POSTPARTUM COLLAPSE ASSOCIATED WITH SPONTANEOUS RUPTURE OF THE UTERINE ARTERY


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Postpartum collapse is always of great concern to the obstetrician—the more so when not associated with obvious haemorrhage or apparent predisposing aetiological cause. The following report is of such a case to which the obstetric flying squad at the Peninsula Maternity Hospital was called.

CASE REPORT

The flying squad was called to Mrs. N., a Coloured female aged 32. Her two previous pregnancies, in 1957 and 1961, had been normal in all respects. The history on arrival was as follows:

She had had a normal antenatal course, going into spontaneous labour at term. After a first stage lasting 20 hours and a second stage of 40 minutes, she gave birth spontaneously to a live baby boy weighing 7 lb. 4 oz. The placenta was delivered without difficulty by fundal pressure. The blood loss was normal. She remained well for 30 minutes after delivery and then suddenly collapsed. Her blood pressure fell to respond to an intravenous injection of 3 ml. of 'levophed'. The flying squad was then summoned.

The flying squad arrived at 11.50 a.m. on 29 February 1963, 1.5 hours after delivery. The findings were as follows: The patient was shocked, cold, clammy and sweaty. The systolic blood pressure was 60 mm.Hg and the pulse rate 110 per minute. On abdominal examination the uterus was found to be well contracted but slightly over to the left; she was slightly tender in the right iliac fossa over and just above the outer half of the inguinal ligament; there were no signs of a mass or of free fluid in the abdomen. On examination of the vulva there was no evidence of external bleeding. She denied having been given any cortisone in the past. She was given 100 mg. of 'solucortef' intravenously and a transfusion of group O Rh-negative blood was commenced. After she had received 400 ml. of blood her systolic blood pressure rose to 100 mm.Hg and she was transferred to the Peninsula Maternity Hospital.

On arrival, abdominal examination showed a soft tender mass in the right iliac fossa, palpable just above the right inguinal ligament and pushing the uterus to the left. On vaginal examination, a well-contracted uterus was palpated. In the right fornix, there was a boggy mass displacing the uterus over to the left. Rupture of the uterus, cervix or fornices could not be detected.

A presumptive diagnosis was made of (1) haematoma of the broad ligament or (2) a twisted ovarian cyst. A second pint of blood was begun and it was decided to observe developments. By 2.00 p.m. on the same day (29 February), the mass had increased in size, the systolic blood pressure had fallen from 115 to 100 mm.Hg and the pulse rate had risen to 120 per minute. On abdominal examination, the mass in the right iliac fossa was found to have reached the umbilicus and extended to the anterior superior iliac spine across the midline, displacing the uterus to the left. Because of the increase in size of the mass and deterioration of the patient's general condition, a diagnosis of active bleeding into the broad ligament was made and it was decided to carry out an examination under anaesthesia and laparotomy.

Under anaesthesia, the abdominal findings were confirmed and vaginal examination failed to detect any signs of trauma to the uterus, cervix or vagina.

At laparotomy, no free blood was found in the peritoneal cavity. There was a large haematoma of the right broad ligament, extending to the pelvic brim and across the uterovesical space to the left broad ligament. The right round ligament was then divided and the retroperitoneal space exposed; about 2 pints of blood clot were removed. The distal end of the right uterine artery was lying free on the lateral wall of the uterus. There was no rupture of the uterus itself, which was confirmed by a vaginal examination by an assistant while the uterus was examined intra-abdominally. Some difficulty was experienced in finding the proximal end of the torn uterine artery. The ureter was visualized at the pelvic brim and followed along the pelvic wall until the uterine artery was found crossing it. The uterine artery was blocked by blood clot and bled profusely as the clot was removed. The artery had ruptured at a point just medial to the ureter, proximal to its ascent of the lateral wall of the uterus. The rupture was clean, with no evidence of aneurysmal dilatation or thinning of the artery wall. Both the proximal and distal ends of the uterine artery were then ligated. The retroperitoneal space was drained extraperitoneally. The broad ligament was closed and the round ligament sutured. The abdomen was closed in layers.

The postoperative course was uneventful and the patient was sent home after 10 days.

HAEMATOMA OF THE BROAD LIGAMENT—AETIOLOGICAL FACTORS

Spontaneous haematoma of the broad ligament is an uncommon, but not rare, complication of pregnancy. When extensive haemorrhage occurs it may lead to secondary intraperitoneal bleeding—a serious condition with a high mortality rate.

The causes of haematoma of the broad ligament are in many cases unknown, because they are self-limiting and do not bring the patient to laparotomy or postmortem examination. The first series of cases was reported by Whitridge Williams in a monograph on subperitoneal haematoma. He found 33 cases of spontaneous haematoma of the broad ligament and ascribed them to capillary haemorrhage. Ruptured uterus is undoubtedly the commonest cause of haematoma of the broad ligament, but can hardly be considered as spontaneous. This diagnosis must, however, always be excluded.

