

ANAESTHESIA MANAGEMENT OF PREGNANT PATIENT WITH PORTAL HYPERTENSION POSTED FOR LSCS AND DEVASCULARISATION OF STOMACH

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PRESENTATION OF CASE

A 30-year-old female, 35 weeks of gestation, primigravida presented to emergency department with complaints of vomiting of blood and passage of dark tarry stools. She also complained of fatigue and weakness. She had similar episode 2 months back and diagnosed as splenic vein thrombosis with portal hypertension. That time, she was managed conservatively and discharged.

DIFFERENTIAL DIAGNOSIS

Upper GI scopy showed multiple linear varices into fundus and proximal body of stomach. USG abdomen was showing splenic vein thrombosis, significant splenomegaly. So, at that point, our differential diagnosis includes portal hypertension precipitated by hypersplenism, cirrhosis of liver, advanced stage of pregnancy, severe congestive heart failure and noncirrhotic portal fibrosis, as these all conditions leads to formation of varices.

CLINICAL DIAGNOSIS

The patient was asymptomatic up to 27 weeks of gestation, then developed vomiting of blood followed by passage of dark tarry coloured stool all of sudden. She visited a private hospital. USG showed splenomegaly with portal hypertension and splenic vein thrombosis. Upper gastrointestinal scopy done, which showed multiple linear fundic varices. At that time, she was managed conservatively with intravenous and oral medications and discharged.

But, again at 35 weeks of gestation, she presented to our hospital with same complains. She was resuscitated for low haemoglobin. Blood transfusion was done and patient was stabilised. She underwent upper gastrointestinal scopy, which suggestive of multiple linear varices into fundus and proximal body of stomach. As varices were continuing to bleed, patient underwent endoscopic gluing for varices. But, after gluing also, patient had 3 episodes of vomiting of blood. So, patient was planned for elective caesarean section as she was near term and devascularisation and fundic resection of stomach followed by splenectomy.

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On examination, patient conscious and well oriented in time, place and person. There was conjunctival pallor and pedal oedema. There was no cyanosis, clubbing, lymphadenopathy and icterus. Fundic height corresponding to weeks of gestation. Family history was not significant. Vitals were within normal limits. Ryle's tube was in situ, draining altered coloured blood. Systemic examination was normal.

USG abdomen showed portal hypertension with splenic vein thrombosis. Significant splenomegaly was there. Upper gastrointestinal scopy done, which showed multiple fundic and hilar varices.

PATHOLOGICAL DISCUSSION

Lots of haemodynamic and physiological changes occur in pregnancy as an adaptation to needs of growing foetus. These changes starts as early as 6 weeks and peaks around 32 weeks of gestation. There is increase in plasma volume, which also leads to increase in stroke volume and heart rate.¹ Maternal cardiac output increases 30-50% due to increase in stroke volume and cardiac output.

This increase in volume is directly related to increase in sodium retention. Increased glomerular and tubular reabsorption of sodium and water is due to increase in plasma aldosterone and oestrogen level. Erythrocytes volume is also increased by 30% in pregnancy. Under the effect of progesterone, there is decrease in systemic vascular resistance. Profound changes in systemic haemodynamic results in hyperdynamic state during pregnancy with increased pulse pressure. All these changes if associated with portal hypertension leads to formation of varices and in extreme conditions leads to rupture of varices. Patients who are suffering from liver cirrhosis, splanchnic arterial vasodilatation occurs due to increased local release of nitric oxide and other vasodilators. It causes variceal bleeding and impairment of circulatory function. Variceal bleeding, splenomegaly, ascites, encephalopathy are various clinical manifestations of portal hypertension.²

DISCUSSION OF MANAGEMENT

On preanaesthetic evaluation, patient with Mallampati classification I, TM joint movement was normal. Neck extension was adequate and breathe holding time of 30 seconds. On haematological examination, haemoglobin found to be 8 g and PT/INR=15/1.6, while all other parameters were normal. High-risk consent was taken. After confirming adequate starvation and availability of blood,

patient was shifted to OR. Neonatologist was standby for resuscitation of baby after LSCS.

Inside OT, monitoring of heart rate, noninvasive BP, SpO₂, capnography and ECG was done. Wedge was kept under right pelvis for left uterine displacement. Under local anaesthesia with all aseptic precautions, right IJV and left radial artery were IJV for CVP and arterial line for BP monitoring as it is also major surgery. Warm IV fluid RL started @ 4 mL/kg/hr. started to maintain CVP of 6-8 cm of H₂O.

