರಿಗೆ ತುಂಬಾ ಸಹಾಯವಾಗುವುದು.

ಈ ಪರೀಕ್ಷೆಯ ವೆಚ್ಚವನ್ನು ಆಸ್ಪತ್ರೆಯ ನಿಯಮದಂತೆ ತಾವೇ ಭರಿಸಬೇಕು. ಆದರೆ ಇದರಲ್ಲಿ ಯಾವುದೇ ಇತರ ವೆಚ್ಚುಗಳು ಇರುವುದಿಲ್ಲ.

ತಾವು ಈ ಪರೀಕ್ಷೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಗೌಪ್ಯವಾಗಿ ಇಡಲಾಗುವುದು.

ತಮ್ಮ ಖಾಯಿಲೆ ಹಾಗೂ ಸಂಶೋಧನೆಯ ವಿವರಗಳನ್ನು ಎಲ್ಲಿಯೂ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಸಂಶೋಧನೆಯ ವಿವರಗಳನ್ನು ಪ್ರಕಟಿಸಲಿಕ್ಕೆ ಉಪಯೋಗ ಮಾಡಬಹುದು. ಆದರೆ ಇಲ್ಲಿ ನಿಮ್ಮ ಗೌಪ್ಯತೆಯನ್ನು ಕಾಪಾಡಲಾಗುವುದು. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ಆಗುವ ಯಾವುದೇ ವ್ಯತಿರಿಕ್ತ ಪರಿಣಾಗಳಿಗೆ ಕೆ.ಎಲ್.ಇ.ಆಸ್ಪತ್ರೆಯ ಮೂಲಕ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುವುದು.

ತಮಗೆ ಯಾವುದಾದರೂ ಸಂಶಯಗಳಿದ್ದಲ್ಲಿ ಅಥವಾ ಹೆಚ್ಚಿನ ಮಾಹಿತಿ ಬೇಕಾಗಿದ್ದಲ್ಲಿ ಈ ಕೆಳಗಿನ ವೈದ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು.

(1) ಡಾ॥ . **ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ** ವೈದ್ಯಕೀಯ ವಿಭಾಗ, ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ.

(2) ಡಾ॥ **ಪ್ರೊಫೇಸರ, ವೈದ್ಯಕೀಯ ವಿಭಾಗ,** ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವವರ ಹಕ್ಕುಗಳ ವಿವರಗಳಿಗಾಗಿ ಈ ಕೆಳಗಿನ ವೈದ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು.

ಡಾ॥ ಗಂಗಾ ಎಸ್. ಪಿಳ್ಳೆ, ಚೇರಮನ್, ಇನಸ್ಟಿಟ್ಯೂಶನಲ್ ಎಥಿಕ್ಸ್ ಕಮಿಟಿ, ಪ್ರೊಫೇಸರ, ಪೆಥಾಲಾಜಿ ವಿಭಾಗ, ಜೆ.ಎನ್.ಮೇಡಿಕಲ್ ಕಾಲೇಜ, ಬೆಳಗಾವಿ. (ಮೋ) 9480275601.

ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಸ್ವ–ಒಪ್ಪಿಗೆ ಪ್ರಮಾಣ ಪತ್ರ :

ಈ ಸಂಶೋಧನೆಯ ಬಗ್ಗೆ ನನ್ನ ಸ್ವಂತ ಭಾಷೆಯಲ್ಲಿ ಸಂಪೂರ್ಣ ವಿವರವಾಗಿ ನನಗೆ ಅರ್ಥವಾಗಿರುತ್ತದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನನಗೆ ಸಂಪೂರ್ಣವಾದ ಒಪ್ಪಿಗೆ ಇರುತ್ತದೆ. ಈ ಸಂಶೋಧನೆಯ ವಿವರಗಳು ಹಾಗೂ ಪರಿಣಾಮಗಳ ಬಗ್ಗೆ ನನಗೆ ಸಂಪೂರ್ಣವಾದ ಮಾಹಿತಿ ಇರುತ್ತದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಸ್ವ ಇಚ್ಚೆಯಿಂದ ಪಾಲ್ಗೊಳ್ಳಲು ಬಯಸುತ್ತೇನೆಂದು ಈ ಮೂಲಕ ದೃಢೀಕರಿಸುತ್ತೇನೆ. ನಾನು ಈ ಸಮ್ಮತಿ ಪತ್ರಕ್ಕೆ ಸಹಿ ಮಾಡುವುದರಿಂದ ನನಗೆ ಲಭ್ಯವಿರುವ ಕಾನೂನಿನ ಯಾವುದೇ ಹಕ್ಕುಗಳನ್ನು ಬಿಟ್ಟುಕೊಟ್ಟಿರುವುದಿಲ್ಲ. ನಾನು ಮೇಲಿನ ವಿಷಯ ಓದಿ ಅಥವಾ ಓದಿಸಿ ಕೇಳಿ ಸಮ್ಮತಿ ಪತ್ರದಲ್ಲಿರುವ ಎಲ್ಲ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸಿರುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು : _____

