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EXTRAHEPATIC PORTAL VEIN OBSTRUCTION WITH PROXIMAL SPLENORENAL SHUNT FOR EMERGENCY LOWER SEGMENT CAESARIAN SECTION: A MANAGEMENT DILEMMA.

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ABSTRACT Even though pregnancy is rare with cirrhosis and advanced liver disease, it may co-exist in the setting of non- cirrhotic portal hypertension as liver function is preserved. Emergency management of pregnancy with non cirrhotic portal hypertension is challenging to the anaesthesiologist. Physiological changes of pregnancy can worsen the already present portal hypertension and predisposes the mother to potentially life-threatening complications like intractable variceal hemorrhage. We report a rare case of EHPVO with proximal splenorenal shunt with variceal bleed who underwent emergency caesarean section under general anaesthesia with satisfactory maternal and neonatal outcome.

KEYWORDS : EHPVO, Proximal splenorenal shunt, variceal bleed, caesarean section, general anaesthesia.

INTRODUCTION

Extrahepatic portal vein obstruction (EHPVO) is the obstruction of portal vein at the extrahepatic level that can sometimes also involve the intrahepatic portal veins or splenic or superior mesenteric vein[1]. The goal of anaesthetic technique for Caesarean section of a pregnant woman with EHPVO is to maintain a stable hemodynamic. Considering the rarity, the optimal anaesthetic management is either controversial or inconclusive.

CASE REPORT

A 28 year-old unbooked Primigravida with term pregnancy presents to our tertiary care centre in labour pains. On per vaginal examination, grade II meconium stained liquor was present. Thus a lower segment caesarean section was planned for the patient. Preoperatively on history, she had amenorrhea since 37 weeks 5 days, which was the result of missed oral contraceptive pills, which she took for around 2 years before the present pregnancy. She complained of intermittent bleeding in vomitus since 5th month of pregnancy for which she consulted obstetrician and was referred to a gastroenterologist. She underwent an upper GI endoscopy on which oesophageal variceal bleed was detected, so endoscopic variceal ligation was advised but patient defaulted. The bleeding vomitus continues till a day prior to the emergency LSCS. There was no other history suggestive of gestational diabetes mellitus, pregnancy induced hypertension or any other co-morbidity. While screening her past medical records we found that she had history of multiple episodes of haemetemesis 15 years back, for which she underwent upper gastrointestinal endoscopy which revealed oesophageal varices and then the patient had undergone the endoscopic oesophageal variceal banding 2-3 times although her symptoms persisted. Also as per her past medical records, she was diagnosed as a case of extra hepatic portal vein obstruction with variceal bleed with ascites with thrombocytopenia in hepatic encephalopathy. She was conservatively managed initially and thereafter underwent proximal splenorenal shunt surgery along with splenectomise. She stayed in intensive care unit for 9 days and thereafter discharged to home.

On Pre-anaesthetic examination, she had intermittent labour pains, conscious, oriented and following verbal commands. Pallor ++, cyanosis -, icterus -, Heart rate of 108 per minute, baseline blood pressure 90/50 mm of Hg, respiratory rate 16/min and NYHA class II. On inspection: No ascites, no dilated veins over abdomen. Fundal height corresponds to gestational age. On auscultation: Air entry bilateral equal and clear, $S_1 S_2$+ no murmur heard. Airway examination: Mouth opening 4.5cm, MMP class II, NROM: normal.

Her investigations revealed Haemoglobin 6.5 gm%, platelet count 1.1 lakh/mm^3, Bleeding time = 6 minutes 10 seconds , clotting time = 9 minutes 25 seconds, urine examination : NAD, LFT and RFT was not available because of urgency of operation. Ultrasonography abdomen (done at 5th month of ANC): liver coarse, portal vein not visualized, replaced by multiple collaterals, mild free fluid present, portal vein cavernoma formation present, splenorenal shunt patency could not be evaluated, single live intrauterine foetus corresponding to gestational age.

ANAESTHESIA CONDUCT

Proper counselling of family and relatives was done about the perioperative risks of excessive bleeding, need of multiple blood transfusion, haemodynamic instability, inotropic support, ICU stay, postoperative ventilation and need of postoperative endoscopic variceal band ligation. Well informed written High risk consent was obtained.

General anaesthesia was planned taking into consideration the urgency of operation, ongoing haemetemesis, severe anaemia and deranged coagulation parameters. All preparations to tackle hypotension were done like inotropic infusions, compatible blood and blood products were kept ready, surgeons were also advised not to exteriorize the uterus to decrease chances of rupture of collaterals and use electrocautery to achieve haemostasis if required.

Two 18 gauge IV cannulas were secured over both the upper limbs and ringer lactate started and patient received prophylactic antibiotics, ranitidine 50mg and ondansetron 4mg intravenously in the pre induction room. In the operating room all routine monitoring was attached including ECG and SPO2 and baseline parameters noted (HR= 104beats/min, BP=94/52mm of Hg, MAP=68mm of Hg). A 15 wegde was given for left uterine displacement.

