Critical care in Obstetrics

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INTRODUCTION:

A critically ill obstetric patient is one who, because of abnormal pregnancy, delivery, puerperium or because of effects of pre-existing systemic disease, anesthesia and surgery and other acquired condition on a normal pregnancy, delivery or in puerperium develops complications threatening her life for which she needs intensive monitoring, therapy and/or life support system. Medline literature review between 1987-94 revealed that percentage of obstetrics patients requiring intensive care is 0.1- 0.3. The mortality of critically ill,obstetric patients ranges from 12% to 20.[1]

Some of the common causes for pregnancy related ICU admissions are haemorrhage, hypertensive disease, rheumatic valvular heart disease, cerebral venous thrombosis, malaria, and viral hepatitis. In low risk patients, ICU admission may be necessitated in patients undergoing emergency caesarian section along with considerable blood loss when mechanical ventilation has to be prolonged for a long time.

Detection of the patient requiring critical care: Prompt recognition of a patient requiring critical care can prevent a salvageable situation from deteriorating. Important signs of serious injury and illness include low-blood pressure or weak peripheral pulses, cold termperature of extremities, and peripheral cyanosis. Poor cardiac output produces constriction of cutaneous arterioles and stimulation of sweat glands, resulting in the characteristic cold, pale and clammy skin. Subtle changes in mental status may indicate serious hemodynamic or metabolic abnormalities. This should not be mistaken for anxiety in a stressed patient. Other warning signs include reduced urinary output, dyspnea or hyperpnea, high termperature, unexplained fatigue, chest pains, and tachycardia or palpitations.

After massive haemorrage, meticulous monitoring of the patient's pulse and BP is mandatory. The consultant should make it a point to get the readings of the ancillary staff cross checked, at least in high risk cases. Recognising shock in an early stage may be missed otherwise, especially on busy days.

Prolonged surgery or surgery following massive haemorrhage will send the patient into shock. After haemorrhagic shock has been tackled, the obstetrician should continue to be vigilant, even after the vital parameters begin to improve, because, such patients may go into systemic inflammatory response and subsequent septic shock.

Principles of management of a patient needing critical care:

Management of a patient in an ICU should be systematic and should include clinical monitoring ,Respiratory support and Cardiovascular support, in addition to Correction of the cause .

Clinical Monitoring

The following parameters should be assessed.

Mental status: Altered mental status in a post-haemorrhage patient may indicate severe shock with a deficit of more than 40% of blood volume. This may be due to inadequate cerebral perfusion.

Heart rate: Tachycardia generally alerts one to the possibility of hypovolemia, but it could also be increased with cardiac impairment, infection, anxiety, fear, fever, exercise, or pain and discomfiture.

Blood pressure. It should be remembered that decreased arterial pressures during shock states may be delayed because the compensatory adrenal stress reaction tends to maintain blood pressure, at least transiently, with declining blood flow. Severely reduced cardiac output for periods of 40 minutes to 2 hours has been demonstrated before a significant reduction in arterial pressure. On the other hand, if fluids are used to restore blood pressure, the cardiac output and oxygen transport may still need to be corrected even if the blood pressure is normal.

Respiration: An acute increase in respiratory rate should prompt the physician to look for signs of pulmonary embolism or pneumothorax.

Auscultation of the lungs in the critically ill patient is very important. Pulmonary oedema, pneumonia, atelectasis etc, are important events that may develop in the moribund patient. Early detection with confirmation with an X-ray is necessary to institute prompt treatment.

Besides conditions of the lung, dyspnea may be caused by increased respiratory effort also[2]. Abdominal loading, caused by ascites, obesity, or pregnancy by itself could lead to elevation of the diaphragm, and may result in less effective ventilation and dyspnea. Massive hemoperitoneum/collection of pus in the abdomen could also lead to abdominal loading and consequent impairment of ventilation and should be thought of in a postoperative patient with distended abdomen and dyspnea. Electrolyte abnormalities, metabolic acidosis and renal failure may also increase respiratory effort..

Urine output: Hourly out put charts should be maintened 30ml/hour of urine is indicative of good kidney function.

Electronic monitoring and invasive haemodynamic monitoring.

Most ICU settings are equipped with multi-parameter electronic monitors, which will give continuous readings of pulse, cardiogram, and in some monitors, blood pressure monitoring. The oxygenation status of the patient is determined by the use of a pulse-oximetre in most Indian ICU's.

