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COMBINED CENTRAL VENOUS OXYGEN SATURATION AND LACTATE AS MARKERS OF OCCULT HYPOPERFUSION AND OUTCOME FOLLOWING CARDIAC SURGERY

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ABSTRACT

Background: Routinely monitored parameters may not reliably detect perfusion abnormalities. However, $ScvO_2$ & lactate levels can detect occult hypoperfusion and identify patients at risk for complications. **Objective:** This study was aimed to examine the occurrence of postoperative occult hypoperfusion, determined by the combination of low $ScvO_2$ & elevated lactate & its effect on outcomes after cardiac surgery. **Study design:** Prospective observational study. **Setting:** Tertiary care hospital. **Patient population:** Patients undergoing different cardiac surgeries under cardiopulmonary bypass. **Results:** The study included 100 patients. Postoperative $ScvO_2$ & arterial lactate were obtained on arrival to the ICU & at 24 hours after ICU admission. Mod erate GTH was defined as ScvO2 <70% and lactate 2 to 4 mmol/L and severe GTH was defined as ScvO2 <70% & lactate >4 mmol/L. Occult hypoperfusion was defined as moderate to severe GTH with MAP>65 mmHg, CVP>8 mmHg & urine output >0.5mL/kg/h**.** 37 patients had occult hypoperfusion on arrival to Cardiac Surgical ICU. Patients with occult hypoperfusion on ICU admission had significantly longer ICU/Hospital LOS & ventilation time (p<0.001). Patients with severe GTH (n=15) had significantly longer Hospital LOS & significantly more no of complications per patient (p<0.05) as compared to those with moderate GTH (n=22). Patients who persisted with occult hypoperfusion at 24Hr (n=14) had significantly longer time on ventilator, ICU/Hospital LOS & no. of complications (p<0.05). **Con-**

clusions: The incidence of GTH is high after cardiac surgery. Postoperative $ScvO_2$ &lactate may be valuable measurements to identify patients with occult hypoperfusion & subsequently guide hemodynamic optimization to positively affect postoperative outcome in patients after cardiac surgery.

KEYWORDS: Routinely monitored hypoperfusion after cardiac surgery.

INTRODUCTION

Cardiac surgical patients are at risk of inadequate perioperative oxygen delivery caused by extracorporeal circulation and limited cardiovascular reserves.[1] The unyielding imbalance between oxygen demand and oxygen supply i.e., tissue hypoperfusion is an initiator for the cascade of events leading to multiple organ failure.[2] After cardiac surgery, most patients have a short intensive care unit (ICU) and hospital stay, however, up to 10% of patients need prolonged postoperative care, mainly because of organ dysfunction or multiple organ failure.[3]

The need for a tool to assess hypoperfusion accurately is paramount in curbing the incidence of organ dysfunction in these patients. Normalization of vital signs, such as blood pressure, urine output and heart rate may not reflect the adequacy of global perfusion.[4]

Central venous oxygen saturation ($ScvO_2$) and lactate indicate circulatory deficiencies and have been used frequently to better guide clinicians to begin and optimize early hemodynamic treatment and determine the resolution of global tissue hypoxia (GTH).[5] Lactate is a by-product of anaerobic metabolism. In states of global hypoperfusion, lactate production exceeds its rate of metabolism and the blood lactate levels rise. Elevated blood lactate has been correlated strongly with mortality in many types of shock.**[6]** After cardiac surgery, hyperlactatemia is relatively common and is associated with morbidity and mortality.[7] In the cardiac surgery population, lactate levels > 3 mmol/L in the early postoperative period are associated with increased risk for morbidity and mortality.[8]

