SUBCAPSULAR HAEMATOMA OF THE LIVER IN PREGNANCY: REPORT ON 4 CASES*

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SUMMARY

The case histories of 4 patients who developed a subcapsular haematoma of the liver with rupture of the capsule are presented. One patient survived the condition and the findings in this patient were similar to those in a case presented by previous authors. The incidence of this condition at Baragwanath Hospital is 1 in 40 000 deliveries. The diagnosis was made in one case only where the patient had a smaller rupture of the capsule than the others who died.

The patients in this report were all middle-aged multiparae who had developed toxaemia of pregnancy at about 34 weeks of pregnancy. The conclusion drawn by Mokotoff and his associates that the condition constitutes a diagnosable syndrome which should be treated by laparotomy is agreed to with reservations.

The obstetrical section of Baragwanath hospital was started in 1951, and since that time 213 719 patients have been admitted to the wards and 154 344 of these were delivered in the section. During this period, 4 patients developed a rupture of a subcapsular haematoma of the liver; an incidence of 1 in 40 000 deliveries. Of the 4 patients only 1 survived the trauma.

The case histories of these patients are presented, because they show remarkable similarities to each other and to those cases which have been reported in the literature.

CASE REPORTS

Case 1

A 38-year-old Bantu patient, para 9, who did not attend an antenatal clinic was self-admitted to the obstetric ward on 4 July 1958 complaining of marked swelling of her feet and ankles. She was obese, her legs were oedematous and the urine contained albumin 2+. Her blood pressure was 214/140 mmHg. The uterus was enlarged to the size of a 32-week pregnancy. The presentation was vertex, right occipito-anterior position. The foetal heart rate was regular at 148/min. The rest of the general examination was non-contributory.

She was given 20 mg morphine sulphate and was transferred to the antenatal ward for observation and treatment of toxaemia of pregnancy. Six hours later the resident medical officer was called to the patient because she had collapsed. On examination of the patient, the blood pressure could not be recorded, the pulse was not palpable and she was not breathing. Resuscitation was commenced but she did not respond and was pronounced dead after half an hour.

Postmortem findings. The body was that of an obese Bantu female. No signs of external trauma were present. The body was pale. The peritoneal cavity contained approximately 1 200 ml of fluid and clotted blood. The heart and lungs were pale, but were normal macroscopically. The uterus contained a foetus which weighed 1 800 g. There was no evidence of placental separation or rupture.

The liver was large and weighed 2 100 g. The entire superior surface of the right lobe was covered by a subcapsular haematoma and the edge of the capsule on the anterior surface was ruptured. The haematoma contained approximately 900 ml of dark clotted blood. On the left lobe, numerous small haemorrhages were present under the capsule. The cut surface of the liver was yellowish and soft. Numerous haemorrhagic areas were present in the parenchyma, each 2 - 5 mm in diameter.

The histology of the liver showed extensive areas of haemorrhagic necrosis, particularly on the right lobe.

Case 2

A 32-year-old patient, para 5, was referred from a municipal clinic because of severe toxaemia of pregnancy. She was admitted to the obstetric ward on 27 September 1966. She stated that she was 8 months pregnant. She did not complain of headaches, epigastric pain or visual disturbances, but had noticed that her feet and ankles had become swollen over the past 3 weeks.

She was an obese Bantu female. Her blood pressure was 210/110 mmHg. Two plus oedema of legs and ankles was noted. The urine contained albumin 2+. The uterus was the size of a 34 week pregnancy, vertex presentation, right occipito-anterior position, and the foetal heart rate was 150/min and regular. No other abnormalities were found on examination.

She was given 20 mg morphine sulphate and this was repeated when she became restless. After 3½ hours she developed a typical eclamptic fit which lasted for 2 minutes. The sedation was repeated using magnesium sulphate 20 mg intravenously. She collapsed and on examination was found to be severely shocked. Blood pressure was not recordable, and in spite of intensive resuscitative measures she died within 30 minutes.

Postmortem findings. The tissues were pale on opening the body. The peritoneal cavity contained approximately 1 200 ml fluid blood and blood clot. The blood clot was concentrated over the dome of the liver. The uterus contained a foetus estimated to be of 34-week size. There was no separation of the placenta. On macroscopic inspection the heart and lungs were normal.

The liver was enlarged and weighed 1 605 g. The undersurface was studded with multiple irregular confluent subcapsular haemorrhagic areas, and the upper surface contained similar, but larger, areas. A large haematoma 10 cm in diameter was situated on the dome of the right lobe and the liver capsule over this area had ruptured. The cut surface of the liver revealed confluent areas of haemorrhage and was pale. The histology showed large areas infiltrated with leucocytes.

The lungs were congested, the meninges of the brain were oedematous and the heart showed sub-endocardial

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haemorrhages. The remaining organs showed no abnormalities.

Case 3

A 34-year-old patient, para 4, had not attended the antenatal clinic and was self-admitted to the obstetric section on 26 September 1967. She stated that her expected date of delivery was 27 November 1967 and that she had noticed that her feet and ankles had become swollen for the past 2 weeks. She had no other complaints.