Pulse oximetry: Pulse oximetre measures oxyhemoglobin saturation value (SPO2) which is directly related to arterial partial pressure of oxygen-PaO2(Amount of oxygen dissolved in

plasma) . A reading of 95% SpO2 or less could indicate hypoxia and should be investigated. An SpO2 reading of 90% or less indicates significant hypoxia and requires immediate action.Pulse oximetre probes are usually kept on fingers and in severe shock, if the pulse oximetre shows no reading, it may mean that there is not adequate flow in the finger capillaries for the probe to pick up a reading. Dyes like methylene blue,nail polish and pigments like bilirubin can affect pulse oximetry reading. Pulse oximetry reading may be incorrect in patients with low perfusion, anemia and increased venous pulsation. External light source may also hinder correct readings.

Central venous pressure (CVP) monitoring:

CVP is the pressure of blood in the thoracic vena cava just before it (blood) enters the heart. Normal values are between 5-10cmH2O. It can be measured by inserting a catheter in the subclavian vein. Measuring CVP alone is useful in detecting extreme cases of hypervolemia, fluid overload or heart failure. CVP does not accurately reflect left ventricular filling in patients with preeclampsia, pulmonary and cardiac disease.

Pulmonary artery catheterization: The Swan-Ganz pulmonary artery catheter introduced into the pulmonary artery is used to measure, in addition to the Continuous central venous pressure (CVP), the pulmonary artery pressures and intermittent capillary wedge pressure (PCWP).

In certain patients, for eg; in patients with significant cardiopulmonary disease, there may be a lack of co-relation between measurements on the right and left sides of the heart. Drugs/fluids used to optimize ventricular preload can be monitored more accurately if the pulmonary artery pressures are known in these patients, so as to avoid pulmonary edema.

However, invasive monitoring using the Swan Ganz catheter has its own hazards and therefore it is recommended only in patients where precise hemodynamic data can improve decision making and where better interventions are possible.

Blood investigations:

Blood should be tested daily for Haemoglobin, blood counts, coagulation parameters, electrolytes, creatinine and charted. Arterial blood gases and pH are useful for screening pulmonary function in critically ill patients.

Haemoglobin: A falling haemoglobin or an inadequately rising Hb should alert one to the possibility of internal haemorrhage.

Coagulation parameters: A rise in prothrombin time, bleeding time, etc should be treated with fresh frozen plasma/cryoprecipitate. Platelet deficiency should be treated.

Electrolyte imbalance:

Sodium: Hyponatremia should be treated. Hyponatremia is defined as plasma sodium< 135mEq/L. Physical stress and postoperative pain can contribute to hyponatremia and should be tackled.

Hyponatremia with hypovolemia: Give IV 0.9%Nacl if patient is nil by mouth or oral salt containing water at a rate appropriate for the estimated volume depletion. Treat till a safe plasma sodium (generally 120-125mEq/L) is achieved.

Hyponatremia with hypervolemia(Oedematous state): Treat with diuretics, salt restriction, fluid restriction(intake <output) and correction of potassium deficit.

Hyponatremia with euvolemia: There is impaired water excretion wit normal or high total body sodium. In such patients fluid restriction is the most important treatment. Adequate restriction of fluid intake will gradually increase serum sodium concentration.

Pottassium: Hypokalemia is defined as persistent reduction of serum potassium below 3.5mEq/L.

Treatment guidelines:

3.5-4mEq/L: No potassium supplement; Increased oral intake of potassium rich food (fruit juices, coconut water, banana, juicy fruits, dry fruits, chocolate, coffee, soup) if patient can take food. If patient cannot ingest , give IV potassium;

3-3.5mEq/L: treat in high risk patients e.g. Congestive heart failure, digitalis therapy, history of acute myocardial infarction or ischaemic heart disease.

<3 mEq/L: Needs difinitve treatment. Potassium chloride (KCL) is usually the preparation of choice and will promote correction of hypokalemia as well as of metabolic alkalosis.

Potassium chloride solution, available in the market contains 20mEq potassium per 15ml solution (1gm=KCL+13.4mEq of potassium)Oral potassium preparation may cause G.I. irritation and shoud e taken with proper dilutionof in a glass of water, after food. IV potassium supplementation should be reserved for severe symptomatic hypokalemia(K^+ <3mEq/L) or for patients who cannot ingest oral potassium. To make tailor made potassium chloride infusion, 100mEq of potassium.(5 ampoules of 10ml., 15%KCl ampoules) is mixed in 1 litre of isotonic saline. Alternately 2 Ampoules can be added to 1 bottle of isotonic saline. Infusion of this saline at the rate of 100ml/hour (25 macrodrops or 100microdrops) will deliver 10mEq KCl per hour. 10ml of 15%KCl =1.5gm KCl=20mEq of potassium. 1 ml of 15% KCl=2mEq of potassium.