Concealed accidental haemorrhage and pre-eclampsia toxicaemia have both been reported as associated factors. Although they are in themselves almost certainly unrelated, they both cause arteriolar spasm. Roberts' case was asso-
cated with pre-ecclamptic toxaemia, and Chassar Moir\textsuperscript{14} states that though broad-ligament haematoma may be associated with toxaemia it is usually caused by rupture of a large vein. Kenny and Doniach\textsuperscript{9} report a fatal case of broad-ligament haematoma associated with pre-ecclamptic toxaemia but brought on by coitus. In 11 cases of accidental haemorrhage, Fletcher Shaw (quoted by Feeney\textsuperscript{5}) found effusions in one or both broad ligaments extending, in 2 cases, to the lower pole of the kidney; 5 of the 11 cases survived caesarean hysterectomy. Similarly, Sheehan (quoted by Roberts\textsuperscript{12}) found 14 gross haematomata of the broad ligament in 60 postmortem examinations in deaths associated with concealed accidental haemorrhage. Chassar Moir\textsuperscript{14} states that the bleeding of concealed haemorrhage may extend into the broad ligament, probably under the influence of reduced clotting power. This is in agreement with the retrospective views of Fletcher Shaw.\textsuperscript{5} It would appear from these observations that concealed accidental haemorrhage may often be associated with haematoma of the broad ligament, the diagnosis being usually missed owing to the severe signs evident in the uterus itself.

The commonest reported cause of haematoma of the broad ligament is rupture of a utero-ovarian vessel\textsuperscript{15}—usually venous in origin. Hodgkinson and Christensen\textsuperscript{7} reviewed 72 cases of spontaneous retroperitoneal bleeding from utero-ovarian vessels up to 1951, with a mortality of 49-3\%. In labour, the mortality was 76-3\%. Jurishica and Gutglass\textsuperscript{6} reported 2 cases—one rupturing into the abdominal cavity and one causing an ileus. They felt this to be the commonest cause of broad-ligament haematoma. Conger and Paternite\textsuperscript{2} report a case of rupture of utero-ovarian vessel with secondary rupture into the peritoneal cavity. They classified rupture of utero-ovarian vessel as into (1) the peritoneal cavity or (2) the broad ligament; and (3) into the broad ligament with secondary rupture into the peritoneal cavity. This last form of haematoma, on rupturing into the peritoneal cavity, may cause an acute, and often fatal, emergency.

The explanation of rupture of the utero-ovarian vessels is partly clarified by the experimental findings of Hodgkinson\textsuperscript{15} that in pregnancy the blood flow in the ovarian vein increases 60-fold and the tension in the walls 2-4-fold.

Other causes of haematoma of the broad ligament are reported, viz. rupture of the ovarian vein above the brim of the pelvis,\textsuperscript{15} rupture of a kidney abscess\textsuperscript{14} and rupture of an aneurysm of the external iliac artery.\textsuperscript{16}

The case reported in this paper is the apparently spontaneous rupture of the uterine artery, unassociated with obstetric trauma or uterine rupture. The only other similar reported case is one of intraperitoneal rupture of a branch of the uterine artery. Ellman,\textsuperscript{17} in reviewing the English literature, found no other case of spontaneous rupture of the uterine artery.

The cause of rupture in this case is obscure. It is unfortunate that the artery was not sent for section although there appeared no abnormality on macroscopic examination. The only clue is a vague pain in the right iliac fossa during labour—its significance is obscure.

Review of the literature has shown the danger of broad-ligament haematoma. In Hodgkinson and Christensen's series\textsuperscript{7} it showed a mortality of 49-3\%. Johnston and Arban,\textsuperscript{8} in criticizing their own management of a case of rupture of a uterine varix, state that they followed the inactive therapeutic concept of watchful waiting in the face of a catastrophe which in reality carries a mortality of 49-3\%. Expectancy is dangerous and any increase in size should be treated by laparotomy. Secondary rupture into the peritoneal cavity may well be sudden and fatal. This eventuality should be anticipated, not awaited, and laparotomy performed.

CONCLUSION

The case discussed is an unusual one but demonstrates several important points in the management of the patient after delivery. All is not necessarily well after the spontaneous delivery of the baby and placenta, and care must be exercised in the immediate postpartum period.

The commonest accident at that time is relaxation of the uterus and delayed postpartum haemorrhage. Less commonly, sudden collapse may be the result of occult rupture of the uterus, inversion of the uterus, adrenal insufficiency or, as in this case, haematoma of the broad ligament.

Careful assessment by an experienced obstetrician is essential, followed by adequate resuscitation. Should operative procedure be indicated, it should be carried out after any shock that may be present has been treated and before further deterioration. The obstetrician should not be guilty of inactive procrastination in the face of a re­prieve, for the blood pressure falls and the pulse rate rises, until shock is irreversible and surgery doomed to failure.

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REFERENCES