Ruptured subcapsular haematoma of the liver in pregnancy; a case report

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Summary

A case of ruptured subcapsular haematoma of the liver, a rare and potentially lethal complication of pre-eclampsia, is presented. The lack of characteristic signs and symptoms is highlighted. This case serves to remind clinicians of the high maternal and perinatal mortality rates associated with complications of hypertension in pregnancy.

Subcapsular hematoma; Liver; Pregnancy

Introduction

Rupture of Glisson’s capsule following a subcapsular hepatic haematoma is an exceedingly rare but serious obstetric emergency, carrying a high perinatal and maternal mortality. It is likely that subcapsular haematoma of the liver is less uncommon than previously thought, and that failure to recognise this condition early may have accounted for the originally dismal prognosis.

A case of rupture of the maternal liver capsule in pregnancy leading to haemoperitoneum is described, illustrating the ease with which the diagnosis can be missed, and the need for a multidisciplinary approach to secure a favourable maternal and fetal outcome.

Case report

A 28-year-old woman, para 2, was admitted to the Maternity Unit of Raigmore Hospital as an emergency with a 1 week history of progressively worsening epigastric pain, vomiting, hypertension and proteinuria at 38 weeks gestation. Her two
previous pregnancies had been uncomplicated. She gave a past history of medical treatment with antacids for peptic ulceration but had not been taking any medication prior to this pregnancy. She did not suffer dyspepsia during her present pregnancy.

One week prior to her admission, she saw her general practitioner because of severe epigastric pain, vomiting and diarrhoea. She was admitted to a peripheral General Practitioner Maternity Unit 66 miles from Inverness, and was treated with oral ranitidine and antacids with symptomatic relief. Her pain was sharp in nature, radiating to her right upper abdomen and loin, with a pleuritic component. She was normotensive, but proteinuria was noted. Her right renal angle was tender and a diagnosis of acute right pyelonephritis was made. She was started on oral cephalixin and her condition improved. She was discharged home after 48 hours. Urine culture was subsequently reported as negative.

Five days later, she was readmitted to the same peripheral hospital with acute exacerbation of her symptoms. Her temperature was 37.6°C and her right loin was more acutely tender. Her blood pressure was 140/100 mmHg, and proteinuria persisted. She was immediately transferred to Raigmore Hospital. She gave no history of headache or visual upset.

She was febrile and mildly dehydrated on arrival. Her blood pressure remained 140/100 mmHg, and her urine showed ++ proteinuria. Her upper abdomen and right renal angle were tender and her uterus was contractile. Vaginal examination showed that her cervix was 3 cm dilated, and forewater amniotomy was performed, revealing clear liquor. A provisional diagnosis of pre-eclampsia with pyelonephritis was made and labour was monitored.

Her haemoglobin was 12.1 g/dl, and white cell count 12000/µl with 85% neutrophilia. Her platelet count was 79000/µl and her coagulation profile normal. Her urea and electrolytes were within normal limits.

Fetal distress developed, requiring emergency Caesarean section soon after admission. A transverse incision was made in the lower abdomen and when the peritoneal cavity was entered, approximately 500 ml of fresh blood was noted. The uterus was intact. A baby boy weighing 2.72 kg was delivered through a transverse lower uterine incision, with Apgar scores of 2 and 6 at 1 and 5 min, respectively.

After closure of the uterine incision, it was evident that the bleeding was from the upper abdomen. Her pelvic organs were normal. The assistance of the surgeon on-call was sought. A tense, enlarged liver was palpated. The lower abdominal incision was closed and a midline upper abdominal incision was made, exposing the liver. A large subcapsular haematoma covering most of the anterosuperior surface of the right hepatic lobe was seen. Fresh blood was oozing from a large tear in the region of the Reidel's lobe. The left lobe of the liver was unaffected by the haematoma.