Calcium: Symptomatic hypocalcemia should be treated as emergency with 10% calcium gluconae (90mg elemental calcium/10ml) 10-20ml IV slowly over 10 minutes.

Respiratory support in the critically ill

A decision has to be taken if the patient needs to be put on a ventilator. The principal indications for ventilatory support are airway protection and respiratory failure. A compromised airway, or an airway at risk of compromise, may be identified by physical examination and ancillary testing.

Respiratory failure is almost always—and most appropriately—a clinical diagnosis. The decision to intubate and mechanically ventilate or to institute noninvasive ventilation support is generally made on clinical grounds without delay for laboratory evaluation.

Clinical criteria

- Apnea or hypopnea
- Respiratory distress with altered mentation
- Clinically apparent increasing work of breathing unrelieved by other interventions
- Obtundation and need for airway protection

Respiratory failure may also be easily identified with laboratory or pulmonary function data. Obtaining a $PaCO_2$ is useful to confirm respiratory failure when a broader differential diagnosis exists—for example, comatosed patients who may be hypercarbic but might have a reversible metabolic or toxicological etiology for their conditions—but adequate stabilization and ventilation of these patients should not be delayed to wait for laboratory results.

Ventilatory support is indicated for both hypercapnic respiratory failure and hypoxemic respiratory failure. It is also indicated for treatment of certain critical conditions such as correction of life-threatening acidemia, for intentional hyperventilation in elevated intracranial pressure or for suspicion of clinical brain herniation from any cause.

Laboratory criteria

Blood gases	PaO_2 <55 mm Hg
	$PaCO_2$ >50 mm Hg and pH <7.32
Pulmonary function tests	Vital capacity <10 mL/kg
	Negative inspiratory force <25 cm H_2O
	FEV_1 <10 mL/kg

Other criteria

- Controlled hyperventilation (e.g., in head injury).
- Severe circulatory shock

Hemorrhagic shock

If the patient is in haemorrgagic shock,one should aim to rapidly recognize the shock state, improve the circulating blood volume, improve cellular perfusion, correct metabolic disturbances while stopping haemorrhage.

IV access: Two large bore intravenous catheters and if possible a central venous catheter should be secured. Any large peripheral vein could be accessed. CVP can be measured through the subclavian vein.

Oxygenation: All patients with haemorrhagic shock should receive oxygen supplementation at nearly 12liters/min, by a closed mask or nasal cannula. Mechanical ventilation is indicated when there is ventilator failure or to relieve the metabolic stress of work of breathing in selected patients.

Fluid replacement: The goals of fluid replacement should be to obtain and maintain a systolic blood pressure at about 100mm Hg,urine output greater than 30ml/hour and maintain a pulmonary wedge pressure between 10 and 15mmHg.

Fluid replacement can be done with crystalloids, (Ringer lactate, Dextrose normal saline, 0.9%normal saline)or colloids. Commonly used colloids are, Dextran 40(10%soution in isotonic saline),Dextran70(6%solution in isotonic saline),Hetastarch(6%solution in isotonic saline),Albumin(5and 25% solutions in isotonic saline). Crystaloids are the initial volume expanding agents used in the ratio of 3:1 to the estimated blood loss. Colloids can remain in the intravascular compartment for longer and smaller volume is required to achieve volume repletion. Disadvantages are that anaphylaxis can occur, and there may be decreased platelet and increased prothrombin time.

Blood and blood products: Order blood transfusions if blood loss is ongoing and thought to be in excess of 2000 mL or if the patient's clinical status reflects developing shock despite aggressive resuscitation. It is advisable that such a patient should first receive 4 units of packed red blood cells, to improve the oxygen carrying capacity, after which whole blood should be administered to replace volume loss and provide the coagulation factors and proteins needed to maintain hemostasis and colloid osmotic pressure

Packed red blood cells (PRBCs) are the primary transfusion product used to increase the oxygen-carrying capacity. A typical volume of about 300 mL is mixed with normal saline before infusion. Diluting PRBCs with Ringer's lactate can cause calcium to precipitate with the citrate used as a preservative in stored blood. A single unit of PRBCs can be expected to raise the hemoglobin and hematocrit by 1 g and by 3%, respectively, in a nonbleeding patient.