Central venous oxygen saturation ($ScvO_2$) obtained from the superior vena cava, a well established surrogate for mixed venous oxygen saturation (SvO_2), reflects the balance between oxygen supply and demand. Central venous oxygen saturation ($ScvO_2$), reflects the balance between oxygen supply and demand.[9] $ScvO_2$ decreases in a compensatory fashion when oxygen supply decreases secondary to low cardiac output, hemoglobin or arterial oxygen saturation, or when oxygen demand increases due to fever, shivering, agitation or a hypercatabolic state. When this compensatory process is overwhelmed, global tissue hypoxia and lactic acidosis may ensue, with or without clinical signs of hypoperfu- sion.[8] Experimental studies have shown that changes in $ScvO_2$ closely reflect circulatory disturbances during periods of hypoxia, hemorrhage and subsequent resuscitation.[9] The prognostic significance of $ScvO_2$ reductions to below 65% has been demonstrated in trauma[10], severe sepsis[11], myocardial infarction[12] and cardiac failure[13]. Only limited data are available describing $ScvO_2$ values in the perioperative period.[14]

MATERIALS AND METHODS

This prospective, observational study was conducted in department of Anaesthesiology and Cardiac Surgical ICU at Sher-I-Kashmir Institute of Medical Sciences, Soura over a period of two years. Approval by our local ethical committee was taken prior to start of the study. A proper written informed consent was taken from all patients included in the study.

The study included 100 patients undergoing different cardiac surgeries under cardiopulmonary bypass. Echocardiography for ejection fraction measurement done within 6 weeks before surgery with ejection fraction of ≥ 45%.Triple lumen Central line in Internal jugular vein (IJV) confirmed by chest X-ray. Exclusion criteria for the surgery were patients undergoing off pump surgery, intraoperative cardiac arrest, presence of PA catheter, presence of ventricular assist device, presence of Cardiogenic shock/arrest prior to surgery, patients on vasopressors before surgery, patients on mechanical ventilation before surgery, Sr.creatinine >1.5mg/dl total bilirubin >1.2mg/dl, WBC count >12,000/mm^3 and platelet count <1, 00,000/mm^3 before surgery.

Anaesthesia technique and medications were similar to all patients. Anaesthesia was induced using combination of sodium thiopentone (5mg/kg), fentanyl citrate (3-5µg/kg), vecuronium bromide (0.08-0.1mg/kg). All patients were ventilated mechanically after intubation. Anaesthesia was maintained using combination of O_2 (50%) in N_2O, isoflurane 0.8-1.3%. Vecuronium bromide was used for muscle relaxation and fentanyl citrate (1 µg/kg/hr) was used for purpose of analgesia. Intraoperative monitoring was done with ECG, radial artery catheter for invasive BP monitoring, triple lumen internal jugular vein (IJV) catheter for measurement of CVP and fluid/medication administration, Foley catheter to measure urine output and nasopharyngeal probe to measure body temperature. Standard median sternotomy and aorta/right atrium cannulation was performed for CPB. CPB with systemic heparinisation (300U/Kg supplemented with additional boluses to maintain activated clotting time >350 secs), pump flow from 45 to 55 ml/kg/min, moderate systemic hypothermia (28^0 c and 30^0c) and intermittent cold blood cardioplegia was used for all of the patients. Patients were weaned off from CPB after nasopharyngeal temperature reached 37^0C. After completion of surgery heparin was reversed with protamine at a 1.5:1 ratio. Adequate blood pressure was maintained on CPB in order to maintain good perfusion (in normal adults MAP> 50mmHg, in adults with hypertension, coronary artery disease and old age MAP>60mmHg). Hypotension was managed using i.v fluids, blood and vasopressors.

Intraoperative CPB time and aortic cross-clamp time were noted for each patient. All patients were admitted postoperatively to the cardiac surgical intensive care unit and each patient received standard postoperative care as specified by treating health care teams.

Heart Rate, mean Arterial Pressure, central venous pressure, oxygen saturation, and urine output was monitored postoperatively. Postoperative blood samples for the measurement of central venous oxygen saturation ($ScvO_2$) and lactate were obtained on arrival to the Cardiac Surgical ICU and 24 hours after Cardiac Surgical ICU admission. Central venous oxygen saturation ($ScvO_2$) measurements were done on blood sample taken from central venous catheter by co-oximetry (catheter tip confirmed in superior vena cava using X-Ray chest). For measurement of lactate arterial sample were taken in fluoride tubes. Lactate was measured on semi-automated analyzer (Beckman Coulter AU460 series) using enzymic assays.

Results were interpreted as: Occult Hypoperfusion, moderate global tissue hypoxia and severe global tissue hypoxia.

