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Anaesthetic Management for Emergency Cesarean Section in a Patient with Budd chiari syndrome

Kiran sharma*

Assistant Professor, Department of Anaesthesiology and Critical care Kalpana Chawla Govt Medical College Kalpana Chawla govt medical college, India

Abstract

Budd Chiari syndrome represents a spectrum of disease states resulting in hepatic venous outflow occlusion . Managing a pregnant patient with Budd Chiari syndrome presents a unique challenge to the anaesthesiologist. We report the management of 30 years old primigravida with Budd Chiari syndrome on anticoagulant therapy, scheduled for cesarean section. Patient had thrombocytopenia with deranged liver function. Patient developed fetal distress and was taken up for emergency cesarean section under general anesthesia. Intraoperative and postoperative period was uneventful. Patient was discharged from hospital on the 7th postoperative day.

Keywords: Budd Chiari Syndrome, Anticoagulants, Thrombocytopenia, Emergency Cesarean Section, Anaesthesia

Corresponding author: : Kiran sharma

Assistant Professor, Department of Anaesthesiology and Critical Care, Kalpana Chawla Govt. Medical College and Hospital, Karnal , Haryana, India. **Tel:** +91 9729617945, **E-mail:** drmohitjindalortho@gmail.com

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Introduction:

Budd Chiari Syndrome is a heterogenous group of disorder characterized by occlusion or obstruction of hepatic veins or inferior vena cava, with progression to end stage liver disease.[1] There are few reports on the anaesthetic management of these patients presenting for cesarean section. We describe the anaesthetic management of patient with Budd Chiari Syndrome on anticoagulant therapy. Patient had thrombocytopenia with deranged liver functions. Patient developed fetal distress and was taken up for emergency cesarean section under general anesthesia. Patient recovered well and was discharged from hospital on the 7th postoperative day.

Case report:

30 years old primigravida weighing 65 kg with 36 weeks period of gestation was posted for cesarean section. She was a known case of Budd Chiari syndrome on anticoagulant therapy. Patient was diagnosed as a case of budd chiari syndrome three years back when she presented with history of pain abdomen followed by bloating and discomfort. Ultrasonography of the abdomen showed blockade of Inferior vena cava (IVC) and right hepatic vein. IVC stenting was done and patient was started on tablet warfarin . After one year stent got blocked and balloon expansion was done. Patient had stopped the anticoagulant on her own and again presented with epigastric discomfort for which balloon expansion was again performed. Patient presented to the hospital at 31 weeks period of gestation with swelling over face and body and was admitted for further management. Her investigations showed Hb 12.0gm%, Platelet count 160,000/ cu.mm. Liver function showed total bilirubin 1.0 mg %, SGOT 33 IU /L, SGPT 37 IU/L, ALP 231 IU /L. Renal function and coagulation profile was normal. Chest X-ray showed IVC stent in situ and no other abnormality was detected . Ultrasonography of abdomen showed liver size 12.5 cm, caudate lobe was enlarged , all hepatic veins were patent , portal vein diameter of 9mm and normal hepatoportal flow. Spleen was 11.8 cm with leinorenal collaterals visualized . Echocardiography showed normal study. Patient was treated with inj. Lasix, inj heparin 5000 IU s.c. twice daily was started. Patient received heparin for ten days but serial platelet count was on the lower side. Lowest platelet count recorded was 60,000 / cu.mm. Heparin was stopped on the tenth day and low molecular weight heparin (inj. Clexane) 0.6ml subcutaneously twice daily was started. Patient was discharged after two weeks of hospital stay.

Patient was readmitted at 36 weeks of gestation for elective cesarean section. Patient developed fetal distress and was taken up for emergency cesarean section under general anesthesia. On examination, patient was icteric, rest all other examination findings were within normal limits. Her investigations showed Hb 13.7 gm%, Platelet count was 60,000 /cu.mm, s. bilirubin 3.2mg % , SGOT 99 IU/L, SGPT 75 IU/L and ALP 292 IU/L. Renal function was normal. PT 15/ 13, PTTK 18/18, APTT 37/34. Patient had received anticoagulant 10 hours prior to surgery. Preoperatively , 4 units of platelets were transfused. Antiaspiration prophylaxis was given with inj ranitidine and inj. Metoclopramide. Monitors were attached for electrocardiography,