When coagulopathy begins to set in, fresh blood by itself is not enough. Fresh-frozen plasma is a secondary transfusion product indicated mainly in states of coagulopathy or with massive transfusion. It comes in 250-mL units and contains all the coagulation factors, especially fibrinogen. One unit will raise the fibrinogen level by 10 mg/% in a nonbleeding patient. To avoid coagulopathy it is best to transfuse 1 unit of fresh-frozen plasma to every 4 units of PRBCs/whole blood, in an actively bleeding patient.

If coagulopathy continues and fibrinogen levels are low, consider giving cryoprecipitate. Cryoprecipitate is a tertiary transfusion product that contains as much fibrinogen as a unit of fresh-frozen plasma but in a volume of only about 15 mL. It also contains factor VIII, factor XIII, and von Willebrand's factor. It also will raise the fibrinogen level about 10 mg/% per unit. Its main indication for transfusion is in a hemorrhaging patient who is volume replete but has low fibrinogen levels.

If platelet count is less than 1lakh, platelet transfusion should be given. A unit of single-donor platelets raises the platelet count by 30,000 to 60,000 in a nonbleeding patient.

Use of Vasopressors or Ionotropic drugs:

If the BP continues to remain low in spite of adequate fluid replacement, continuing to give fluids may not be enough. Use of CVP or PCWP should be resorted to to know if the patient has been hydrated enough. In such patients, vasopressors have to be given.

Norepinephrine: In a patient with a systolic BP<70, after hypovelemia has been corrected, the pressor of choice should be Norepinephrine. . Dosage: 0.5-30umg/min IV. 1mg of Noradrenalin in 250 ml of 5% Dextrose can be given at 3microdrops / minute upto 45 microdrops / minute. On an average, about 8-10 drops may be enough. The use of norepinephrine is associated with improved mean arterial pressure, sustained aortic and mesenteric blood flow, and better tissue oxygenation when compared with fluid resuscitation alone, irrespective of time of administration. The early use of

norepinephrine plus volume expansion is associated with a higher proportion of blood flow redistributed to the mesenteric area, lower lactate levels, and less infused volume.

Dopamine: If the systolic BP is >70 Dopamine could be used as a vasopressor. It is the most commonly used vasopressor. Used in a dose of 3 to 10umg /kg/min, dopamine activates the B1adrenergic receptors and increases hea rt rate and myocardial contractility and hence improves the cardiac output. Two ampoules of Dopamine, (Each amp. Contains 200umg of the active drug in 5ml_ are added to 400ml of NS and started at either 8-10microdrops per min.

Dobutamine, Ephedrine, Vasopressin, Metaraminol, Methoxamine, and Phenylephrine are other vasopressors used.

Ancillary measures:

While all efforts are made to maintain a stable cardiovascular system, adequate attention should also be given to

1. Maintain electrolyte balance and check if calcium levels are normal.
2. Normalise body temperature
3. Restore urine output. Recognise the need for dialysis if kidney function fails.
4. Reverse systemic acidosis and bring down lactate levels.
5. Stop the cause of haemorrhage. Recognise re-bleed promptly. A falling Hb or an Hb which does not rise inspite of multiple blood transfusions should alert the clinician to the possibility of a bleed/rebleed which may need a second laparotomy. A bed side ultrasound would be helpful in such cases.

Septic shock

Sepsis is one of the five leading causes of pregnancy-related death around the world. The maternal mortality ratio is >1,000 per 100,000 live births (as estimated by World Health Organization, the United Nations Children's Fund, and the United Nations Population Fund).

Management: .Any source of infection should be identified and removed. For women with an infected abortion the uterine contents must be removed promptly by curettage. . Hysterectomy is seldom indicated unless gangrene sets in. With pyelonephritis, ureteral catheterization, percutaneous nephrostomy or flank exploration may be life saving.

Antibiotics: Choice of antibiotic should ideally be dictated by epidemiologic and hospital data, which is not always available in the Indian set up.