ಭಾಗವಹಿಸುವವರ ಸಹಿ : _____

ಭಾಗವಹಿಸುವವರ ಹೆಬ್ಬಟ್ಟಿನ ಗುರುತು : _____

ಸಾಕ್ಷಿದಾರರ ಹೆಸರು : _____

ಸಾಕ್ಷಿದಾರರ ಸಹಿ : _____

ಸಂಶೋಧಕರ ಹೆಸರು : _____

ಸಂಶೋಧಕರ ಸಹಿ : _____

ಸ್ಥಳ : _____

ದಿನಾಂಕ : _____

ANNEXURE II – PROFORMA

TITLE: "COMPARISON OF CAPILLARY BLOOD GLUCOSE VERSUS ARTERIAL BLOOD GLUCOSE IN DIABETIC PATIENTS ADMITTED IN ICU ON VASOPRESSOR SUPPORT – A ONE YEAR CROSS SECTIONAL OBSERVATIONAL STUDY"

PROFORMA

NAME:

AGE/SEX:

IP No.

ADDRESS:

COMPLAINTS AT PRESENTATION:

COMORBIDITIES:

RELEVANT CLINICAL FINDINGS:

VITALS:
TEMPERATURE: ^{0}F
PULSE RATE: /MIN
RESPIRATORY RATE: /MIN
BLOOD PRESSURE: / mm Hg MEAN ARTERIAL PRESSURE:
SPO2

VASOPRESSOR ON FLOW	NOS.	DURATION
INFUSION RATE		

INVESTIGATIONS
Hemoglobin, g/Dl
PCV(Hematocrit)
Total count
Blood urea nitrogen : mg/dL
Serum Creatinine : mg/dL
LFT :
SERUM ALBUMIN:
FBS PPBS RBS HbA1C
OTHERS RELEVANT-

NEED FOR ICU ADMISSION/CAUSE OF SEPSIS –

CAPILLARY BLOOD GLUCOSE
ARTERIAL BLOOD GLUCOSE

ANNEXURE III – KEY TO MASTER CHART

AKI	-	Acute kidney injury
ARDS	-	Acute respiratory distress syndrome
B/L	-	Bilateral
Ca Cx	-	Carcinoma cervix
CEG	-	Clarke's error grid analysis
CKD	-	Chronic kidney disease
CLD	-	Chronic liver disease
COPD	-	Chronic obstructive pulmonary disease
CPAP	-	Continuous positive airway pressure
Cr	-	Creatinine
Dopa	-	Dopamine
ESRD	-	End stage Renal disease
GE	-	Gastroenteritis
GI	-	Gastrointestinal
HOCM	-	Hypertrophic obstructive cardiomyopathy
HTN	-	Hypertension

HTN	-	Hypertension
IHD	-	Ischaemic heart disease
ILD	-	Interstitial lung disease
LRTI	-	Lower respiratory tract infection
mg/dL	-	Milligrams per deciliter
mm Hg	-	Millimeters of mercury
MODS	-	Multiorgan dysfunction syndrome
Norad	-	Noradrenaline
Norad	-	Noradrenaline
PVD	-	Peripheral Vascular disease
SBP	-	Subacute bacterial peritonitis
SIMV	-	Synchronised intermittent mechanical ventilation
T2DM	-	Type 2 diabetes mellitus
TB	-	Tuberculosis
TC	-	Total count
UTI	-	Urinary tract infections

This document was exported from Numbers. Each table was converte
objects on each Numbers sheet were placed on separate worksheets.
calculations may differ in Excel.