On examination she appeared healthy, but obese. Her blood pressure was 170/120 mmHg. Her urine contained albumin 2+. A slight vaginal haemorrhage was present. The foetus was presenting as a vertex, right occipito-anterior position, and was estimated to be of 34-week size. Foetal heart sounds were present and the rate was 146/min.

She was given sodium amytal gr. 3 and was transferred to the antenatal ward for observation and treatment of toxaemia of pregnancy. On 1 October 1967, at 8.55 a.m., the patient's condition deteriorated and she was found to be in severe shock, with a rapid, feeble pulse, unrecordable blood pressure and sighing respiration. Abruptio placentae was diagnosed and after resuscitative measures, a vaginal examination was performed. The cervix was soft and effaced and was dilated to 2 fingerbreadths; the membranes, which were tense, were ruptured and clear liquor was drained off.

At 1.55 p.m. the condition of the patient was not satisfactory and the senior consultant was asked to see the patient. He diagnosed a rupture of the uterus on the basis of severe shock, no external blood loss and positive abdominal paracentesis, and decided to perform a laparotomy.

At laparotomy a large amount of fluid blood and blood clot was found in the peritoneal cavity, but the uterus was intact. On palpation of the organs in the cavity a large haematoma of the liver was found. The capsule over the haematoma was ruptured and was bleeding actively. The uterus was emptied using a transverse lower-segment incision and twin female infants were delivered. The uterus was repaired and the abdominal incision was extended a further 10 cm. On inspection of the liver, the tear in the capsule was seen to extend for 15 cm on the superior and lateral border of the right lobe, and in the midline of the same lobe a smaller rupture was discovered. An attempt was made to repair the ruptures with sutures, which cut out. The area was covered with omentum and Surgicell which was sutured lightly in position. The abdomen was closed in layers after cleaning out the peritoneal cavity. The patient received 2 units of blood, 2 units of plasma and 1 litre of Ringer's solution during the operation.

Her condition remained serious and in spite of supportive measures she died 12 hours after operation.

Postmortem findings. The tissues were found to be pale. The peritoneal cavity contained about 500 ml fluid blood. The liver was enlarged and weighed 2 850 g. A large subcapsular haematoma occupied the whole of the anterior aspect of the liver on the right lobe, with smaller areas on the left lobe. Suture material with omentum and Surgicell was *in situ* over what appeared to be an extensive rupture of the capsule. The cut surface of the liver was pale.

Histology of the liver showed irregular areas of haemorrhage with coagulative necrosis. The subjacent liver tissue showed large areas of haemorrhagic necrosis involving the peripheral lobes. The sinusoids contained fibrin thrombi. The lungs were congested and the heart contained subendocardial haemorrhages.

Case 4

A 30-year-old Bantu patient, para 2, was referred to the hospital by the municipal clinic with a diagnosis of severe pre-eclamptic toxaemia and was admitted on 2 June 1970. She was not sure of her dates but thought she was due to deliver in May 1970. She did not complain of headaches or visual disturbances but had noticed swelling of her lower legs for 2 weeks, and discomfort in the right hypochondrium for 2 days.

She was obese and her blood pressure was 230/160 mmHg. The urine albumin was 2+. She had marked pitting oedema of her legs and ankles. The foetus was presenting as a vertex, right occipito-anterior position and was estimated to be the size of a 34-week pregnancy. Foetal heart sounds were present, the rate being 148/min.

The patient was not in labour and was transferred to the antenatal ward for observation and treatment of toxaemia of pregnancy. The next day at 5.25 a.m. she had what was described as a 'show' but uterine contractions could not be felt. She stated that epigastric pain had disappeared. At 6.15 a.m. spontaneous onset of labour was followed by rapid delivery of a live infant which weighed 2 500 g. The placenta was healthy and blood loss was 250 ml. At 8 a.m. the resident medical officer was called to the patient because she was complaining of pain in the region of the right hypochondrium and right flank.

Tenderness was elicited on palpation in this region but no rebound tenderness could be obtained. A tentative diagnosis of renal tract infection or cholecystitis was made and she was given sedation. The haemoglobin estimation was 8·5 g/100 ml. At 3 p.m. the registrar saw the patient and found her to be extremely tender in the right hypochondrium with marked rebound tenderness. She had not vomited at all. Her temperature was 100°F, pulse rate 140/min, blood pressure 170/90 mmHg and the haemoglobin estimation was 6 g/100 ml.

On vaginal examination the cervix was found to be closed. Excitation tenderness could be elicited. It was felt that free fluid was present in the pouch of Douglas and a colpotomy aspiration produced bright red blood. The differential diagnosis of rupture of the uterus or rupture of a subcapsular haematoma of the liver was made and the patient was prepared for laparotomy. She received 4 units of blood and further supplies were ordered from the blood bank.

