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Case Report

Acute fatty liver of pregnancy

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ARTICLE INFO

Article history:

Received 24 February 2012

Accepted 10 June 2012

Available online 15 September 2012

Keywords:

Fatty liver

Disseminated intravascular coagulation (DIC)

Haemorrhage

related liver diseases that mimic AFLP such as HELLP syndrome: haemolysis (H), elevated liver enzymes (EL) and low platelet count (LP), and intrahepatic cholestasis of pregnancy. Bleeding and disseminated intravascular coagulation (DIC) are one of most common complications.

Case report

20-year-old female patient primigravida at 37-week period of gestation reported to labour room with complaints of nausea, vomiting and pain in the upper part of the abdomen for last 03 days. She was a booked case with last visit 15 days back and her pregnancy had been uncomplicated. On admission, her vitals were stable with blood pressure of 140/96 mm of Hg and urine protein was negative. On examination, jaundice and pedal oedema were present. The result of the complete blood count was as follows: haemoglobin 10.6 Gm%, TLC – 17,500/mm³, platelets – 317,000/mm³. The biochemical markers were as follows: total bilirubin – 8 mg%, AST-367 IU/L, ALT – 665 IU/L, urea – 52 mg%, creatinine – 2 mg%. The coagulation profile was as follows: PT – 14 s (control – 13 s), APTT – 27 s (control – 30 s) and INR of 1.13. USG obstetric showed single live intrauterine foetus at 36 week POG weighing 2.8 kg with AFI of 3 cm. Ultrasound liver showed grade I fatty changes. Peripheral blood smear demonstrated microspherocytes with nucleated RBC. Viral hepatitis markers (Hepatitis A, B, C, and E) were negative.

With all these findings, diagnosis of acute fatty liver of pregnancy was made. With anticipation of blood loss and disseminated intravascular coagulation, blood and blood products, recombinant factor VII (rFVIIa) was kept ready.

Introduction

Fatty liver is characterised by accumulation of microvesicular fat that literally crowds out normal hepatocytic function. Grossly, the liver is small, soft, yellow and greasy. Acute fatty liver of pregnancy (AFLP), a life-threatening obstetric emergency with a prevalence of 1 in 7000–16,000 deliveries.¹ Also known as acute fatty metamorphosis or acute yellow atrophy – most common cause of acute liver failure during pregnancy. The most common presentation is malaise, nausea, vomiting and epigastric pain followed by jaundice. Laboratory tests usually demonstrate high bilirubin levels, deranged liver and renal functions, coagulopathy and hypoglycaemia. At presentation, one should keep in mind other pregnancy

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<http://dx.doi.org/10.1016/j.mjafi.2012.06.013>

Dinoprostone gel applied intracervically for pre-induction cervical ripening and after 06 h labour augmented with Inj oxytocin. After 03 h with oxytocin she had a non-reassuring foetal heart tracing for which emergency caesarean was planned. Pre-operative coagulation profile was as follows: PT – 17 s, APTT – 37 s and INR of 1.86.

The patient underwent an emergency caesarean section and she delivered a live male baby with Apgar of 5, 8. Baby was resuscitated by paediatrician and anaesthetist. She was transfused 2 units of fresh frozen plasma (FFP) pre-operatively and 2 units of FFP intra-operatively. Pelvic drain was placed. Post-operatively, patient was shifted to ICU where for 02 days her vitals were stable, she was transfused 2 units of packed red blood cell (PRBC) as post-operatively her haemoglobin was 7.8 Gm%. Her coagulation profile was within normal limits. Her serum bilirubin level went upto 10.2 mg%, AST – 1010 IU/L, ALT – 660 IU/L and TLC – 21,200/mm³.

On 4th post-operative day patient had developed ascites and bilateral pleural effusion which was managed by diuretics and supportive care. On 9th post-operative day her serum bilirubin was 1.5 mg% and she was discharged on 12th post-operative day.

Discussion

Hypertensive and liver disorders of pregnancy comprise a spectrum of conditions associated with adverse fetomaternal outcomes.² The patient's initial presentation may vary during the antepartum, intrapartum and even the postpartum period. Although severe preeclampsia is commoner than AFLP and other liver disorders related to pregnancy, our patient's presentation and laboratory test results were in favour of an AFLP. Ch'ng et al first set the Swansea criteria for diagnosing AFLP.^{3,4} Six or more of the following features are used to diagnose AFLP in the absence of other explanations: vomiting, abdominal pain, polydipsia/polyuria, encephalopathy, elevated bilirubin >14 µmol/L (0.8 mg%), hypoglycaemia <4 mmol/L (72 mg%), elevated urate >340 µmol/L (5.7 mg%), leukocytosis >11 × 10⁹/L; ascites or bright liver on ultrasound, elevated transaminases, elevated ammonia >47 µmol/L (27.5 mg%), renal impairment creatinine >150 µmol/L (1.7 mg %), coagulopathy (PT >14 s or APTT >34 s), or microvesicular steatosis on liver biopsy. Using these criteria we anticipated the diagnosis of AFLP. The patient had vomiting, abdominal pain, elevated bilirubin and transaminases, elevated creatinine and coagulopathy at presentation. A patient with severe preeclampsia/HELLP will usually present with proteinuria.