Numbers Sheet Name	Numbers Table Name
Sheet1	
	Table 1
Sheet2	
	Table 1
Sheet3	
	Table 1

ed to an Excel worksheet. All other
Please be aware that formula

Excel Worksheet Name

Sheet1

Sheet2

Sheet3

Serial no.	Patient no.	Sex	Age	Diagnosis	Co-morbidities	Vasopressors		Sugars			Ventilatory support	Lab Investigati	
						Number	**Type**	**Capillary**	**Lab Arterial**	**Arterial**		Hematocrit	TC
1	825607	M	56	Adenocarcinoma of bronchus with metastasis	Nil	1	Norad	218	220	229	Nil	41	10.4
2	832581	F	28	Amitraz Poisoning	Nil	2	Norad, Dopa	121	134	126	Nil	30	15
3	867543	F	75	B/L Pneumonia, Septecemia, ARDS	COPD,HTN	1	Norad	259	340	361	CPAP	34	26.8
4	829697	F	66	Left Lower Lobe Pneumona	Nil	1	Norad	146	152	158	CPAP	48	11.3
5	830376	M	60	LRTI	HTN	1	Norad	166	160	169	Nil	45	12.7
6	854698	F	35	B/L Pneumonia	Nil	1	Norad	265	270	277	Nil	38	12.3
7	829551	M	48	Viral Fever, Acute GE, MODS	Nil	2	Norad, Vasopressin	141	132	122	Nil	50	11
8	829350	F	61	B/L Pneumonia,	T2DM, CKD	1	Norad	145	141	130	Nil	33	16.8
9	829087	M	40	ILD	Nil	1	Norad	105	110	116	Nil	44	8.5
10	828365	M	22	Upper GI Bleed Secondary to Portal HTN	Nil	1	Norad	110	133	150	Nil	29	14
11	829122	M	57	Sepsis with MODS	T2DM,HTN	1	Norad	267	252	231	Nil	48	17
12	830755	M	50	B/L Pneumonia	T2DM	1	Norad	262	270	279	Nil	44	14.4
13	830340	M	40	Tb Pleural Effusion	IHD	1	Dopa	104	110	122	Nil	45	6.9
14	824600	F	45	Peritonitis	Ca Cervix	1	Norad	153	160	170	Nil	31	13.1
15	825607	M	56	Ca Bronchus	Nil	1	Norad	206	220	236	SIMV	46	10.4
16	829121	M	55	Acute exacerbation of COPD	Nil	1	Norad	104	110	120	CPAP	48	5
17	827928	F	31	HOCM	Nil	1	Dopa	124	123	107	Nil	44	18.1
18	828387	M	35	Acute GE, AKI	Nil	1	Norad	255	230	242	Nil	30	7.2
19	827010	M	50	ALD, Portal HTN, SBP	Nil	1	Norad	112	120	130	CPAP	35	27
20	828010	F	45	Sepsis	Nil	2	Dopa, Norad	57	62	52	Nil	30	15
21	828041	F	65	Diselectrolytemia	CKD	2	Norad, Dopa	136	140	143	Nil	27	13.7
22	828022	M	54	UGI Bleed secondary to Warfarin Overdose	PVD	1	Norad	134	130	124	SIMV	35	35
23	828342	F	64	Acute exacerbation of COPD	Nil	1	Norad	225	220	260	CPAP	39	11.9
24	827824	F	44	Subendocardial Ischaemia	Nil	2	Norad,vaso	142	140	138	Volume control	35	6.8
25	827927	F	31	Pyelonephritis	Nil	2	Norad,Dopa	99	90	84	CPAP	43	22
26	837546	M	59	CLD,ARF	T2DM	1	Dopa	132	130	121	CPAP	34	8
27	827888	M	48	Left hemiparesis secondary to IC bleed	Nil	1	Dopa	100	99	88	Nil	42	13
28	826607	M	73	Right lobar Klebsiella pneumonia	Nil	1	Norad	88	90	93	CPAP	40	16
29	826356	M	50	Acute kidney injury	ESRD	1	Norad	110	112	114	Nil	28	22
30	827501	M	70	Pneumonia, AKI	CKD	1	Norad	138	142	165	Nil	29	24
31	826756	M	70	Sepsis with MODS	CKD	2	Norad, Dopa	183	189	175	Nil	33	12.9
32	828346	F	45	Peritonitis	Nil	2	Norad, Dopa	124	110	107	Nil	36	15
33	831526	M	42	Septic shock ,MODS	CLD	1	Norad	186	180	167	Nil	26	17
34	823654	F	57	Viral pneumonia	Nil	1	Norad	126	120	121	Nil	30	7
35	812765	M	70	Dengue shock syndrome	T2DM,HTN	2	Norad, Vasopressin	137	130	124	Nil	34	10
36	830587	F	37	Viral pneumonia,ARDS	Hyperthyroid	1	Norad	116	114	137	Nil	46	16
37	831207	F	40	UTI,Sepsis	Nil	1	Norad	180	180	199	Nil	45	7.9
38	832209	F	62	Aspiration pneumonia	COPD	1	Norad	154	150	104	Nil	46	5.1
39	823108	M	68	Acute exacerbation of COPD	HTN	1	Norad, Vasopressin	166	160	188	CPAP	50	15
40	826520	F	75	Septic shock	T2DM	2	Norad, Dopa	140	145	178	SIMV	37	18.6
41	3448274	F	60	Upper GI Bleed Secondary to Portal HTN	Nil	1	Norad	138	130	128	Nil	41	10.4
42	1472597	F	40	Sepsis with MODS	Nil	2	Norad, Dopa	130	131	126	Nil	30	15
43	583261	F	46	B/L Pneumonia	Nil	1	Norad	111	120	90	CPAP	34	26.8
44	597610	F	54	Tb Pleural Effusion	Nil	1	Norad	150	142	140	CPAP	48	11.3
45	4174467	M	58	Peritonitis	Nil	1	Norad	129	125	130	Nil	45	12.7
46	3322233	M	26	Ca Bronchus	Nil	1	Norad	135	136	134	Nil	38	12.3
47	2476805	M	57	Acute exacerbation of COPD	CKD	2	Norad, Vasopressin	145	142	135	Nil	50	11
48	803866	F	63	HOCM	PVD	1	Norad	90	105	111	Nil	33	16.8
49	981719	F	40	Acute GE, AKI	Nil	1	Norad	166	120	139	Nil	44	8.5
50	719695	F	58	ALD, Portal HTN, SBP	Nil	1	Norad	142	149	140	Nil	29	14
51	592849	M	55	Sepsis	Nil	1	Norad	130	123	111	Nil	48	17

