

CASE REPORT: ACUTE FATTY LIVER OF PREGNANCY IN A 30-YEAR OLD NIGERIAN PRIMIGRAVIDA

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ABSTRACT

Acute fatty liver of pregnancy is an uncommon, potentially fatal disorder that usually occurs in the third trimester of pregnancy or in the early post partum.

We present here a 30-year-old Nigerian primigravida with acute fatty liver of pregnancy. She was successfully managed and discharged.

Key Words: Acute fatty liver of pregnancy.

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INTRODUCTION

Pregnancy induces physiological, hormonal and physical changes¹. These changes may be responsible for the incidence of acute hepatic failure (AHF) in pregnancy both pre- and post- partum. Acute fatty liver of pregnancy (AFLP), pre-eclampsia and haemolysis, elevated liver enzymes, and low blood platelet count (HELLP) syndrome have been demonstrated as being the main causes of severe hepatic failure in pregnancy¹.

AFLP is an uncommon, potentially fatal disorder that usually occurs in the third trimester of pregnancy^{1,2,3}. Very few cases have been reported to occur in the second trimester². It is a rare but potentially fatal complication of pregnancy⁴. The first clinical descriptions came from Stander and Cadden⁵ who described 'acute yellow atrophy of the liver', a rare and fatal complication of pregnancy in 1934. The histological appearance of a micro vesicular fatty infiltrates, the clinico-pathological process and a mortality of 70% was soon described^{6, 7}. Most studies have estimated the incidence of AFLP as 1:10000-15000 pregnancies with mortality of 10-20%. Castro and colleagues⁸ found an incidence of 1: 6659 births in their study. It is commoner in the third trimester, twin pregnancies and male births and also in nulliparous mothers⁹. AFLP is of unclear pathogenesis and aetiology, but there is some evidence that some cases have been associated with a genetic deficiency of fatty acid beta- oxidation¹⁰. Its clinical picture is similar to pre-eclampsia including liver failure manifestations¹⁰. Onset of AFLP is between 30th and 38th weeks of gestation¹¹. It is sometimes preceded by prodromal illness, but classically presents with malaise, nausea, vomiting, headaches and pruritus¹¹. Patients require supportive

care, fluid resuscitation, nutrition, correction of coagulopathies and thrombocytopenia¹¹. We report a case of acute fatty liver of pregnancy in a 30-year-old Nigerian primigravida with liver dysfunction, (decreased albumin, prolonged prothrombin time, elevated liver enzymes) and disseminated intravascular coagulopathy (DIC) slowly returning to normal after delivery. This is the first of its kind to be reported in literature in Nigeria.

Case Presentation

A 30-year-old Nigerian primigravida in her third trimester presented at one of the highly specialized centres in the country with two weeks history of nausea, vomiting, weakness and loss of appetite. There were no previous histories of fever, headaches, diarrhoea or dark urine. No history of drug ingestion save the routine antenatal drugs (folic acid, Vitamin Bcomplex, ferrous sulphate and proguanil hydrochloride). Physical examination carried out showed a conscious young lady with low-grade fever, not pale, marked jaundice of the palms and the soles of the feet with moderate bilateral pitting pedal oedema. The chest was clinically clear, pulse was 78/min regular and of normal volume, blood pressure was 120/70 mmHg right arm supine, first and second heart sounds were heard and normal with no murmurs. Abdominal examination showed a fundal height of 36 weeks with good foetal heart sounds. Liver was palpable about 2cm below the right costal margin, slightly tender with total span of 10cm; spleen and kidneys were not palpably enlarged. An initial assessment of malaria in pregnancy was made to keep in view the possibility of viral hepatitis and acute fatty liver of pregnancy.

She was admitted and the following investigations were quickly ordered for; full blood count (FBC) with absolute platelets count, malaria parasites (MP), electrolyte, urea and creatinine (E&U+Cr), random

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blood sugar (RBS), urine analysis and culture, liver function tests (LFTs) including protein and albumin and the clotting factors. Hepatitis B antigen and C antibodies were also requested for. Meanwhile she was placed on 5% dextrose saline infusion, vitamin B complex into the drip and parenteral metoclopramide pending the outcome of the results. Results that came few hours later revealed the following;

Packed cell volume (PCV) of 37%, white cell counts (WBC) of $18,400\text{mm}^3$, with the differentials as follows; neutrophils =68%, lymphocytes =30%, eosinophils =02%, and platelets = 84×10^9 . MP was positive for trophozoites of *P. falciparum* and total count of $240/\text{mm}^3$.

LFT and E & U + Cr results were as shown below:

Parameter	Test value	Normal range
Bilirubin (total)	102mmol/l	0-20
Bilirubin (direct)	88.6mmol/l	0-5
Protein	58.0gm/l	58-80
Albumin	30.0gm/l	35-50
Globulin	28.0gm/l	20-45
SGOT	110.0 iu/l	0-18
SGPT	100.0 iu/l	0-22
Alkaline phosphatase	154.0 iu/l	9-35
Gamma GT	375.0 iu/l	4-28
Prothrombin time (PT)	20s	12-14s
PTTK	52s	35-45s
Clotting time	17min 20s	5-11min
Sodium	136mmol/l	120-140
Potassium	4.9mmol/l	3-5
Urea	4.4mmol/l	2.5-5.8
Creatinine	175mmol/l	50-110
Calcium	2.0mmol/l	2.25-2.75
Uric acid	0.47mmol/l	0.12-0.36

RBS = 2.9mmol/l

Hepatitis B surface antigen (HbsAg) using DiaSpot HbsAg Rapid Test Kit and Hepatitis C antibodies (anti-HCV) using Clinotech Diagnostics Rapid Test & ELISA kits made in Canada were negative. Other HBV viral markers especially IgM anti-HBc could not be done due to lack of facilities for such markers. Urinalysis showed no protein and glucose, pus cells were 2-3/hpf and no casts were seen. Abdominal ultrasound showed a singleton baby girl, well placed placental and hepatomegaly with massive fatty infiltrations. From the picture above, a definitive diagnosis of acute fatty liver of pregnancy was made. She was then placed on the following; 10% dextrose water alternating with 5% dextrose saline, absolute bed rest, high carbohydrate, low protein and no fat diet, iv vitamin k 10mg daily and amodiaquine hydrochloride.

