An unusual case of uterine rupture

A case report

J. T. NEL

Summary

An unusual case of uterine rupture in a primigravida, in which the left leg of the infant ruptured through the posterior fundal part of the uterus, is presented. No definitive predisposing cause could be found. Some of the clinical signs and symptoms resembled those of abruptio placentae. Recording of internal uterine pressure revealed increased basal tone and frequent small contractions, as are often seen with abruptio placentae. This could possibly have been due to prostaglandin release from the injured decidua.

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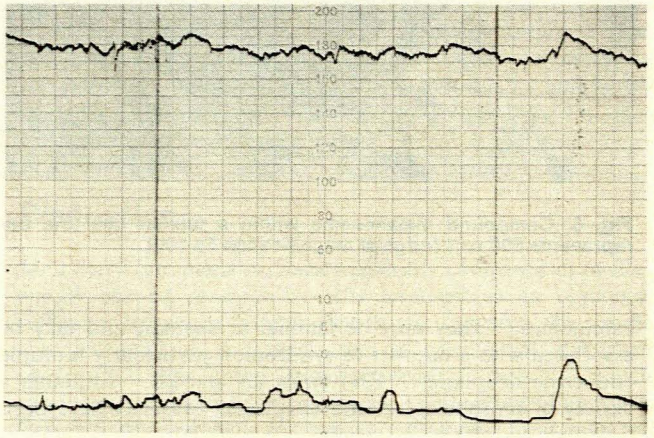

Fig. 1. Cardiotocographic pattern with fetal tachycardia and frequent, small uterine contractions.

Case report

A 19-year-old unbooked Coloured primigravida was admitted to the labour ward of Tygerberg Hospital with a history of sudden onset of acute abdominal pain. The pain had been continuously present since its onset about $2^1/_2$ hours before admission, but the patient was not aware of any contractions of labour. No history of antepartum haemorrhage or vomiting could be obtained. She did not know the date of her last menstrual period.

On examination she was found to be moderately dehydrated. Her blood pressure was 140/110 mmHg, her pulse rate 85/min, and her temperature normal. Abdominal examination revealed a hard, tonically contracted uterus of about 38 weeks' pregnancy size. The abdomen was tender, but there was no rebound tenderness. Because of the tense uterus the fetal parts were difficult to feel, but the presenting head was felt to be unengaged. The fetal heart rate was 180/min and regular.

On vaginal examination the cervix was 3 cm dilated and 60% effaced. A small amount of caput succedaneum could be palpated on the fetal head. The sagittal suture could be felt, lying in the transverse diameter. Clinical pelvimetry revealed a moderately small gynaecoid pelvis.

The membranes were ruptured and the amniotic fluid was found to be clear with no blood present. A Hewlett Packard cardiotocographic monitor was applied, with direct monitoring of the fetal heart rate and recording of internal uterine pressure. Fetal tachycardia of 180/min and frequent, small uterine contractions were present (Fig. 1). The basal tone of the uterine pressure was slightly increased.

Special examinations revealed the following: (*a*) urine — ketones negative, proteins 1 +; (*b*) haemoglobin value 13,5 g/dl; (*c*) haematocrit 41%; (*d*) blood clotting time 2,5 minutes; (*e*) platelet count 217 000/μl; and (*f*) fibrinogen degradation products 20 - 30 μg/ml.

Lateral radiographic pelvimetry revealed a biparietal diameter of approximately 8,6 cm, antero-posterior pelvic inlet of 9,9 cm, antero-posterior midpelvic measurement of 11 cm and antero-posterior outlet measurement of 10,6 cm. The sacrum was straight. A small amount of moulding of the fetal cranial bones was present, but there were no signs of severe cephalo-pelvic disproportion.

A clinical diagnosis of possible abruptio placentae was made, although the clinical picture was atypical (e.g. absence of blood in the amniotic fluid and the normal haematocrit). Although the clinical picture was not typical of this either, a threatening or definitive uterine rupture was also considered to be a possibility.

At emergency laparotomy no blood was present in the abdomen, but the infant's left leg was found to have ruptured through the left posterior side of the fundus of the uterus, the hip being tightly enclosed by a small perforation in the uterine muscle (Fig. 2). A 2 822 g female infant, with Apgar scores of 5 at 1 minute, 9 at 5 minutes and 10 at 10 minutes, was delivered by transverse lower segment caesarean section. The placenta, which was fundally inserted, was then delivered. There were no signs of an abruptio placentae.

The small area of uterine rupture of 2,5 x 1 cm was repaired in two layers with interrupted chromic catgut sutures.

The postoperative course of the patient and her baby was uneventful. The left leg of the infant was swollen on delivery, owing to pressure from the surrounding uterine muscle (Fig. 3). This oedema gradually subsided over the next few days. The dorsalis pedis pulse was always present.

The patient was again questioned about possible predisposing causes for the uterine rupture but there was no history of previous miscarriages, or of previous dilatation and curettage, or attempts at criminal abortion.

There was also no history of previous uterine sepsis or perforation.