Preoxygenation started with 3 no face mask @ 5 lit/min. For antiemetic prophylaxis, IV ranitidine 50 mg and ondansetron 4 mg were given IV. Rapid sequence induction was done using Inj. Thiopentone 250 mg IV and Inj. Succinylcholine 50 mg. Patient was intubated with 7 no. PVC cuffed endotracheal tube. Air entry was checked on both sides of lung and tube was fixed at 19 cms. Within 5 mins., baby was delivered, cried immediately after birth, birth weight of baby was 2.5 kg, after delivery of baby Inj. Midazolam 1 mg with Inj. Fentanyl of 80 mcg were given as sedation and analgesia. Inj. Pitocin 20 units was started. Maintenance of anaesthesia was done with O₂ (50%) + nitrous (50%) + Inj. Dexmedetomidine was started at dose of 0.5 micrograms/kg/min. During intraoperative period, vitals were maintained within normal limits.

After uterine closure by obstetrician, surgeons proceeded with gastric devascularisation. Anaesthesia was maintained with O₂ + N₂O + dexmedetomidine infusion @ 0.5 micrograms/kg/min. Intermittently, Inj. Atracurium was used as relaxant. Inhalational agents were avoided to prevent uterine relaxation. IV Pitocin was continued and CVP maintained about 4-6 cm of H₂O. After devascularisation, surgeons proceeded with splenectomy. After splenectomy, there was continuous bleeding through Ryle's tube, so surgeons decided to go with fundoplication with jejunostomy.

Procedure was uneventful. During intraoperative period, urine output was 500 mL and Ryle's tube aspirate containing blood was around 200 mL and blood loss of 2 litres. It was replaced with 4 pints of crystalloids + 3 units of whole blood + 2 units of fresh frozen plasma + 1 pint of colloid. Inj. Paracetamol 1 g was given for analgesia. In postop period, patient was extubated after adequate reversal of neuromuscular blockade and return of adequate tone, power and spontaneous respiration. Post extubation, patient was shifted to ICU for further monitoring. Oxygen was supplemented with Hudson mask 4 lit./min. Warm IV fluid Ringer lactate 2 mL/kg/hr. was started.

FINAL DIAGNOSIS

Our case had a final diagnosis of portal hypertension, which is secondary to splenic vein thrombosis and splenomegaly. These all conditions leads to formation of varices, which bleed. So, when conservative treatment fails, patient needs to undergo surgical repair to control bleeding. Our patient underwent procedure starting from nonsurgical in the form

of gluing of varices to surgical splenectomy and fundoplication of stomach.

Prognosis of such case depends on underlying aetiology of portal hypertension and derangement of liver function. Maternal complications are variceal bleeding, abruptio placenta and subcapsular haematomas. Perinatal prognosis is also poor because of preterm delivery and intrauterine growth retardation.³

The physiological haemodynamic changes associated with pregnancy worsen the portal hypertension thereby putting mother at risk of potentially life-threatening complications like variceal haemorrhage. It is common during second trimester because of maximally expanded blood volume, compression of IVC by large foetus and dilatation of collaterals. Variceal bleed may also occur during labour due to straining. So, assisted delivery or elective caesarean section is recommended.²

Management of portal hypertension in pregnant women is similar to that in nonpregnant patients. Beta blockers are given to reduce portal venous pressures. Management by gluing, banding and sclerotherapy has been successfully employed during pregnancy. It is possible to do shunt surgery during the second trimester.

In case of emergency, immediate delivery is the only definitive therapy in impending portal hypertension with variceal bleed. But, haemodynamic stability should be first confirmed before taking patients for any procedure.⁴

Our patient was posted for LSCS with devascularisation surgery due to risk of variceal rebleeding. Our anaesthesia concerns were to protect foetus from sedative effect of anaesthetic drugs. Prevent maternal stress response to avoid variceal bleed and same time rapid sequence induction to avoid aspiration of blood. Due to major surgery, major blood loss was expected and also maintaining uterine tonicity was important to prevent postpartum haemorrhage. So, to prevent uterine relaxation, inhalational agents were avoided and dexmedetomidine was used for maintenance of anaesthesia. Uterotonic drug like ergometrine was avoided and oxytocin was given by slow infusion.

REFERENCES

- [1] Aggarwal N, Negi N, Aggarwal A, et al. Pregnancy with portal hypertension. *Journal of Clinical and Experimental Hepatology* 2014;4(2):163-171.
- [2] Khanna P, Garg R, Roy K, et al. Emergency cesarean delivery in primigravida with portal hypertension, esophageal varices, and preeclampsia. *AANA Journal* 2012;80(5):379-384.
- [3] Sandhu GS, Shakti V, Sanjan D. Pregnancy with portal hypertension. *J Obstet Gynecol India* 2007;57(3):261-262.
- [4] Sandhu B, Sanyal AJ. Pregnancy and portal hypertension. *Portal hypertension: pathobiology, evaluation, and treatment*. Totowa, NJ: Humana Press 2005:365-381.