On the night of her first day on admission, she had spontaneous rupture of the membrane with poor

uterine contractions. Labour was augmented with the use of 5units of syntocinon in 500ml of 5% dextrose water. She was delivered of a live baby girl 4 hours after the induction commenced. She had retained placenta that was removed manually with difficulty. She sustained vaginal tear that was sutured. She lost about 2.5litres of blood shortly after the delivery and the blood was not clotting. She went out into hypovolaemic shock and was resuscitated with fresh frozen plasma and fresh whole blood transfusion. Progress and management outcome were monitored by serial tests: E & U + Cr, FBC, LFT, RBS, and coagulation factors that gradually returned to normal before her discharged home.

All together she had 12 pints of blood and blood products before she was discharged.

She had the following complications during admission; intermittent hypoglycaemia, acute renal insufficiency, DIC/Coagulopathy, puerperal sepsis and ascites. She was successfully managed for all the complications.

She was finally discharged home healthy with her baby girl after 5 weeks stay on admission.

DISCUSSION

Acute fatty liver of pregnancy is a rare entity that occurs exclusively during third trimester of pregnancy or early postpartum period^{12, 13, 14}. Usually the AFLP symptoms start one to two weeks before hospitalization with nausea, emesis, general uneasiness, jaundice, epigastric pain and other symptoms¹³. Laboratory investigations usually show high white cell count, bilirubin, transaminases, coagulation period and ammonia on one hand and decreased the platelets, haemoglobin, glycaemia, fibrinogen and antithrombin 111 on the other hand¹³. The hepatic biopsy should be left for those atypical cases.

The cause and pathogenesis of AFLP are not completely clear¹¹, but there is some evidence that genetic deficiencies of fatty acid beta-oxidation are associated to its occurrence¹⁵. Other predisposing factors include primiparity, multiple pregnancy, male foetal sex and pre-eclampsia.

Liver biopsy establishes the diagnosis and typically shows microvesicular, centrilobular fatty changes of hepatocytes¹⁵. Differential diagnoses include HELLP-syndrome (Haemolysis, Elevated Liver enzymes and Low platelet counts), cholestasis of pregnancy, pre-eclampsia and viral or drug-induced hepatitis¹⁵.

Maternal outcome has improved enormously during the last decade¹³. Foetal prognosis has also improved; nevertheless there is a mortality rate of 20%¹³. Early diagnosis, pregnancy interruption and handling in special care or treating complications has led to good materno-foetal results¹³.

In the case presented, she had been having prodromal symptoms of nausea, vomiting, weakness and malaise 2 weeks before presenting in keeping with the findings of Vigil-De Gracia P¹³. She also presented in the third trimester of her pregnancy in keeping with the existing literature and other case reports from other countries^{1,2,3}. A multidisciplinary approach was adopted in the management involving the Physician and the Obstetrician. The case being presented was transferred to the intensive care unit (ICU) after delivery.

The complications encountered in the management of this patient included:

Recurrent hypoglycaemic attacks that was treated with 50% dextrose and 10% dextrose. Coagulopathy/DIC as evident by the low platelet counts and the markedly deranged clotting profile, this was treated with fresh whole blood and fresh frozen plasma and vitamin K injections. Acute renal insufficiency was treated through strict fluid management and challenging with frusemide. Ascites that occurred shortly after delivery due to hypoalbuminaemia was treated with albumin infusion. Puerperal sepsis was managed with potent antibiotics (ceftriaxone and metronidazole) parentally. Our patient did not develop hepatic encephalopathy.

In all she had 7 pints of fresh whole blood and 5 units of fresh frozen plasma. She was discharged home after 5 weeks of hospital admission with full correction of her earlier deranged clinical and laboratory parameters. Repeat HBsAg done on the day of her discharge was negative.

The diagnostic pitfall here was that of distinguishing HELLP syndrome from AFLP as both have been demonstrated as being the main causes of severe hepatic failure in pregnancy. They are thought to represent a spectrum of the same pathological process¹. They are also the causes of significant maternal and perinatal morbidity and mortality.

This case was unique because it represented the first of its kind to be reported in literature in Nigeria to the best of our knowledge. An early diagnosis of the illness and multidisciplinary management based on uterine evacuation and intensive medical care improved the prognostic outcome of this patient.

CONCLUSION

AFLP should be suspected in pregnant patients in the third trimester who present with jaundice, nausea, vomiting and abdominal pain. It is to be suspected also in those with obstetric complications such as acute foetal distress or rupture of membranes with symptoms of pre-eclampsia and laboratory evidence of hypoglycaemia and thrombocytopenia. ICU treatment including early termination of pregnancy and the infusion of fresh frozen plasma / fresh whole blood or albumin alternatively are the diminishing keys for maternal and foetal morbid-mortality rates and complications. Monitoring of platelet counts and antithrombin activity during pregnancy is recommended for identifying women at an increased risk of AFLP.

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