Case Report

Acute renal failure during acute fatty liver of pregnancy

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Acute fatty liver (AFL) is characterized by jaundice and liver dysfunction. It can occur at the end of pregnancy or in the early puerperium and is a rare cause of acute renal failure (ARF) [1,2].

We report here a case of a woman with AFL in a pregnancy complicated with ARF.

Case

The patient, who was 28 years old and in her first pregnancy (34 weeks), was admitted to the obstetric ward because of jaundice. She complained of weakness, anorexia, and vomiting. No history of high blood pressure was reported. On the first day of hospitalization she spontaneously delivered a ‘mors in utero’.

The immediate postpartum period was followed by a severe haemorrhage and it became necessary to perform a subtotal hysterectomy because of persistent atony of the uterus. However, jaundice advanced and the clinical situation worsened. Twenty-four hours after delivery the urinary output was 1.2 l/day and laboratory tests showed a minimal proteinuria, pronounced anaemia but with a normal platelet count and bilirubin levels were 4.97 mg% (direct) and 6.63 mg% (indirect). There were moderately increased blood levels of urea and creatinine but a pronounced increase of uric acid (13.4 mg%). The glomerular filtration rate (GFR) was calculated to be 15 ml/min. SGOT was 102 U and SGPT was 55 U. On the second day of hospitalization, a moderate microscopic haematuria was observed. Plasma electrolytes levels were potassium 2.8 meq/l and calcium 0.98 meq/l. The platelet count was decreased (84 000/h.p.f.). On the third day the levels of urea (380 mg/dl) and creatinine (5.6 mg/dl) increased. Blood transfusions resulted in an improvement in anaemia and a normal urine output was observed. Direct bilirubinemia predominated (5 mg/dl) over indirect bilirubinemia (1 mg/dl). Some ascitic fluid was detected.

On the ultrasound of the abdomen a brightness of the liver was noticed and both kidneys were of normal dimensions with a slightly hyperechogenic parenchyma.

Despite intensive therapy, the patient’s conditions deteriorated 12 days after hospitalization. The Hct was 17%, haemoglobin 5.5 g/dl, platelets 45 720, bleeding time 1.5 min, coagulation time 2 min, level of prothrombin 56%, alkaline phosphatase 194 U, and proteinuria 4.8 g/dl. Normal urinary output persisted but blood urea was 350 mg/dl and creatinine 6.4 mg%, SGOT was 74 U, and SGPT was 61 U.

Soon thereafter, the patient had profuse melena and haematemesis and she died on the 13th day after delivery.

Discussion

This case is an example of ARF in the immediate postpartum period of an ‘à terme’ pregnancy in a patient with functional and organic liver abnormalities. Before the hepatic malfunction, the patient had neither high blood pressure nor proteinuria, excluding the diagnosis of pre-eclampsia. In addition, during the pregnancy no abnormality of blood coagulation was detected. Urinary output also remained constant (1–1.5 l/24 h). At admission, her renal function was not changed. However, after stillbirth, her condition deteriorated progressively. First, liver function deteriorated quickly and the size of both liver and spleen increased; icterus became more pronounced and bilirubin levels reached 11.6 mg%. Total blood protein decreased to 4.6 g/dl and profound anaemia developed (Hct 23%); the latter was mainly a consequence of uterine haemorrhage. All these events could explain the alterations in renal function and the rapid increase of uraemia and creatininaemia immediately after delivery. GFR decreased to 15 ml/min. The
patient’s uraemia was disproportionately increased compared with her creatininaemia. On this basis, first a thrombotic microangiopathy could be suspected. In fact, the deteriorated clinical status included alterations of consciousness and obtundation. Further examinations such as the negative Coombs test, the predominant increase of conjugated bilirubin (8 mg%) the absence of reticulocytes (28%), and the slight thrombocytopenia (84,000/p.h.f.) led us to exclude a postpartum haemolytic uraemic syndrome (HUS). The findings suggested rather a major hepatic injury combined with ARF. This was confirmed by the ultrasound assessments of the liver and spleen (increased dimensions), the presence of ascitic fluid, conjugated hyperbilirubinaemia, increased levels of serum transaminases, and decreased levels of serum proteins.

Renal abnormalities became evident and they were corroborated by the ultrasound finding of hyper-echogenicity in both the kidneys, the presence of cellular elements in the urine, and above all the progressive and disproportionate increase in levels of blood urea in comparison with serum creatinine. In addition, the urine contained urate crystals persistently.