Serial no.	Patient no.	Sex	Age	Diagnosis	Co-morbidities	Vasopressors		Sugars			Ventilatory support	Lab Investigati	
52	2918718	F	29	Diselectrolytemia	T2DM	1	Norad	140	138	141	Nil	44	14.4
53	3855206	M	61	UGI Bleed secondary to Warfarin Overdose	Nil	1	Dopa	136	134	130	Nil	45	6.9
54	3695518	F	67	Acute exacerbation of COPD	Nil	1	Norad	124	124	120	Nil	31	13.1
55	3205955	F	72	Subendocardial Ischaemia	CKD	1	Norad	165	152	160	SIMV	46	10.4
56	1621427	F	30	Right lobar Klebsiella pneumonia	Nil	1	Norad	123	130	126	CPAP	44	8.5
57	732077	F	78	Acute kidney injury	CLD	1	Dopa	148	150	145	Nil	29	14
58	782600	M	62	Pneumonia, AKI	Nil	1	Norad	110	140	98	Nil	48	17
59	3262115	F	25	Sepsis with MODS	T2DM,HTN	1	Norad	210	210	182	CPAP	44	14.4
60	625734	M	61	Peritonitis	Hyperthyroid	2	Dopa, Norad	168	155	148	Nil	45	6.9
61	3304686	M	69	Septic shock ,MODS	Nil	2	Norad, Dopa	123	138	145	Nil	31	13.1
62	742070	M	60	Viral pneumonia	COPD	1	Norad	130	133	140	SIMV	46	10.4
63	2863622	M	58	Dengue shock syndrome	HTN	1	Norad	155	160	167	CPAP	48	5
64	3451044	F	42	Viral pneumonia,ARDS	T2DM	2	Norad,vaso	95	90	93	Volume control	44	18.1
65	789112	M	48	UTI,Sepsis	COPD,HTN	2	Norad,Dopa	198	180	178	CPAP	30	7.2
66	4091075	M	48	Aspiration pneumonia	Nil	1	Dopa	126	135	148	CPAP	35	27
67	1378178	M	60	Acute exacerbation of COPD	HTN	1	Dopa	65	60	58	Nil	30	15
68	2107020	M	37	Septic shock	Nil	1	Norad	110	103	98	CPAP	27	13.7
69	4260485	F	60	B/L Pneumonia, Septecemia, ARDS	Nil	1	Norad	134	155	144	Nil	35	35
70	799462	F	68	Left Lower Lobe Pneumona	T2DM, CKD	1	Norad	125	123	127	Nil	39	11.9
71	2579054	F	50	LRTI	Nil	2	Norad, Dopa	145	135	120	Nil	27	13.7
72	655957	F	53	B/L Pneumonia	Nil	2	Norad, Dopa	150	133	135	Nil	35	35
73	3680644	F	36	Viral Fever, Acute GE, MODS	T2DM,HTN	1	Norad	122	136	135	Nil	39	11.9
74	4267236	F	50	B/L Pneumonia,	T2DM	1	Norad	140	123	120	Nil	35	6.8
75	3589824	M	50	ILD	IHD	2	Norad, Vasopressin	100	108	98	Nil	43	22
76	1682689	M	57	Upper GI Bleed Secondary to Portal HTN	HTN	1	Norad	148	145	143	Nil	34	8
77	3422377	M	41	Sepsis with MODS	T2DM	1	Norad	97	119	110	Nil	42	13
78	4270672	F	28	B/L Pneumonia	Nil	1	Norad	165	180	145	Nil	40	16
