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REVIEW ARTICLE

ACUTE FATTY LIVER OF PREGNANCY: A CASE REPORT

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ABSTRACT

Jaundice in pregnancy can be caused by many conditions which can be either related to or not related to pregnancy. Intra hepatic-cholestasis of pregnancy (ICP), HELLP syndrome, and AFLP are diseases associated with pregnancy. Acute viral hepatitis with or without Liver failure, worsening of any underlying chronic liver disease or any tropical fever syndromes are the conditions which can occur during pregnancy but are not peculiarly associated with pregnancy. Acute Fatty Liver of Pregnancy is a rare disorder of pregnancy occurs in 1 in more than 10,000 deliveries with high mortality rate of approx. 12.5-18% (Ch'Ng, 2002; Rajasri *et al*., 2007). It usually occurs in 3rd trimester or in immediate post-partum period. Diagnosis is usually made on exclusion of other potential causes and is usually clinical. Urgent diagnosis is important as timely intervention with early delivery and supportive care, improves the maternal and fetal mortality and morbidity.

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INTRODUCTION

A 23 years old primigravida was referred from a peripheral hospital to our tertiary care center with history of LSCS done on the same day. Few hours after the surgery, she developed sudden onset breathlessness and decreased urine output. She was not known to have any medical illness in the past. She had an uneventful anti-natal period throughout the entire pregnancy. She delivered a live baby without any complications. At the time of presentation in ER of our hospital, she was conscious and following commands. Her blood pressure was 150/90 mm Hg, pulse rate was 140 beats/ minute; temperature was 97.6 F. Her work of breathing was high and she was tachypneic with RR of 28/min and spo2 89% on room air. She was already catheterized and her urobag was empty. Her RBS 76mg/dl; ABG showed pH-7.27, PO2-123.8, PCO2-29.8, HCO3-13.4, and Lactate-13.73 suggestive of severe lactic acidosis with compensated respiratory alkalosis. In view of breathlessness, hypoxia, increased work of breathing and lactic acidosis patient was electively intubated and put on mechanical ventilation with volume control mode. After initial stabilization of airway, breathing and circulation, patient was shifted to ICU. Thorough evaluation was done to look for the cause of her illness. CBC revealed microcytic anemia, marked leukocytosis, neutrophilia with shift to left and thrombocytopenia.

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PBS showed moderate dimorphic anemia with no evidence of any abnormal cell. Kidney function was deranged with Creatinine of 2.7 mg/dl. LFT showed marked transaminitis with ALT and AST of 1627 and 852, total bilirubin 6.91 with direct bilirubin 5.69. Coagulation parameters showed high PT-INR, increased APTT with decreased fibrinogen level suggestive of presence of DIC. Procalcitonin was 100, Pro BNP was >35000; Viral markers and tropical fever panel testing were negative. There was evidence of repeated hypoglycemia and patient required continuous dextrose infusion to maintain sugar levels in optimum range. Urine, blood, high vaginal and endocervical swabs for culture and sensitivity showed no growth of any organism. A chest x-ray (CXR) showed congestive pulmonary changes and blunted bilateral costophrenic angles. Her abdominopelvic computed tomographic (CT) scan without contrast enhancement revealed only hyper dense free fluid (ascites). 2D Echocardiographic screening at bedside revealed LVEF of 50 % with moderate TR. Patient was empirically started on broad spectrum antibiotics, transfusions of various blood products as per the coagulation parameters along with the guidance of Sonoclot and supportive care in terms of mechanical ventilation, vasopressor support and enteral nutrition. She required hemodialysis on regular intervals for AKI and anuria. In view of presence of jaundice, transaminitis in range of 1000s, coagulopathy, ascites, with multiorgan dysfunction with pulmonary edema, renal failure, rapid worsening of clinical condition in immediate postpartum period, repeated hypoglycemia and exclusion of other potential etiologies, diagnosis of acute fatty liver of pregnancy with liver failure

was kept as first differential. Possibility of acute viral hepatitis with liver failure was ruled out as report of hepatitis A, E B, C were negative. Possibility of acute liver failure secondary to ischemic hepatitis was also ruled out as left ventricular ejection fraction was 50% with no evidence of global hypokinesia. Possibility of septic shock with MODS was also remote as surgical site was healthy and CECT abdomen was not showing any collection. A possibility of HELLP was ruled out as there is no history of preeclampsia, hypertension, proteinuria in the antenatal period, no evidence of hemolysis, very high liver enzymes, sudden worsening in postpartum period, and presence of liver failure with jaundice, coagulopathy and ascites which goes against the diagnosis of HELLP.

Possibility of HELLP in our case was ruled out as there was no evidence of hemolysis (normal reticulocyte count, no increase in indirect bilirubin, and normal haptoglobin levels), very high transaminases in the range of 1000s, rapid worsening after the delivery in immediate post-partum period, presence of jaundice, coagulopathy, liver failure and intractable hypoglycemia, high ammonia levels and low albumin levels (Knight 2008). Acute fatty liver of pregnancy is a rare and fatal complication of pregnancy usually presents in 30th to 38th week of pregnancy but may present as late as 4 days postpartum (Bacq *et al*., 2007). This entity was first described by Stander and Cadden back in 1934. An enzyme deficiency associated with AFLP is

