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Research Article

RUPTURED ECTOPIC PREGNANCY WITH CHRONIC KIDNEY DISEASE WITH ACUTE ON CHRONIC ANAEMIA - ANAESTHETIC CHALLENGES: A CASE REPORT

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A B S T R A C T

Chronic Kidney Disease (CKD) results in progressive and irreversible reduction in Glomerular Filteration Rate (GFR). Pregnancy in CKD patients have poor outcomes both for the mother and the fetus. Patients with CKD have a tendency to develop oedema and congestive cardiac failure or may become severely volume depleted when salt and water intake is decreased. The renal function deteriorates and hypertension can worsen leading to preeclampsia or eclampsia. Physiological anemia of pregnancy may worsen with CKD. Major anaesthetic challenges include cautious fluid management and pressure and volume overload, avoiding nephrotoxic drugs, electrolyte and acid base balance and good quality intraoperative and post operative pain management.

INTRODUCTION

CKD is classified into five stages using KDOQI (Kidney Outcomes Quality Initiative). Prevalence of CKD Stages 1 to 5 is 13·4% and 10·6% in stages 3 to 5. It is more prevalent in women than men. Renal failure is associated with hypertension, diabetes, peripheral vascular disease, cardiac disease and uremic encephalopathy. CKD is associated with normocytic, normochromic anemia due to insufficient production of erythropoeitin. It leads to decreased tissue oxygen delivery and utilization, increased cardiac output, ventricular dilation and hypertrophy. Two types of hypertension are seen in CKD due to salt and water retention and malignant hypertension due to high renin output.

Case Report

A 22 year old female, k/c/o CKD stage V for past 3 years, presented to the emergency with complaints of severe abdominal pain and amenorrhea for 8.3 weeks. She was not on any medications and gave no past history of hemodialysis. On examination patient was disoriented, cachexic and had pallor. Pulse- 130/min., Blood Pressure- 110/70mm Hg, bilateral pitting pedal edema and deformity in B/L lower limb likely s/o renal osteodystrophy. Rest examination was WNL. Investigations revealed hemoglobin 3.7 mg/dl, Total leucocyte count - 13,500/cu mm and normal platelet count. I/O- 2100/50ml, urea-109mg/dl, Creatinine- 5.65mg/dl with bedside albumin 1+ .

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ABG values were pH-7.28, pCO_2- 20.3 mmHg, pO_2-56.3 mmHg, HCO_3 15.7 mmol/l. USG whole abdomen revealed ruptured ectopic pregnancy with hemoperitoneum with bilateral moderate hydrouretronephrosis. USG Chest revealed bilateral mild pleural effusion. A well informed written high risk consent was taken and one PCV was transfused preoperatively. General anaesthesia was planned as the patient already had uraemic features. Routine monitors along with central venous catheter were connected. CVP was 4 cms of water. Patient was induced and maintained as per institutional protocol. Adequate analgesia ensured. Bicarbonate correction was done intra operatively. Patient was extubated uneventfully (maintaining SPO2 100% @ room air) ABG – pH- 7.40, pCO2- 25mmHg, pO2- 85mmHg, HCO2- 22 mmol/l

DISCUSSION

Pregnancy with CKD is a challenge to Anaesthesiologist. Pre operative optimization is best in elective procedures. Aim is to ensure adequate hydration and euvolemia. If anemia is present it is Normocytic, normochromic type, usually long standing and well tolerated; however, if bleeding is expected and the hematocrit is < 20% or Hb is < 7 g/dl transfusion should be considered. It occurs due to decrease in renal erythropoietin, increased RBC membrane fragility & hemolysis. Intra operative fluid administration should be restricted and volumes used should be aimed at replacing specific losses. Modifications to be made according to blood pressure, central venous, pressure, capillary wedge pressure and urine output.

Earlier normal saline was the preferred intravenous fluid as it has no potassium. However, recent studies reported that

normal saline can cause a hyperchloremic metabolic acidosis which may increase the incidence of hyperkalemia. Therefore balanced salt solutions (lactated Ringer solution, plasmalyte) are preferred.

Patients can develop hyperkalemia, hypocalcemia, hyperphosphotemia. Rapid transfusion of multiple units of packed red blood cells may increase potassium. Metabolic acidosis worsens transfusion-induced hyperkalemia and may trigger arrhythmias and cardiac arrest. Serum potassium should be < 5.5 mEq/L. Regular insulin (0.5–1.0 U/kg) rapidly decreases serum potassium. Hyperkalemia can also be corrected with loop diuretics, cation exchanger (sodium polystyrene sulfonate), sorbitol & hemodialysis being the last resort. 10% calcium gluconate (0.5–1.0 mg/kg IV) can be given to protect the heart.

Acidosis decreases the central nervous system threshold to the toxic effects of local anaesthesia, total anaesthetic dose should be reduced by 25%. Uraemic patients compensate for their acidosis by hyperventilating; failure to maintain this during anaesthesia results in an increase in PaCO2 & a fall in blood pH.

Patients with chronic renal failure tolerate general anaesthesia quite well and preoperative transfusion is usually not indicated. If required thawed frozen erythrocytes washed in saline should be used. A mild metabolic acidosis occurs when GFR drops below 25% of normal as a result of impaired renal excretion of acid and an overproduction of lactic acid. Initially, the anion gap is normal, but as renal function deteriorates there is an increase in the anion gap due to the retention of sulphates and phosphates.

An FiO2 of 0.4 or 0.5 is appropriate due to decreased oxygen carrying capacity. Volatile anaesthetics decrease GFR due to a decrease in renal perfusion pressure either by decreasing systemic vascular resistance or cardiac output, which is exacerbated by hypovolemia and release of catecholamines and antidiuretic hormone as a response to painful stimulation during surgery. Cisatracurium and atracurium are metabolized by ester hydrolysis and Hoffmann elimination. Excretion of neuromuscular reversal agents rely on renal excretion. Uraemic patients are very sensitive to barbiturates and benzodiazepines, secondary to decreased protein binding. Anticholinergics are not recommended as these patients have dry, friable mucous membranes. Also they have a delayed gastric emptying time (300 minutes) along with hyperacidity and increased gastric volume.

Regional anaesthesia techniques that achieve a sympathetic block of levels T4 to T10 may attenuate catecholamine induced renal vasoconstriction and suppresses cortisol and epinephrine release. In patients with chronic renal failure it is a logical choice, as it avoids the effects of muscle relaxants, narcotics and potent volatile anaesthetics.

Platelet dysfunction in uraemia is characterized by a prolonged bleeding time, a decrease in platelet adhesiveness and abnormal prothrombin consumption. Accumulation of guadininosuccinic acid, phenol and phelolic acid cause a decline in factor III availability, resulting in an inhibition of secondary platelet aggregation. The bleeding tendency could theoretically be a problem with spinal or epidural anaesthesia, due to the risk of haematoma formation.

CONCLUSION

Avoiding intra operative renal insults and maintaining euvolemia, adequate cardiac output, and renal perfusion pressure are the best interventions to prevent postoperative AKI. Pregnancy with CKD is a challenge to Anaesthesiologist. These patients can be well managed by thorough preoperative assessment and optimization, formulating good anaesthetic plans and prompt diagnosis and management of complications.

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