79	4269012	M	77	Tb Pleural Effusion	Nil	2	Norad, Vasopressin	120	108	110	CPAP	28	22
80	792828	F	64	Tb Pleural Effusion	Nil	2	Norad, Dopa	140	135	125	SIMV	29	24
81	1998165	M	49	Peritonitis	Nil	1	Norad	138	180	129	Nil	33	12.9
82	2135172	M	39	Ca Bronchus	Nil	2	Norad, Dopa	125	130	127	Nil	36	15
83	732509	M	41	Acute exacerbation of COPD	Nil	1	Norad	145	153	156	CPAP	26	17
84	6403995	M	61	HOCM	Nil	1	Norad	128	144	145	CPAP	30	7
85	3340304	M	69	Acute GE, AKI	HTN	1	Norad	123	154	144	Nil	34	10
86	1460559	F	51	ALD, Portal HTN, SBP	Nil	1	Norad	184	155	164	Nil	46	16
87	777458	M	55	Sepsis	Nil	2	Norad, Vasopressin	128	124	144	Nil	45	7.9
88	3649721	M	62	Diselectrolytemia	T2DM, CKD	1	Norad	126	187	138	Nil	46	5.1
89	703862	F	53	UGI Bleed secondary to Warfarin Overdose	Nil	1	Norad	125	130	115	Nil	50	15
90	2533855	M	58	Acute exacerbation of COPD	Nil	1	Norad	90	89	78	Nil	37	18.6
91	3905037	F	65	Subendocardial Ischaemia	T2DM,HTN	1	Norad	144	104	98	Nil	41	10.4
92	785261	M	61	Pyelonephritis	T2DM	1	Norad	145	130	154	Nil	30	15
93	1454738	M	21	CLD,ARF	CKD	1	Dopa	110	108	103	Nil	43	22
94	4293324	F	19	Left hemiparesis secondary to IC bleed	Nil	1	Norad	123	115	112	Nil	34	8
95	3268371	M	71	Right lobar Klebsiella pneumonia	CLD	1	Norad	125	135	152	SIMV	42	13
96	4295441	F	38	Sepsis with MODS	Nil	1	Norad	164	160	154	CPAP	40	16
97	4270255	F	52	Peritonitis	T2DM,HTN	1	Dopa	98	99	115	Nil	28	22
98	2804479	M	60	Septic shock ,MODS	Hyperthyroid	1	Norad	198	180	173	Nil	29	24
99	547730	M	55	Viral pneumonia	Nil	1	Norad	156	153	145	CPAP	33	12.9
100	3460694	F	48	Dengue shock syndrome	COPD	2	Dopa, Norad	134	143	143	Nil	36	15
101	829134	F	47	Viral pneumonia,ARDS	HTN	2	Norad, Dopa	126	138	128	Nil	26	17
102	897923	F	50	UTI,Sepsis	T2DM	1	Norad	142	154	124	SIMV	30	7
103	796720	M	65	Aspiration pneumonia	Nil	1	Norad	128	130	126	CPAP	34	10
104	2094682	F	34	Acute exacerbation of COPD	Nil	2	Norad,vaso	142	143	123	Volume control	46	16
105	3670283	F	49	Septic shock	Nil	2	Norad,Dopa	70	83	72	CPAP	45	7.9
106	791465	M	62	B/L Pneumonia, Septecemia, ARDS	Nil	1	Dopa	254	219	210	CPAP	46	5.1