Occult hypoperfusion was defined as moderate-to-severe global tissue hypoxia (GTH) with mean arterial pressure (MAP) >65 mmHg, central venous pressure >8 mmHg, and Urine Output >0.5 ml/kg/h**.**

Moderate global tissue hypoxia (GTH) was defined as central venous saturation ($ScvO_2$) <70% and lactate >2 to<4mmol/L. **Severe global tissue hypoxia (GTH)** was defined as central venous saturation ($ScvO_2$) <70% and lactate >4 mmol/L.

Patients were not discharged from the Cardiac Surgical ICU if any of the following was present.

- Hemoglobin oxygen saturation less than 90% on spontaneous breathing with fraction of inspired oxygen (FiO_2)≥ 0.4.
- Respiratory rate exceeding 30 per minute, or need for mechanical ventilation.
- Serum creatinine level exceeding 1.35mg/dl.
- Urine output of less than 800 ml per 24 hours
- Infusion of a vasoactive/ inotropic agent, either dopamine or dobutamine exceeding 2 mcg/kg/ min.
- Need of monitoring or treating newly acquired dysrhythmias.

Clinical variables to ascertain Multiple Organ Dysfunction Score (MODS) on days 1, 2 and 7 were collected.

Duration of mechanical ventilation, ICU length of stay (LOS) and hospital length of stay (LOS) were examined. In hospital mortality and postoperative complications were reviewed and were included in data analysis.

Postoperative complications included development of

1.Renal dysfunction or failure defined as a serum creatinine increase >0.5mg /dl above its preoperative value, serum creatinine increase >50% above its preoperative value, a serum creatinine >2mg/dl or dialysis requirement;

2. Prolonged ventilation defined as ventilation>48 hours,

3.Cardiogenic shock, cardiac arrest,

4.Development of acute respiratory distress syndrome,

5.Sepsis or infection.

DATA ANALYSIS

All the continuous variables of the study have been shown in terms of descriptive statistics like mean, standard deviation and categorical variables in terms of frequency and percentage. The standard statistical tests like student Independent t-test, repeated measurement analysis and Chi –square test, have been used to analyze the statistical data. All the results obtained, have been discussed on 5% level of significance i.e. p-value <0.005, considered significant. Moreover, the appropriate statistical graphs have been used to represent the data. The statistical software SPSS V20 has been used for analysis.

OBSERVATIONS

Of the 100 patients 46 were males and 54 were females. Sixty three patients had valve surgery, thirty one patients underwent ASD repair and six patients had surgery for intracardiac tumors.

Occult hypoperfusion was present in 37 patients on arrival to Cardiac Surgical ICU.Patients with occult hypoperfusion on ICU admission had significantly longer ICU LOS, Hospital LOS and ventilation time ($p<0.001$). CPB time and cross clamp time differ significantly among patients with and without occult hypoperfusion on ICU admission ($p<0.001$). MODS on day 1, 2 and 7 were significantly higher in patients with occult hypoperfusion.(table 1).

Twenty two patients had moderate GTH and fifteen patients had severe GTH among patients who had occult hypoperfusion on ICU admission. Patients with severe GTH had significantly higher MODS on Day 2 and Day 7, had significantly longer Hospital LOS, CPB time, Cross Clamp Time and significantly more no of complications per patient ($p<0.05$) as compared to those with moderate GTH. There was significant difference in EF (%) between patients with moderate and severe GTH ($p<0.05$). Patients with time on ventilator, longer ICU LOS, Severe GTH had numerically longer higher MODS on Day 1, more blood loss but none of them were statistically significant($p>0.05$).