Recommended antimicrobial regimens for high-risk patients with intra-abdominal infection:

Single agents:

1.Imipenem/cilastatin

2.Meropenem

3.Piperacillin/tazobactam

Combination therapy

1. Aminoglycoside (amikacin, gentamicin, netilmicin, tobramycin) plus an anti-anaerobe (clindamycin or metronidazole)

2.Aztreonam plus clindamycin

3.Ciprofloxacin plus metronidazole

4.Third/fourth generation cephalosporin (cefepime, cefotaxime, ceftazidime, ceftizoxime, ceftriaxone) plus an anti-anaerobe (clindamycin or metronidazole)

Genital tract infections: For genital tract infections, the following drugs may be chosen:

Combination reginens:

a.penicillin (5 million units IV every 6 hours) or ampicillin (2 g IV every 6 hours) plus clindamycin (900 mg IV every 8 hours)

b.metronidazole (500 mg IV every 12 hours) plus gentamicin (1.5 mg/kg IV every 8 hours or 7 mg/kg ideal body weight IV every 24 hours) or aztreonam (1 to 2 g IV every 8 hours)

Single agents:

a.. imipenem-cilastatin (500 mg IV every 6 hours)

b.meropenem (1 g every 8 hours).

Correction of hypotension: Fluid therapy could be on the same lines as for haemorrhagic shock. Blood transfusion should be cautiously given as it may be assossiated with increased mortality. Patients can tolerate and may even benefit from hemoglobin levels lower than the traditional 10 g/dL If hypotension and organ hypoperfusion do not respond to volume infusion, then inotropic drugs (to improve cardiac performance) and vasopressor therapy (for hypotension) are indicated. Dopamine or norepinephrine is recommended as the first-line drug

Adjuvant management: Temperature should be brought down using cooling blankets. Daily haemodialysis or continuous venovenous haemofiltration should be used in patients with overt acute renal failure.

Newer modalities of therapy:

1.Use of steroids in septic shock has been controversial. A meta-analysis showed that hydrocortisone in doses from 200-300mg for 5 days or more reduced duration of shock, systemic inflammation, and mortality without causing harm Only patients with refractory septic shock and adrenal insufficiency benefit from hydrocortisone and 50 micrograms /day oral fludrocortisone can be added.

2. Vasopressin replacement therapy in doses ranging from 0.01-0.04IU/min improved haemodynamics and decreased catecholamine requirements. However,vasopressin might induce myocardial, cutaneous, or mesenteric vasoconstriction.
3. Use of polyvalent intravenous immunoglobulins were found to reduce mortality in studies, but high quality trials found no evidence that immunoglobulins were beneficial. Immunoglobulins , in experimental studies, have been postulated to improve opsonization , prevent nonspecific complement activation , protect against the antibiotic-induced liberation of endotoxin , neutralize endotoxin as well as a wide variety of superantigens .Dose: 0.5mg/Kg/day.

Pre-eclamptic toxemia

In a patient with severe preeclamptic toxemia needing ICU care, one should be alert to prevent eclampsia. PET may present with severe hypertension with a potential for end-organ damage, including retinal hemorrhage, papilledema, pulmonary edema, severe headache, and renal failure. Acute cerebral complications (eg, intracranial hemorrhage, massive cerebral edema) are particularly worrisome because they account for more than 75% of maternal deaths secondary to PEC. The goal of treatment is to prevent end-organ damage while still maintaining adequate uteroplacental perfusion. If urgent lowering of blood pressure is required, intravenous labetalol or intravenous hydralazine may be used. Some evidence suggests that labetalol may be the better choice.

Pulmonary edema develops most commonly (70%–80% of cases) in the postpartum period. It can be explained by the postpartum changes that include a significant drop in colloid oncotic pressure and the increase in preload that occurs with uterine contractions, the relief of vena caval obstruction after delivery of the conception products, and the mobilization of extravascular fluid that occurs in the initial 24 to 72 hours postpartum. Pregnant women generally respond to lower doses of diuretics than nonpregnant women, and most patients respond to 10 mg of furosemide administered intravenously. Renal failure in the setting of PEC is usually rapidly reversible. For patients with oliguria and rising creatinine, treatment with small fluid boluses (250 mL) may improve urine output. Fluids should be given with caution to prevent pulmonary oedema.

Pulmonary Embolism

Pregnancy by itself is a thrombophilic condition and thrombophilia prophylaxis should be given to patients with risk of developing deep vein thrombosis to prevent pulmonary embolism. Diagnosis of pulmonary embolism in pregnancy is difficult, but if diagnosed , should be treated with heparin.

Acute fatty liver of pregnancy, Amniotic fluid embolism, HELLP synfrome, Tocolytic induced pulmonary oedema and peripartum cardiomyopathy are some of the other specific conditions needing critical care, but treatment of all the disorders is beyond the scope of this article.

In conclusion, critical care of the high risk patient should be done as a team effort with the help of an intensivist, but the obstetrician should be aware of all the treatment and investigative modalities, by which he/she can actively participate in the ICU care of the patient.

References: