Anaesthetic Management of Obstetric Cholestasis Posted For Emergency Caesarean Section

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Abstract: Liver diseases that are most unique to pregnancy consist of hyperemesis gravidarum, acute fatty liver of pregnancy, intrahepatic cholestasis of pregnancy, and hemolysis, elevated liver enzymes and low platelets syndrome. In this review, risk factors, etiology, symptoms, diagnosis, prognosis and treatment of each entity followed by principles of anaesthetic management based on the case reports or retrospective records will be addressed.

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I. Introduction

Obstetric cholestasis is a reversible pregnancy specific liver disorder characterised by pruritis without rash, elevated serum amino transferases and bile acid levels, spontaneous relief of signs and symptoms within two to three weeks after delivery.¹

It is also known as intrahepatic cholestasis, pruritis gravidarum or icterus gravidarum. Incidence in India is 1.24%, etiology is complex likely result from cholestasis effects of reproductive hormones and their metabolites in genetically susceptible women.²

Here we are reporting a case of perioperative management of multigravida (G4P2L2A1) 33 weeks of gestational age(preterm) ,breech presentation with premature rupture of membranes and obstetric cholestasis posted for emergency caesarean section under general anaesthesia. 3,4,5

II. Case Report

A 25 year old unbooked preterm multigravida (G4P2L2A1) 33weeks of gestational age with history of pruritis since two months with yellow colour discoloration of sclera, skin and passage of urine since two weeks. Similar history present in previous pregnancies. On examination, HR 96/min, NIBP 126/80 mmHg, icterus was present. Systemic examination was within normal limits. Patient had full meals before 2hours on arrival to the hospital. Blood was sent for LFT,coagulation profile(PT,aPTT,INR) and viral markers.

Investigations available at the time of surgery were Hemoglobin 10.6g/dl, wbc count: 22.9, platelet count: 23910 cells/mm3, blood grouping and Rh typing: B POSITIVE

Anaesthetic Management

- Universal precautions were taken and general anaesthesia were planned in view of jaundice in pregnancy
- Rapid sequence induction with thiopentone 250 mg, succinylcholine 75mg and intubated with 7mm endotracheal cuffed tube.
- Maintained on o2:air till extraction of baby
- A low birth weight male baby of weight 1.7kg was delivered cried immediately after birth. Apgar score 8 at 1minute and 9 at 3 minutes, 10u oxytocin in 500ml NS started I.V. followed by 5u I.M. was given.
- After extraction till end of the surgery anaesthesia was maintained with isoflurane 0.8-1%, O2:N2O-1:3 ratio, and atracurrium 0.1 mg/kg. Fentanyl 50 mcg, inj.midazolam 1 mg given
- At the end of surgery, the residual neuromuscular blockade was antagonized with appropriate doses of
 neostigmine (0.05 mg kg-1) and glycopyrrolate (0.01 mg kg-1). Extubation was performed when respiration
 was adequate and patient was able to obey simple commands. Patient was then shifted to PACU for
 observation. Estimated blood loss was 600ml and urine output was 280ml.
- Investigations were revealed as LFT: Total bilirubin 6.5mg/dl, serum conjugated bilirubin 4.62mg/dl,serum unconjugated bilirubin 1.88mg/dl,alkaline phosphatase ALKP 326IU/L ,PT,aPTT,INR were within normal limit. serology- NON REACTIVE, USG abdomen & pelvis shows Hepatomegaly(16.8cm) with increased echotexture on postoperative day 1.

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- Diagnosis of obstetric cholestasis was made by exclusion. TAB. UDILIV 150mg BD were started for treatment of jaundice. Patient improved symptomatically and clinically by 6th post-operative day.
- Liver function tests were repeated and revealed as Total bilirubin 1.2mg/dl, ALKP 135mg/dl,USG on postoperative day 7 was normal.

III. Discussion

Obstetric cholestasis occurs in late second or early third trimester of pregnancy characterised by pruritis mainly in the palms and soles, elevated serum aminotransferases, raised serum bile acid levels with spontaneous relief of symptoms and normalisation of laboratory findings immediately post-partum in our patient. serum, alkaline phosphatase levels raises 7-10 times normal levels but difficult to interpret due to elevation of placental isoenzymes.^{6,7}

Sometimes symptoms are so severe that early termination of pregnancy is considered Incidence is 0.02% to 2.4% of all pregnancies, varying widely with geographic allocation and ethnicity. In Indian population, incidence is 1.24% (2). Aetiology is complex, likely to result from the cholestasis effects of reproductive hormones and their metabolites in genetically susceptible women (4)

There is increased chance of recurrence in subsequent pregnancies up to 45-70%.obstetric cholestasis is always a exclusion. Hence other causes of jaundice should be ruled out.⁸

During pregnancy, increased progesterone levels inhibit release of cholecystokinin which can lead to incomplete emptying of gall bladder resulting in cholestasis1. Intrahepatic cholestasis of pregnancy (IHCP) is a liver disorder that is confined to pregnancy. With an incidence of 1.24% in Indian population, IHCP mainly presents in third trimester as pruritus (80%) and jaundice (20%). The abnormalities in liver function tests are: bilirubin level less than 5 mg/dl with mild or no elevation in transaminases and elevated bile acids. IHCP is strongly associated with family history, rare in black ethnicity and has a high incidence of recurrence in subsequent pregnancies (45-70%). Related to the fetus, it can result in preterm delivery (20%) and meconium staining (25%). Incidence of fetal distress and death is high if early delivery is not induced. The presence of multiple gestations has been identified as a risk factor for the development of intrahepatic cholestasis of pregnancy, with reported rates that range from 9.5% to 20.9%.

Anesthesia choices for delivery might be challenging in IHCP. Due to the physiologic decrease in gall bladder contractility, pregnant women tend to have a sort of physiologic cholestasis. The cholestasis might lead to malabsorption of vitamin K. Vitamin K is a cofactor responsible from synthesis of coagulation factors II, VII, IX and X. Therefore, coagulation abnormalities might be expected in parturients with IHCP (11).

Maternal prognosis is usually good but increased risk of foetal outcome such as preterm delivery, meconium staining of amniotic fluid, foetal bradycardia, foetal distress and foetal loss particularly when associated with fasting serum bile acid levels > 40micromol/L. Undiagnosed coagulopathy increases the risk of intrapartum and postpartum haemorrhage.

Aim of treatment of obstetric cholestasis is to relieve pruritis as it causes sleepless nights due to itching, decrease the increased levels of bile acids and improve perinatal outcome

IV. Conclusion

Obstetric cholestasis has low maternal morbidity but has grave effects on the fetus. This case emphasizes the fact that IHCP should be considered as an important differential diagnosis of parturient with jaundice in third trimester. Early diagnosis and careful foetal assessment and appropriate medical intervention will improve both maternal and foetal outcome.

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