Table 1: Table comparing different variables between patients with occult hypoperfusion (OH) and patients without occult hypoperfusion (NO OH) on ICU admission

Variables	Occult hypoperfusion (n=37) (Mean±SD)	Without occult hypoperfusion (n=63) (Mean±SD)	Mean difference	p-value
Ejection fraction	53.62±5.775	58.92±6.670	5.299	<0.001
CPB Time (in min)	102.11 ± 23.883	83.33 ± 25.989	18.775	<0.001
Cross clamp time (in min)	85.95 ±23.066	59.87 ±24.662	26.073	<0.001
Blood loss (ml)*	1001.22±215.70	950.79±250.63	50.42	0.309
MODS Day 1**	4.03 ± 1.443	2.75 ±1.218	1.281	<0.001
MODS Day 2**	2.16 ± 1.280	0.81 ±0.820	1.281	<0.001
MODS Day 7**	0.49 ±.837	0.11 ±0 .406	1.353	0.003
No. of complications*** per patient	1.054±1.053	0.412±.795	0.641	<0.001
Mechanical ventilation (in hr)	35.08 ± 19.664	12.68 ± 10.317	22.399	<0.001
ICU LOS (in hr)	88.00 ± 36.535	40.56±20.163	47.444	<0.001
Hospital LOS (in day)	14.35 ±3.853	9.98± 2.379	4.367	<0.001

*Intraoperative blood loss.

**Based on the ratio of arterial partial pressure of oxygen to fraction of inspired oxygen, serum creatinine, bilirubin, pressure-adjusted heart rate, platelet count and Glasgow Coma Scale.

***Complications include development of renal dysfunction or failure defined as a serum creatinine increase > 1.5mg/dl serum creatinine increase >50%, or dialysis requirement; prolonged ventilation defined as ventilation >48 hours; cardiogenic shock; cardiac arrest; or development of acute respiratory distress syndrome, sepsis, or an infection.

Twenty two patients had moderate GTH and fifteen patients had severe GTH among patients who had occult hypoperfusion on ICU admission. Patients with severe GTH had significantly higher MODS on Day 2 and Day 7, had significantly longer Hospital LOS, CPB time, Cross Clamp Time and significantly more no of complications per patient ($p<0.05$) as compared to those with moderate GTH. There was significant difference in EF (%) between patients with moderate and severe GTH ($p<0.05$). Patients with Severe GTH had numerically longer time on ventilator, longer ICU LOS, higher MODS on Day 1, more blood loss but none of them were statistically significant ($p>0.05$).(Table 2).

Table 2: Table comparing different variables between patients with severe and moerate global tissue hypoxia (GTH) on ICU admission.

Variables	Severe GTH(N=15) (Mean±S.D)	Moderate GTH(N=22) (MEAN±S.D)	Mean difference	p- value
Ejection fraction (%)	50.60±4.222	55.68±5.859	5.082	0.007
CPB time (in min)	113.53±28.814	94.32±16.354	19.215	0.03
Cross clamp time (in min)	96.67±26.043	78.64±17.940	18.030	0.017
Blood loss (ml)*	1012.04±196.96	985.33±246.90	26.712	0.717
MODS day 1**	4.27±1.907	3.86±1.037	0.403	0.464
MODS day 2**	2.93±1.387	1.64±.902	1.297	0.001
MODS day 7**	1.07±1.033	0.09±.294	0.976	0.003
Mechanical ventilation (in hrs)	36.13±20.563	34.36±19.485	1.770	0.792
ICU LOS (in hr)	96.93±48.928	81.91±24.423	15.024	0.285
Hospital LOS (in day)	16.33±4.746	13.00±2.390	3.333	0.021
No.of complications*** per patient	1.533±0.99	0.727±0.984	0.806	0.019

* Intraoperative blood loss.

** Based on the ratio of arterial partial pressure of oxygen to fraction of inspired oxygen, serum creatinine, bilirubin, pressure-adjusted heart rate, platelet count and Glasgow Coma Scale.

***Complications include development of renal dysfunction or failure defined as a serum creatinine increase > 1.5mg/dlserum creatinine increase >50%, or dialysis requirement; prolonged ventilation defined as ventilation >48 hours; cardiogenic shock; cardiac arrest; or development of acute respiratory distress syndrome, sepsis, or an infection.

37.84% (n=14) patients persisted with occult hypoperfusion and it subsided in 62.16% (n=32) patients out of 37 patients who had occult hypoperfusion on arrival to ICU. Patients who persisted with occult hypoperfusion at 24 Hrs had significantly longer time on ventilator, ICU LOS, Hospital LOS and significantly higher MODS on Day 1 (p<0.05) compared to those in whom it subsided. No. of complications per patient was significantly higher among patients who persisted with occult hypoperfusion at compared to those in whom it subsided (p<0.05).(table 3).