non invasive blood pressure, oxygen saturation. Baseline heart rate, blood pressure , oxygen saturation was recorded. Preoxygenation was done with 100% oxygen for 5 minutes . Rapid sequence induction of anaesthesia was done with inj thiopentone 250mg ,cricoid pressure was applied , 75 mg suxamethonium was given to facilitate endotracheal intubation ,trachea was intubated with no. 7.0 mm ID endotracheal tube. Anesthesia was maintained with oxygen and nitrous oxide (50 % and 50%) and 0.5% isoflurane. Inj. Atracurium in bolus of 10mg was given for muscle relaxation when the patient had respiratory effort and inj.fentanyl 50µg was given after delivery of baby. Healthy baby weighing 2.5 kg was delivered with Apgar Score of 8 at birth. After delivery of baby concentration of N2O was increased to 60%. Inj. Oxytocin infusion was given for uterine contraction after delivery. During the intraoperative period, patient was monitored for ECG, heart rate, non invasive blood pressure , oxygen saturation and bleeding from surgical site. Duration of surgery was one hour. All the vitals, blood loss and urine output were maintained well. Residual neuromuscular blockade was reversed with injection neostigmine 2.5 mg and inj. Glycopyrolate 0.4 mg. Trachea was extubated after adequate recovery of neuromuscular function. Postoperatively, patient was shifted to recovery room , put on oxygen by face mask and was monitored for vitals and bleeding. On the second postoperative day, inj. clexane was restarted . Her postoperative period was uneventful . Patient was discharged on the 7th postoperative day with the advise to start tablet warfarin.

Discussion:

Budd Chiari Syndrome is a heterogenous group of disorder characterized by occlusion or obstruction of hepatic veins and / or IVC, with progression to end stage liver disease. [1]. Budd Chiari Syndrome is a rare condition but exact frequency is unknown. Membranus webs are a common cause of Budd Chiari syndrome in Asian countries. Most patients have an underlying thrombotic diathesis. Causes are both genetic and acquired. Genetic causes including Antithrombin deficiency, Protein C deficiency, Protein S deficiency, and Factor V Leiden mutation.[2] Aquired causes are myeloproliferative disorder, Paroxysmal Nocturnal Hemoglobinuria,[3] Antiphospholipid Syndrome , [4]pregnancy and use of oral contraceptives.

Patient may present during pregnancy when the condition must be distinguished from HELLP syndrome and acute fatty necrosis of liver. Presentation could be fulminant, acute, subacute and chronic. Diagnosis is done by USG Doppler, Hepatic venography, MRI, CT, Echocardiography and liver biopsy. Treatment of Budd Chiari Syndrome includes treating the underlying cause. Stable patient with no symptoms need no intervention. Patients with ascites, pain abdomen and jaundice needs medical management and lifelong anticoagulation. Patients with treatment failure or recurrence or appearance of severe symptoms needs angioplasty with or without in situ thrombolysis. Patients with coagulopathy, encephalopathy, hepatorenal shunt needs portosystamic shunting and patients with shunt failure needs liver transplantation.[5,6]

Pregnancy precipitates Budd Chiari Syndrome, there is difficulty in diagnosis due to fatty liver of pregnancy and HELLP syndrome. Warfarin is avoided in the first trimester because of teratogenic effect. Unfractionated heparin does not cross placenta, it can be given as subcutaneous injection. Use of heparin needs APTT monitoring and there are chances of bleeding and heparin induced thrombocytopenia. Low molecular weight heparin do not cross the placenta and thus have a fetal safety profile equivalent to that of unfractionated heparin.[7]

Parturients with coagulation defects whether related to thrombocytopenia or to anticoagulant therapy , presents a unique challenge to the anaesthesiologist.[8] The risk of spinal or epidural hematoma in these patients has not been quantified fully but is a factor that one must consider on a case by case basis in determining whether neuraxial anesthesia is appropriate for the parturient.[9]

Both general and regional anesthesia has been given in these patients. [10, 11 ,12]

Our patient was a known case of Budd Chiari Syndrome on anticoagulant therapy. Patient had thrombocytopenia and deranged liver functions. Patient developed fetal distress and underwent emergency cesarean section under general anesthesia. Patient had received LMWH 10 hours prior to surgery. The effect of LMWH cannot be determined by laboratory test, so the patient must be off it for atleast 24 hours prior to neuraxial anesthesia.

To conclude, we would like to stress that Budd chiari syndrome is a rare disease. Managing these patients is a challenge as there is dilemma in the use of anticoagulants and patient needs regular follow up.(Table I)

Table 1: Anesthetic Consideration in Pregnancy with Budd Chiari Syndrome

1. Pregnancy precipitates Budd Chiari Syndrome
2. Dilemma of anticoagulation
. warfarin – teratogenic (14 – 36 weeks , safer)
.unfractionated Heparin – does not cross placenta
-needs APTT monitoring
- risk of Heparin induced Thrombocytopenia
3. Choice of anesthetic technique
. General Anesthesia- altered pharmacokinetics of drugs used
- Can precipitate deranged liver function
. Regional Anesthesia – risk of spinal or epidural hematoma

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