At 6 p.m. the abdomen was opened and the peritoneal cavity was found to contain approximately 1 200 ml of fresh blood, both fluid and clotted. The uterus and appendages were inspected and palpated and were found to be intact. The spleen was normal, but on palpation of the

liver a haematoma was found on the inferior surface of the right lobe. The abdominal incision was extended into the upper abdomen and the liver was visualized. The haematoma which had ruptured, was sealed off with the omentum. The whole liver was inspected but no further haematomata were found. The rupture of the capsule was not bleeding actively so that the abdomen was closed after inserting drains from the bed of the liver and right iliac fossa.

The next morning at 2 a.m. the resident medical officer was called to the patient. He found that the dressings were soaked with blood. The blood pressure was 115/100 mmHg, the pulse rate 140/min and the haemoglobin level was 11·4 g/100 ml. The abdomen was distended and was tender on palpation. The drains were functioning. He re-dressed the area and gave her morphine sulphate 15 mg, and 3 units of fresh frozen plasma.

At 6 a.m. the patient was much improved and was comfortable. At 9.30 a.m. fresh blood was obtained from the transfusion service and she was given 4 units, followed by 2 units of plasma. She developed a temperature of 105·4°F at 12 noon and was found to be pale. The pulse rate was still elevated as was the jugular venous pressure. Her breathing was stertorous. She was given 0·5 mg Digoxin which was ordered for use thrice daily and then twice daily. Because her urinary output was low she was given 40 mg Lasix.

The following day she was seen by a physician who was of the opinion that her increased pulse rate was due partly to overload of the circulation and partly to the hyperpyrexia. She had basal atelectasis and possibly septicaemia and he ordered the appropriate treatment. At 9 a.m. her urinary output was 3 400 ml. Her general condition was much improved but she showed evidence of bilateral basal pneumonia. Treatment with Ceporan 1 g/6 hours was commenced.

Two days later her improvement was such that she was permitted to sit up out of bed. The attending physician now directed his attention to the hypertension which was to be treated with Aldomet.

From 10 to 15 June 1970 she continued to improve. On the 15 June, however, she showed signs of a pulmonary embolus which was confirmed by X-ray and electrocardiography, and treatment with heparin was given. Once again she started to improve and developed no further setbacks. She was finally discharged to join her baby in the unit for premature infants, and both left hospital on 11 August 1970, a little over 2 months after admission.

DISCUSSION

Mokotoff *et al*[1] reported a case of rupture of a subcapsular haematoma of the liver where the patient survived after laparotomy and repair of the liver. At the time their patient was the sixth reported survival in this condition. They reviewed the literature and found reports of 35 other cases.

The 4 cases presented here showed many similarities to each other and to the cases reported in the literature. All were multiparous women in their thirties, who had signs of toxaemia of pregnancy. All developed a state of severe shock without external blood loss to account for it. Mokotoff *et al*[1] feel that these features, together with right hypochondrial pain and vomiting, constitute a syndrome capable of diagnosis and treatment by laparotomy.

The cases presented here however, differ from the above in that only 1 patient complained of hypochondrial pain (the one who survived) and none vomited. The diagnosis in the case described by Mokotoff *et al.*[1] and in the surviving patient described here was made because a discussion on the condition had taken place a day or so before the case presented at the hospital. The condition is rare (the incidence at Baragwanath Hospital is 1: 40 000), and probably would not be seen by the average practising obstetrician in his practice. In 3 of the 4 cases presented the extent of the rupture was such that repair would be difficult if not impossible.

The constant feature in all reported cases was the involvement of the right lobe of the liver. The reason for this is not known at present. The formation of desseminated intravascular clot occurs in many conditions including toxaemia of pregnancy according to McKay,[2] and the haemorrhagic necrosis which resulted in the cases reported supports the theory of Lee,[3] who states that if the usual protective mechanism for the removal of coagulative changes is impaired, then haemorrhagic necrosis results.

Rodriquez Erdmann[4] was able to produce a Swartzmann phenomenon in rabbits by the injection of phospholipids after blocking the reticulo-endothelial system with thorotrast. The reaction was produced by generalized intravascular deposition of fibrin.

It is peculiar that in the cases presented here and in those of the literature this entity has only been reported in multiparous toxaemic women in the middle-age group. A further unusual occurrence is that of the 471 patients who have been treated at this hospital for eclampsia in 18 years, only one patient of the 56 who died as a result of this condition, developed subcapsular haematomata in the liver. The extensive haemorrhagic changes described in the 4 cases were not found in the other patients who had eclampsia.

The similarities in the surviving cases were also striking. Spontaneous delivery, multiparity, toxaemia of pregnancy and the age-group have already been mentioned. The syndrome developed 36 hours after delivery and in both the previously described case[1] and ours, the diagnosis was made because of a recent discussion of the condition. The right lobe was involved in both cases. Postoperatively both patients developed hyperpyrexia which failed to respond to the administration of antibiotics. Oliguria was not a feature of the cases. The transaminases in both cases were raised, as could be expected. The blood urea rose significantly and only returned to normal levels by the 25th day. The embolic phenomena, although occurring in different areas, occurred on similar days during the postoperative period.

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