Table 3: Table comparing outcome between patients with persistent and subsided occult hypoperfusion at 24 hrs.

Variables	Persistent hypoperfusion at 24hrs (n=14)	Hypoperfusion subsided at 24hrs (n=23)	Mean difference	p-value
Mechanical ventilation (in hr)	50.29±17.094	25.83±14.947	24.460	<0.001
ICU LOS (in hr)	106.00±31.77	77.04±35.428	28.957	0.017
Hospital LOS (in day)	16.00±3.530	13.35±3.761	2.652	0.04
MODS Day1	4.86±1.099	3.52±1.410	1.335	0.0047
MODS Day2	2.5±0.76	1.95±1.492	0.543	0.215
MODS Day7	0.642±0.744	0.391±0.22	0.251	0.383
No. of complications* per patient	1.69±0.92	0.695±0.974	0.9472	0.006

* Intraoperative blood loss.

**Based on the ratio of arterial partial pressure of oxygen to fraction of inspired oxygen, serum creatinine, bilirubin, pressure-adjusted heart rate, platelet count and Glasgow Coma Scale.

***Complications include development of renal dysfunction or failure defined as a serum creatinine increase > 1.5mg/dlserum creatinine increase >50%, or dialysis requirement; prolonged ventilation defined as ventilation >48 hours; cardiogenic shock; cardiac arrest; or development of acute respiratory distress syndrome, sepsis, or an infection.

DISCUSSION

Due to compensatory mechanisms that maintain a normotensive and normoxemic state in the early stages of shock, routinely monitored parameters such as blood pressure, heart rate, arterial oxygen saturation, and urine output may not reflect the adequacy of global perfusion.[4] The ideal marker of adequate resuscitation should be able to assess resolution of hypoperfusion. Role of SvO_2, $ScvO_2$, and blood lactate have been studied extensively in number of clinical situations such as sepsis, septic shock, critically ill patients and general surgical patients. Lactate is a byproduct of anaerobic metabolism, elevated in hypoperfusion states when pyruvate cannot enter the Krebs cycle due to insufficient oxygen supply and it is shunted to lactate.[6] In states of global hypoperfusion, lactate production exceeds its rate of metabolism and the blood lactate levels rise.[4] Elevated blood lactate has been correlated strongly with mortality in many types of shock.[4, 6–11, 21, 22] The rapidity at which lactate is cleared from the blood during resuscitation better correlates with outcome, including mortality or organ failure, than a single measurement of lactate.[6, 11–14] **Jan Bakker et al[15]** in their study on documented septic shock patients observed that the group of patients who survived had significantly lower lactate levels and during course of septic shock there was a significant decrease in blood lactate levels. **Jean Michel Maillet et al[16]** in their study found that Hyperlactatemia was common following cardiac surgery and in their study a threshold of 3 mmol/L at ICU admission was able to identify patients at risk of morbidity and mortality after cardiac surgery.

Meregalli et al[7] in their study on noncardiac high risk surgical patients admitted to ICU hypothesised that patients without signs of clinical shock can still be hypoperfused and were at risk of complications. They concluded that lactate was superior to hemodynamic variables as a predictor of morbidity and mortality on ICU admission. **Ranucci et al[17]** in their trial reported that Hyperlactatemia during cardiopulmonary bypass was associated with an increased morbidity, related mainly to a postoperative low cardiac output syndrome resulting in insufficient oxygen delivery.