After preoxygenation with 100% O2 for 3 minutes, rapid sequence induction was done with thiopentone 5mg/kg i.v, succinylcholine 1.5mg/kg i.v and trachea was intubated with 7.5 mm internal diameter cuffed oral endotracheal tube and mechanically ventilated with volume control mode with tidal volume of 8 ml/kg. Post induction blood pressure reaches 84/ 46 mm Hg (MAP 58 mm Hg), which didn't responded to fluid boluses, so noradrenalin infusion was started @0.5mcg/kg/hr and titrated to MAP 65 mm Hg. Anaesthesia was maintained with O2:N2O mixture 50:50 with isoflurane 0.4 to 0.8%.A healthy male baby weighing 2.9 kg was delivered with APGAR score 9 at 2and 5 minutes of birth. After delivery of the baby, oxytocin 10 international units were started in the running IV drip and good uterine tone was achieved. Fentanyl 2mcg/kg body weight IV and PCM 15 mg/kg body weight IV was given to the patient after the delivery of the baby. There was dilemma regarding nasogastric tube insertion as blind insertion could jeopardise the ongoing GI bleed, although fear of continuous upper GI bleed which can be unquantified without a nasogastric tube and also predisposes the patient to hepatic encephalopathy, prompted us to insert the nasogastric tube. A 14 no nasogastric tube, was inserted after lubricating with water based jelly atraumatically by manually gripping and lifting the thyroid cartilage. Dark coloured aspirate was suctioned from the nasogastric tube. Intraoperatively the estimated blood loss was around 1.3 litres. A total of 1 litre cystralloids, two units packed cell and 4 FFPs was transfused to the patient intraoperatively. Urine output intraoperatively was 250ml. At the end of surgery, pulse rate was 92/min and BP was 94/56 (MAP 64 mm Hg) on noradrenaline infusion@1mcg/kg/hr. Prophylactically 800 mg misoprostol tablets were kept per rectally and oxytocin infusion continued to prevent post partum haemorrhage. Patient was shifted to intensive care unit on ventilatory and inotropic support with noradrenaline infusion. Strict orders were given regarding vigilant monitoring of the nasogatric aspirate and post partum haemorrhage if any and not to overload the patient with the IV fluids. In ICU patient was stabilized hemodynamically, NG aspirate

becomes 100ml/4hrly, noradrenaline infusion was tapered and stopped on POD1. Gastroenterologist opinion was sought and patient was advised broad spectrum antibiotics, lactulose and beta blocker and an UGI endoscopy was advised. Bedside Doppler Ultrasonography abdomen was done which revealed decreased blood flow across the splenorenal shunt. With patient's family consent UGI endoscopy was done which revealed variceal bleed and hence endoscopic variceal ligation was done on the post operative day 2. Thereafter patient was transfused 2 packed cells. Patient was extubated on the POD3 and shifted to ward on POD4 and discharged on POD 7 on dual antiplatelet medications Tab ecosprin 150 mg and Tab clopidogrel 75 mg once a day with advice to follow up in gastroenterology opd after one week.

DISCUSSION

EHPVO is a rare condition with no definitive guidelines for its anaesthetic management. The etiological factors though not limited but includes home delivery and umbilical sepsis[2]. Hypercoagulable and prothrombotic state is an important risk factor for the EHPVO[3,4]. Idiopathic groups still form 30% of portal vein thrombosis (PVT). The presentation of EHPVO can be acute (recent) or chronic. In acute EHPVO, the presenting complaints are usually abdominal pain, ascites, jaundice, or fever[5]. In chronic EHPVO, patients present with repeated episodes of haemetemesis, due to re-routing of blood from oesophageal collaterals. Also there is extensive collateral formation known as portal cavernoma. The incidence of variceal bleeding in pregnancy in patients with EHPVO ranges from 20% to 34%.[6, 7] Definitive management includes portosystemic shunts, although in patients with shunt in situ, there is an 11% chance of rebleeding.[8] The rebleeding can further be exacerbated in pregnancy, as in pregnancy there is increased blood volume, increased cardiac output, and mesenteric vasodilatation[9] which all adds to the already increased portal pressures in EHPVO. Increasing intra-abdominal pressure in the second and third trimesters also contributes to portal hypertension by increasing the inferior vena cava pressure[10]. This all will lead to formation, opening of the gastroesophageal collaterals, ultimately increasing the risk of variceal bleeding.

Our patient though had undergone the shunt surgery but was prone to thrombotic condition due to the consumption of the oral contraceptive pills for two years before the present pregnancy which might be the cause of her decompensation. Our concerns in the case were: Severe anaemia, ongoing haemetemesis, deranged coagulation parameters, shunt patency status unknown, and full stomach. With spinal anaesthesia there was a risk of uncontrollable hypotension[11] as patient was already severely anaemic, ongoing UGI bleed and deranged coagulation profile. With epidural anaesthesia there was a risk of epidural hematoma due to deranged coagulation parameters, also the onset of anaesthesia would be delayed but the nature of surgery demands immediate effect for fetal well being as patient was having meconium stained liquor of grade II. Moreover epidural anaesthesia can add to hypotension. This all prompted us to opt for the General anaesthesia with rapid sequence induction.

What more could have been done →

Our case proceeded like

Caesarean→Transfusion→Inotropic support→ICU stay → EVL

If time permits the scenario would have proceeded in the reverse direction

EVL→Transfusion→Caesarean

Also shunt patency would have been assessed preoperatively by Doppler Ultrasonography and CT/MR angiography. Thromboelastography (TEG) could have been done to assess for the need specific transfusion of the blood and blood products. Senkstaken Blakemore tube would have been used to control the acute UGI bleed. Unfortunately all these were either unavailable in our setup or the urgency didn't permitted us to employ them.

CONCLUSION

EHPVO though a rare clinical entity, pregnancy outcomes are good if managed in a tertiary care centre. Operative deliveries are reserved for the obstetrical indications only. General anaesthesia offers a safe approach for emergency management of such cases, although more case reports are required to form a definitive conclusion and guideline regarding the management of such cases. Multidisciplinary approach and timely intervention can improve the maternal and neonatal outcome.

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