Serial no.	Patient no.	Sex	Age	Diagnosis	Co-morbidities	Vasopressors		Sugars			Ventilatory support	Lab Investigati	
107	3203790	M	47	Left Lower Lobe Pneumona	Nil	1	Dopa	156	129	142	Nil	50	15
108	668375	M	56	LRTI	Nil	1	Norad	98	110	103	CPAP	37	18.6
109	1164962	F	40	B/L Pneumonia	CKD	1	Norad	122	123	120	Nil	45	7.9
110	4324843	F	24	Viral Fever, Acute GE, MODS	HTN	1	Norad	132	132	131	Nil	46	5.1
111	4288515	M	20	Acute exacerbation of COPD	T2DM	2	Norad, Dopa	156	154	155	Nil	50	15
112	4330611	F	68	Subendocardial Ischaemia	Nil	2	Norad, Dopa	158	156	152	Nil	37	18.6
113	4336915	F	30	Right lobar Klebsiella pneumonia	Nil	1	Norad	123	120	98	Nil	41	10.4
114	752827	M	73	Acute kidney injury	Nil	1	Norad	164	168	124	Nil	30	15
115	4337592	M	24	Pneumonia, AKI	Nil	2	Norad, Vasopressin	150	143	133	Nil	34	26.8
116	3281625	F	20	Sepsis with MODS	Nil	1	Norad	133	134	124	Nil	48	11.3
117	3283106	F	56	Peritonitis	Nil	1	Norad	90	95	89	Nil	45	12.7
118	4349455	F	18	Septic shock ,MODS	Nil	1	Norad	92	92	91	Nil	38	12.3
119	3647136	F	25	Viral pneumonia	HTN	2	Norad, Vasopressin	69	82	90	CPAP	50	11
120	640608	M	59	Dengue shock syndrome	Nil	2	Norad, Dopa	159	134	129	SIMV	33	16.8
121	783794	M	47	Viral pneumonia,ARDS	Nil	1	Norad	143	129	120	Nil	44	8.5
122	4295441	F	38	Sepsis with MODS	Nil	1	Norad	128	124	144	CPAP	37	18.6
123	4270255	F	52	Peritonitis	T2DM,HTN	1	Dopa	126	187	138	Nil	45	7.9
124	2804479	M	60	Septic shock ,MODS	Hyperthyroid	1	Norad	125	130	115	Nil	46	5.1
125	547730	M	55	Viral pneumonia	Nil	1	Norad	90	89	78	CPAP	50	15

Serial no.	Patient no.	Sex	Age	Diagnosis	Co-morbidities	Vasopressors		Sugars			Ventilatory support	Lab Investigati	

Serial no.	Patient no.	Sex	Age	Diagnosis	Co-morbidities	Vasopressors		Sugars			Ventilatory support	Lab Investigati	

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