The utility of $ScvO_2$ in place of SvO_2 continues to be debated, however; $ScvO_2$ has been shown to correlate clinically with concomitantly measured SvO_2. Furthermore, it possesses an appealing attribute of not requiring placement of a more invasive pulmonary artery catheter. Although not numerically equivalent, ranges of values are pathologically equivalent.[8, 5, 18] **Rousti et al[19]** studied relationship between oxygen delivery, oxygen extraction ratio (O_2ER) and $ScvO_2$ in cardiac surgical patients. Increased O_2ER as a result of imbalance between oxygen delivery and consumption because of compromised cardiac function lead to decrease in $ScvO_2$. **Pearse et al[20]** measured ScvO2 and other biochemical, physiological and demographic data for 8 hours after major surgery and found that Significant fluctuations in ScvO2 occurred in the immediate post-operative period. These fluctuations were not always associated with changes in oxygen delivery, suggesting that oxygen consumption was also an important determinant of ScvO2. Reductions in $ScvO_2$ were independently associated with postoperative complications. **Filippo et al**[21] conducted a study on patients with brain injury after major trauma, found that ScvO2 value less than 65%, measured in the first 24 hours after admission, was associated with higher mortality and prolonged ICU/hospital LOS. In the group with lower ScvO2 also injury scores and lactate levels were worst. **Collaborative study group on perioperative $ScvO_2$ monitoring**[22] in their multicentre trial on high risk surgical patients found that low $ScvO_2$ perioperatively was related with increased risk of postoperative complications in high risk patients.

Till date there are only few studies done on cardiac surgical patients using both lactate and $ScvO_2$ or SvO_2 in combination. **Polonen et al**[3] in their protocolised care aiming $SvO_2 > 70\%$ and lactate <2 mmol/L, found that protocol group had shorter hospital stay and less number of complications. Faster hospital discharge and decreased morbidity in protocol group suggests that optimizing cardiovascular function either with volume loading or with the use of inotropes in addition to volume resuscitation has significantly contributed to improved outcome. **Ranucci et al**[23] retrospectively studied paediatric cardiac surgical patients who had undergone cardiac surgery with continuous intraoperative $ScvO_2$ monitoring and serial lactate measurements. They hypothesized that combined use of $ScvO_2$ and lactate during CPB may offer a predictive index for major morbidity after cardiac operations in paediatric patients and strategies targeted to preserve oxygen delivery during CPB may reduce the occurrence of low $ScvO_2$ and high lactate. In another study on cardiac surgical patients using combined index of $ScvO_2$ and lactate **Hu et al**[5] observed that patients with low $ScvO_2$ (<70%) and high lactate(>2 mmol/L) had longer ICU LOS, longer hospital LOS, longer time on ventilator and had more complications. They concluded that this combined index may be valuable in identifying patients with occult hypoperfusion and guide hemodynamic optimization. More recently in a study conducted by Ran Xu et al using combined index of $ScvO_2$ and lactate patients with occult hypoperfusion(OH) were identified and OH treatment pathway was implemented. Initial treatment included volume resuscitation and/or blood transfusion, followed by additional interventions when $ScvO_2$ remained < 70%. They concluded that an OH screening and treatment pathway following cardiovascular surgery was associated with significantly shorter hospital LOS and lower ICU readmission rate.

SUMMARY AND CONCLUSION

Although the morbidity and mortality rates after cardiovascular surgery are much lower compared with septic patients, there remain about 10% of those who do develop complications after cardiac surgery and therefore, undergo a prolonged ICU course and use a larger number of health care resources.[1]

Using $ScvO_2$ and lactate in combination, the clinician may be better able to discern if an elevated lactate is due to hypoperfusion issues using the $ScvO_2$ value. Evidence does show a strong relation between the degree and duration of hypoperfusion with the development of organ failure and death.

Following conclusions were drawn from our study

1.The incidence of occult hypoperfusion after cardiac surgery was remarkably high (37%), and the use of $ScvO_2$ and lactate facilitated rapid identification of these patients.

2.Patients who developed Occult hypoperfusion had significantly prolonged hospital stay, ICU stay, longer mechanical ventilation time and had more complications.

3.Patients who do develop complications after cardiac surgery and, therefore, undergo a prolonged ICU course and use a larger number of health care resources.

4.A significant no. of patients persisted with occult hypoperfusion at 24 hr (n=14). These patients had poor outcome in terms of ICU LOS, Hospital LOS and time on ventilator, emphasizing need to implement protocols targeting normalization of Lactate (<2mmols/L) and $ScvO_2$ (>70%).

Our study supports the use of $ScvO_2$ and lactate in combination to identify patients with hypoperfusion otherwise undetected by standard means of monitoring and potentially prevent the degree of hypoperfusion and the development of organ failure.

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