Table 1. Showing all the serial Lab Parameters during the course of her Illness

Date	10-06-2020	04-06-2020	31-05-2020	Units	Reference
Hemoglobin	7.2	6.5	7.1	gm/dL	12.0 - 15.0
Hematocrit	23	21	24	%	36.0 - 46.0
Total Leukocyte Count (TLC)	21.5	43.5	55.7	x10^3/µL	4.0 - 10.0
Neutrophils	92	95	93	%	40.0 - 80.0
Platelet Count	76	60	65	x10^3/µL	150.0 - 410.0
Peripheral Smear.	Impression: - Moderate Dimorphic Anemia, Moderate Leukocytosis And Moderate Thrombocytopenia.				
Creatinine, serum	2.19	2.03	1.93	mg/dl	0.5 - 0.9
Urea, serum	27.1	28.8	29.1	mg/dl	15-40
Sodium, serum	132	133	133	mmol/L	136.0 - 145.0
Potassium, serum	3.7	3.6	5.8	mmol/L	3.5 - 5.1
Chloride, serum	94	91	88	mmol/L	98.0 - 107.0
Bilirubin Total, serum	3.3	9.84	8.61	mg/dl	<>
Bilirubin Direct, serum	2.1	7.02	5.21	mg/dl	0.0 - 0.3
AST/ SGOT, serum	212	1447	5483	U/L	< 33
ALT/ SGPT, serum	104	904	1621	U/L	< 33.0
Alkaline Phosphatase, serum	208	230	195	U/L	35.0 - 104.0
PT Test , Plasma	14.2	26.3	32.4	Seconds	10.68 - 14.12
INR , Plasma	1.3	2.19	2.71	-	
D- Dimer, Quantitative	<245	-	17880	ng/ml	
NT-proBNP, Serum			> 35000	pg/ml	0.0 - 125.0

Table 2. The Swansea criteria

Vomiting	Abdominal pain
Elevated transaminases (AST or ALT) (>42 international unit/L)	Elevated ammonia (>47 micromol/L)
Polydipsia/polyuria	Elevated urate (5.7 mg/dl or >340 micromol/L)
Encephalopathy	Acute kidney injury, or Creatinine 1.7 mg/dl or >150 micromol/L
Elevated bilirubin (>0.8 mg/dl or >14 micromol/L)	Coagulopathy or prothrombin time >14 seconds
Hypoglycemia (<72 mg/dl or >4 mmol/L)	Ascites or bright liver on ultrasound scan
Leukocytosis (>11,000 cells/micro L)	Micro-vesicular steatosis on liver biopsy

DISCUSSION

Pregnancy with jaundice can be because of many causes like preeclampsia with or without HELLP, intrahepatic cholestasis of pregnancy, viral hepatitis, Cholelithiasis with cholangitis, and acute fatty liver of pregnancy (Mikolasevic *et al*., 2018). Intrahepatic cholestasis of pregnancy usually presents during the third trimester and main presenting symptom is marked pruritus with increased levels of bile acids. In this disease bilirubin is usually not more than 6 mg/dl and there is no evidence of liver failure (Lammert *et al*., 2000). Viral hepatitis with acute liver failure can present at any gestational age of pregnancy and is diagnosed on the basis of positive viral markers (A, E, B OR C) report (Beniwal, 2003). Cholelithiasis with cholangitis can occur at any stage of pregnancy and patient usually presents with high grade fever, pain abdomen and jaundice. USG abdomen is usually diagnostic (Chloptsios *et al*., 2007). Preeclampsia usually happens in second half of pregnancy and patient usually has hypertension and proteinuria. Liver enzymes can be deranged in severe pre eclampsia without HELLP (no hemolysis and no thrombocytopenia). Preeclampsia with HELLP is a common cause of jaundice and deranged liver enzymes in pregnancy. fetal long-chain 3-hydroxyacyl CoA dehydrogenase (LCHAD) deficiency that results in fetal fatty oxidation defects. LCHAD catalyzes a step in beta-oxidation of mitochondrial fatty acids that forms 3-ketoacyl-CoA from 3-hydroxyacyl-CoA. The accumulation of long-chain 3-hydroxyacyl metabolites produced by the fetus or placenta is toxic to the liver and may be the cause of maternal liver disease (Liu *et al*., 2017). AFLP generally presents as signs and symptoms of acute liver failure including jaundice, ascites, encephalopathy, disseminated intravascular coagulopathy, and intractable hypoglycemia. Patients are prone for developing acute kidney injury and multi-organ failure (Kennedy *et al*., 1994). A diagnostic model called as Swansea criteria is being proposed based on the clinical, laboratory, radiological and biopsy values. According to this criteria, 6 to 9 positive values out of 14, strongly favors the diagnosis of AFLP (Castro et al., 1996). Diagnosis is usually clinical and imaging techniques do not contribute much in diagnosing AFLP. Ultrasound abdomen shows fatty infiltration. MRI also showed transient excess of liver fat reflected as > 5 % of proton density fat fraction (Watson, 1990). The definitive management of the patient with acute fatty liver of pregnancy (AFLP) includes prompt delivery

of the fetus irrespective of gestational age. Delivery initiates resolution of this life-threatening disease (Kennedy *et al*., 1994) In case the initial presentation of AFLP is in immediate postpartum period as in our case, it requires a thorough evaluation to rule out other etiologies of postpartum liver dysfunction and jaundice like HELLP and severe pre eclampsia. Medical management includes supportive care along with management of coagulopathy and encephalopathy. In case of acute liver failure, patient may be transferred to a referral center for liver transplant evaluation and management.

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