Despite intensive therapy, the clinical situation deteriorated progressively until the patient died on the 13th day of hospitalization.

Comments

It is clear that this patient suffered from multiple organ failure. ARF at the end of pregnancy can be the result of many causes. High levels of blood urea can be a result of extra-renal causes, mainly loss of water and electrolytes due to vomiting, diarrhoea, and diabetes. These causes were absent in our patient. Uterine haemorrhage and secondary hypotension can be responsible directly for acute tubular necrosis in 7–39% of cases [3,4]. In our patient the haemorrhage was certainly an aggravating factor, but it was not a very significant one; furthermore, it was treated quickly by giving blood transfusions and isotonic fluids. The pre-renal and haemodynamic abnormalities were probably additive factors in the aggravation of her renal dysfunction.

Another cause of renal failure in near-term pregnancy could be obstruction caused by stones in the urinary tract or by the pregnant uterus, but both these possibilities were excluded by ultrasound examinations. Another aggravating factor in our case could be tubular obstruction by urate crystals, which were abundant in the urine. Other possible causes were all excluded: such as microangiopathic syndromes like HUS or thrombotic thrombocytopenic purpura (TTP). We excluded these entities on the basis of absence of significant haemolysis despite the fact that blood platelets decreased significantly before the death of the patient. Furthermore, it is not usual to find increased transaminases in HUS and TTP, similar to our findings. In fact, HUS in near-term pregnancy can occur as a complication of pre-eclampsia; usually complete recovery is observed after delivery. Postpartum HUS is not a very clear entity and is often characterized by severe hypertension, which was absent in our patient [1]. In addition, we did not find the increased levels of reticulocytes that are characteristic of HUS. Pre-eclampsia with eventual haemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome could easily be excluded in our patient because of absence of hypertension and proteinuria.

Another excluded diagnosis is bilateral cortical necrosis, which in fact is a rare lesion, but some authors find it as a cause of ARF during pregnancy in 30% of cases [3]. This pathology commonly is a consequence of ‘abruptio placentae’, or of septic or haemorrhagic shock. Although a haemorrhagic syndrome was present in our case, it was not accompanied by total anuria, which is typical in renal cortical necrosis [5,6].

Although hepato-renal syndrome can occur in some cases with acute alterations of liver function, this is associated with oliguria or anuria, which was not present in our patient. The last and most likely diagnosis of AFL has to be discussed. This is a rare but dangerous complication occurring in the last trimester of pregnancy. The aetiology is unknown although several factors have been suggested to play a role such as tetracycline toxicity, viral infections, or intravascular coagulation [7]. AFL is characterized by progressive jaundice and liver failure. High blood pressure is found in only 20–50% of cases [1]. The most striking feature of this syndrome is a high level of bilirubin associated with a moderate increases of transaminases. The most relevant pathologic findings in the liver are fatty microvascular infiltrations (steatosis), mainly in the centro-lobular area. Ultrasound and CT scan can confirm the diagnosis. In our patient a bright sparkling hepatic structure compatible with fatty liver infiltration was seen. The incidence of ARF in AFL is over 60% in the US [8], but this ARF is frequently of a moderate degree and usually does not require dialysis. In our case, serum creatinine did not exceed 6 mg% and oliguria was absent. Hyperuricaemia in this case is in keeping with the diagnosis of AFL where characteristically hyperuricaemia is disproportionally increased in comparison with renal dysfunction [9]. The high mortality seen in AFL is due to liver failure, as was the case in our patient.

AFL is observed in primigravidae in 28–39 weeks of pregnancy and is rarely seen postpartum. When the women survive, this syndrome does not reappear in subsequent pregnancies [10]. AFL does not produce renal lesions and structurally the kidney is usually within normal limits. However, in some cases ‘focal lesions’ of acute tubular necrosis have been observed [3]. Some authors have reported ‘glomerular lesions’ and even ‘intraglomerular thrombosis’ [1].

The pathogenesis of ARF in AFL during pregnancy is not well understood. AFL has some similarities...
with Reye’s syndrome, which is common in childhood but is rarely accompanied by ARF. Sometimes, AFL is complicated by symptoms suggestive of disseminated intravascular coagulation. Also, in our patient, thrombocytopenia, hypofibrinopenaemia, and haemorrhagic phenomena manifested in the form of melena, signs compatible with disseminated intravascular coagulation.

In conclusion, the history of the disease in our patient, the clinical picture, the laboratory examinations, and the outcome suggest the diagnosis of AFL complicated by ARF in a near-term pregnancy.

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References


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