Acute fatty liver almost always manifests late in pregnancy with a mean gestational age of 37.5 weeks.⁵ Fatty liver is more common in nulliparas with a male foetus, and 10–20% cases there is a multifetal gestation.⁶ The signs and symptoms at initial presentation may vary, making the diagnosis difficult or there might be delay in the diagnosis. Similarly, the differential diagnosis may be difficult in a patient with abnormal hepatic function in pregnancy since a number of pathologies such as HELLP syndrome, viral hepatitis and intrahepatic cholestasis of pregnancy share some of the same symptoms and signs. Persistent nausea and vomiting are major symptoms with half of affected women might have hypertension, proteinuria and oedema, alone or in combination.

There are variable degrees from moderate to severe liver dysfunction manifest with hypofibrinogenemia, hypoalbuminemia, hypocholesterolemia and prolonged clotting times. Serum bilirubin levels usually are less than 10 mg% and serum transaminase levels are modestly elevated and usually less than 1000 IU/L.

The diagnosis of AFLP remains challenging since there is no specific non-invasive diagnostic test to identify it. Ultrasound and CT scans of the liver have been used, but the specificity and sensitivity of these studies are insufficient to make a diagnosis and the likelihood of false negative results is high.⁷ Our patient had grade I fatty liver on ultrasound. Liver biopsy is the gold standard test, but it is invasive and requires a patient with normal coagulation status.⁸

Key to good outcome is intensive care support and good obstetrical management. Spontaneous resolution usually follows delivery. Maternal deaths are caused by sepsis, haemorrhage, aspiration, renal failure, pancreatitis and gastrointestinal bleeding.⁹ Although maternal mortality rates in the past approached 75% but Sibai (2007) cites an average mortality rate of 7% with 70% preterm delivery rate and perinatal mortality rate of approximately 15%, which in the past was nearly 90%.¹⁰ Prompt recognition and early diagnosis with delivery reversed these survival statistics over time.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. Reyes H, Sandoval L, Wainstein A, et al. Acute fatty liver of pregnancy: a clinical study of 12 episodes in 11 patients. *Gut*. 1994;35(1):101–106.
2. National High Blood Pressure Education Program Working Group. Report on high blood pressure in pregnancy. *Am J Obstet Gynecol*. 1990;163:1691–1712.
3. Joshi D, James A, Quaglia A, Westbrook RH, Heneghan MA. Liver disease in pregnancy. *Lancet*. 2010;375:594–605.
4. Knight M, Nelson-Piercy C, Kurinczuk JJ, Spark P, Brocklehurst P. A prospective national study of acute fatty liver of pregnancy in the UK. *Gut*. 2008;57:951–956.
5. Castro MA, Goodwin TM, Shaw KJ, et al. Disseminated intravascular coagulation and antithrombin III depression in acute fatty liver of pregnancy. *Am J Obstet Gynecol*. 1996;174:211.
6. Fesenmeier MF, Coppage KH, Lambers DS, et al. Acute fatty liver of pregnancy in 3 tertiary care centres. *Am J Obstet Gynaecol*. 2005;192:1416.
7. Usta IM, Barton JR, Amon EA, Gonzalez A, Sibai BM. Acute fatty liver of pregnancy: an experience in the diagnosis and management of fourteen cases. *Am J Obstet Gynecol*. 1994;171:1342–1347.
8. Castro MA, Goodwin TM, Shaw KJ, Ouzounian JG, McGehee WG. Disseminated intravascular coagulation and antithrombin III depression in acute fatty liver of pregnancy. *Am J Obstet Gynecol*. 1996;174:211–216.
9. Martin Jr JN, Briery CM, Rose CH, Owem MT, Bifill JA, Files JC. Postpartum plasma exchange as adjunctive therapy for severe acute fatty liver of pregnancy. *J Clin Apher*. 2008;23(4):138–143.
10. Sibai BM. Imitators of severe preeclampsia. *Obstet Gynecol*. 2007;199:956.