The ruptured Glisson's capsule was packed with Novacol®, an absorbable local haemostatic agent, and sustained pressure applied to achieve haemostasis. Peritoneal lavage was performed and the abdomen was closed. She was transfused with 4 units of whole blood, 2 units of fresh frozen plasma and 6 units of platelet-rich plasma. She was transferred to the Intensive Therapy Unit. Intravenous cefotaxime and metronidazole were started. Her liver function tests in the immediate postoper-
Fig. 1. Computed tomogram of the upper abdomen at the level of the 11th thoracic vertebra, showing the large subcapsular haematoma affecting the right lobe of the liver [1,2].

ative phase showed alkaline phosphatase of 141 U/l and alanine aminotransaminase of 228 U/l. Her liver enzymes and platelets reverted to normal by the 4th postoperative day. Her condition improved and she was transferred to the postnatal ward on the 3rd day. An ultrasound scan of the liver showed features consistent with an organising hepatic haematoma in the right lobe and this was also demonstrated on computerised tomography (Fig. 1). She was allowed home on her 12th postnatal day and a repeat ultrasound scan 2 weeks later confirmed a resolving liver haematoma. At her routine 6-week postnatal review, she was symptom-free and her uterus well involuted. There was no hepatomegaly. Her son was well and her husband proposed to seek vasectomy.

Discussion

While the incidence of subcapsular haematoma is difficult to define, it is probably commoner than previously thought [1]. The aetiology is still not clear, but its association with pre-eclampsia and eclampsia have been described [2–5], and its pathogenesis may be related to Disseminated Intravascular Coagulopathy (DIC) [6]. Histological features include periportal necrosis and fibrin deposition without an inflammatory reaction, consistent with pre-eclampsia [7] and the right lobe of the liver is affected in 75% of patients [8]. These features correlate with the clinical presentation in our patient. The actual cause of rupture of Glisson’s capsule is still
unknown. Many causes have been suggested, e.g., trauma or raised intra-abdominal pressure associated with vomiting as well as parturition [9-11]. We postulate that the haematoma in our patient was associated with pre-eclampsia and the severe bouts of vomiting accounted for rupture of the Glisson's capsule.

Symptoms and signs are non-specific. As illustrated in this case, this life-threatening condition was missed in favour of the commoner diagnoses of acute pyelonephritis and peptic ulceration. Other differential diagnoses quoted include acute appendicitis, cholecystitis, acute pancreatitis, pulmonary embolism, abruptio placentae and twisted ovarian cyst [8]. A high index of suspicion is clearly required and ancillary facilities such as ultrasonography, should be readily used to assist in diagnosis. It was fortuitous that fetal distress ensued, necessitating Caesarean section in this patient, thus elucidating the diagnosis. In doubtful cases, computerised tomography could be used to confirm the diagnosis as well as to determine the extent of involvement, as was the case in this patient.

Maternal mortality rates of 33% to 96% and perinatal mortality rates of 61% to 77% have been reported [8]. The importance of early diagnosis and intensive multidisciplinary approach to management to improve the overall outcome cannot be overemphasised.

Surgical intervention is recommended, and measures include hepatic lobectomy or application of haemostatic packs. Aggressive surgery has not been shown to improve outcome [8,12]. In our case it was felt that application of haemostatic packs would be appropriate and it was indeed successful. Management in the Intensive Therapy Unit postoperatively is of the utmost importance, as it is vital to detect early hepatic rebleeding, progression of DIC, circulatory collapse or sepsis. Prompt delivery, haemostasis and correction of fluid, electrolyte and acid-base imbalances may be all that are needed to control the consumptive coagulopathy, as seen here. Apart from thrombocytopaenia, our patient's coagulation profiles were within normal limits before operation, suggesting that she was only in the early stages of DIC. Nevertheless, it was felt that transfusion of fresh frozen plasma was appropriate. The use of purified human antithrombin III and low-dose heparin should be considered if the first-line treatment described above fails [3,12].

Our patient highlights the non-specific presentation of this rare but serious complication of pre-eclampsia, and the importance of a multidisciplinary approach to its management.

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References

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