Spontaneous Postpartum Subcapsular Haematoma of the Liver

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SUMMARY

A case of spontaneous postpartum subcapsular haematoma of the liver is presented. Thus far there have been 54 reported cases in the literature and this is the 17th survivor. Our case is unusual in that the diagnosis was made pre-operatively, because of the classical presentation with signs of pre-eclampsia, shock, haemoperitoneum and a mass in the upper quadrant of the abdomen.

HISTORY AND PHYSICAL FINDINGS

A 40-year-old Black para 10, was referred to our Gynaecological Unit from an outlying hospital where she had delivered a macerated, stillborn fetus vaginally, 3 days previously. On the first day after delivery she collapsed and required 3 units of whole blood. The following day she collapsed again and there was evidence of haemoperitoneum.

On arrival at the Casualty Department the patient was pale (haemoglobin concentration 8.5 g/100 ml). Her blood pressure was 180/120 mmHg, pulse rate 120 beats/minute and the urine contained 1+ of albumin. The abdomen was grossly distended with intraperitoneal free fluid, which proved to be blood on paracentesis. A palpable, tender mass associated with upper abdominal tenderness and voluntary rigidity was present in the right upper quadrant of the abdomen. Her lower abdomen was normal and the uterus freely mobile and there was no evidence of vaginal bleeding.

A diagnosis of a haemoperitoneum, caused by either a ruptured uterus or a ruptured liver, was made.

COURSE AND MANAGEMENT

The patient was sent directly from the Casualty Department to the operating theatre where she was resuscitated on the operating table prior to laparotomy. The presence of hypertension rendered central venous pressure monitoring an important guide to the blood replacement, and as soon as the central venous pressure had reached 10 cm of water, the patient was anaesthetised and a laparotomy performed. A lower midline incision was made. At laparotomy the peritoneal cavity contained about 4 litres of blood but there was no rupture of the uterus. On exploring the abdomen a large haematoma of the liver was found. In order to obtain adequate exposure the incision was extended to the upper midline. The haematoma had avulsed Glisson's capsule from virtually the whole of the right lobe of the liver, but there was no obvious rupture of the liver. The left lobe had many subcapsular haemorrhages. The blood clots were removed from the peritoneal cavity and a biopsy specimen was taken from the liver. Because of a generalised ooze from the raw area over the right lobe with no distinct laceration to be sutured, the raw area of the liver was packed with Sterispon to promote haemostasis, and two corrugated rubber drains—one in the right subphrenic space and the other in the pouch of Douglas—were left in situ, and the abdomen was closed. At this stage the patient had received 8 units of blood, the central venous pressure was 11 cm of water, the blood pressure 110/70 mmHg and the pulse rate 100 beats/minute.

Except for a slight fall in haemoglobin concentration, requiring blood transfusion on the second postoperative day, the patient made an uneventful recovery and was discharged from hospital on the tenth postoperative day and is at present well.

The histology of the biopsy specimen showed the classical features seen in the pre-eclampsia/eclampsia syndrome, as well as areas of frank haemorrhage into the liver tissue.

DISCUSSION

Subcapsular haematoma of the liver with or without an associated rupture of the liver is a rare complication of pregnancy. Abercombie reported the first case in 1844. The last recorded case is that of Owen and Kandalaft who claimed theirs to be the 53rd reported case and only the 16th survivor. The present case is therefore the 54th recorded and the 17th survivor. In South Africa only 4 previous cases, 1 of whom survived, have been reported.

The pathogenesis and clinical presentation of this condition have been adequately reviewed and do not warrant repetition here.

It is rare for the diagnosis to be made prior to laparotomy. This is only the second of the recorded cases in which the diagnosis was made clinically (the other case being that of Salzman and Mulhary). This is probably due to the fact that the patient presented in a classical
manner with signs of pre-eclampsia (the evidence of association varies from 76%\(^3\) to 81\(^4\)) sudden onset of shock and collapse, haemoperitoneum and a tender mass in the upper quadrant of the abdomen. In addition, the patient was of high parity (94% of the recorded cases have been in patients of high parity), and the age of 40 years was also in keeping; Notelovitz and Crichton\(^5\) found the average age among reported cases to be between 21 - 43 years with an average of 35 years.

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REFERENCES

Salicylate Hepatitis
A CASE REPORT

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SUMMARY

A case report of salicylate hepatitis is discussed and a predictable direct drug dosage mechanism is suggested as the pathogenesis. However, a striking eosinophilia and eosinophil infiltration of the portal tract also raises the possibility of a hypersensitivity cholestatic mechanism. Raised transaminase levels in patients on salicylate therapy appear to be a fairly frequent phenomenon which has not been widely stressed. It would seem that a sustained blood salicylate level of 25 mg/100 ml is required to cause an elevated transaminase level, and a level in excess of 30 mg/100 ml is necessary to cause actual hepatitis. Rapid reversal of the elevated transaminases occurs on cessation of salicylate therapy.


The general use of salicylate and its acetyl derivative aspirin in the treatment of rheumatic disease is testimony both to their relatively low toxicity and to their therapeutic efficacy. However, side-effects of salicylate are well known and include gastro-intestinal bleeding,\(^1\) disturbances of the blood clotting mechanism,\(^2\) water retention,\(^3\) allergic reactions in sensitive cases,\(^4\) acidosis and, occasionally, renal papillary necrosis or an increased excretion of renal tubular cells.\(^5,6\) Evidence is now accumulating which suggests that salicylates can harm hepatic function.\(^7-12\) This is not entirely surprising, because salicylate can interfere with a wide variety of metabolic processes. The following case report gives an example of salicylate hepatitis, and may assist in the understanding of the pathogenesis.

CASE REPORT

A Cape Coloured male aged 14 years was admitted to hospital on 9 April 1974 with a clinical diagnosis of acute rheumatic fever. Subsequent investigations supported this diagnosis and treatment was commenced with absolute bed rest, phenoxymethyl penicillin 500 mg orally twice daily and Disprin in an initial total dose of 3 g (administered in divided doses). The dose of salicylate was progressively increased, until by 3 May 1974 the total daily dose was 10 g. At this level the rheumatic fever process appeared to be under control, the patient's fever disappeared and the sedimentation rate began to fall.

Three weeks later (while still on 10 g salicylates a day) the patient suddenly developed a temperature of 39.5°C and complained of anorexia and nausea. A maculopapular diffuse skin rash was also noted. No change was detected in the cardiovascular system.