Department of Obstetrics and Gynaecology, University of Stellenbosch and Tygerberg Hospital, Parowvallei, CP
J. T. NEL, M.B. CH.B., M.MED.(O.&G.), *Senior Specialist and Senior Lecturer*

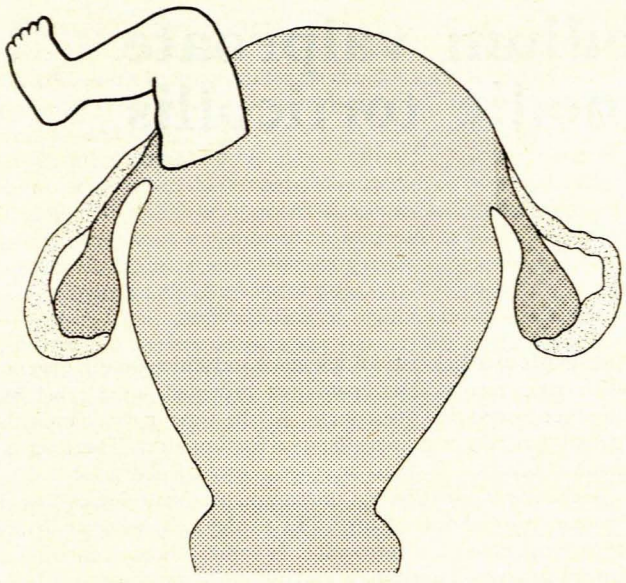

Fig. 2. A schematic representation of the infant's left leg protruding through the left posterior part of the uterine fundus.

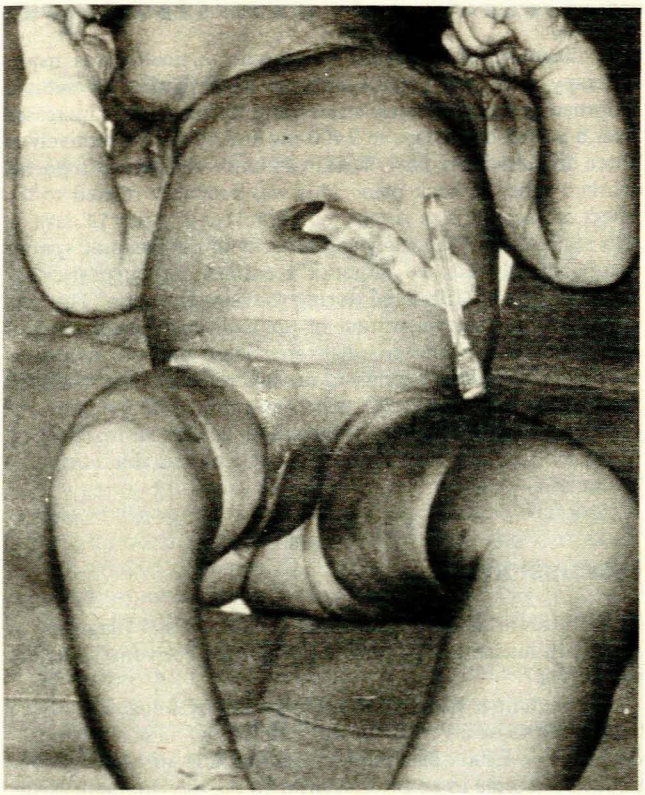

Fig. 3. Oedematous left leg caused by pressure of the uterine muscle (photograph taken on the morning after delivery).

Discussion

Uterine rupture is comparatively infrequent in the primigravida. Groen,[1] for example, reported only two primigravidas in an analysis of 144 cases of rupture of the pregnant uterus in a rural Nigerian hospital. The method of presentation of this particular case and the absence of definitive prediposing factors make it even more unusual.

Analysis of a large series of cases of uterine rupture indicates that ruptures during labour usually involve the lower segment, and those before labour the upper segment.[2] In our patient it is not clear whether the rupture had taken place before or after the onset of labour. The above findings would tend to support the first view.

The work of Dyer and Barclay[3] indicates that the weakest part of the uterus is the posterior wall of the fundus. According to the findings of Donnelly[4] and Birger,[5] the left lateral wall of the uterus is more prone to rupture than the right. The explanation for this may be that passive venous congestion develops more readily in the left broad ligament because of the 90° angle of entrance of the left ovarian vein into the left renal vein. The dextrorotation of the uterus which occurs in up to 80% of cases may accentuate this predisposition.[6] In the absence of definitive predisposing causes, the infant in our case may thus have pushed her leg through the weaker left postero-lateral part of the uterine fundus.

Lateral radiographic pelvimetry in this case could be criticized, as a laparotomy was indicated in any case, but it did serve to confirm the clinical findings of only slight cephalo-pelvic disproportion; there was very little delay as the mobile radiographic team were immediately available.

The cardiotocographic pattern is interesting; the increase in basal tone with frequent small uterine contractions is very like the pattern seen in abruptio placentae. Odendaal[7] speculates that prostaglandin release could be the cause in abruptio placentae; perhaps the same factor played a role here, prostaglandins being released from the area of torn decidua.

There may be considerable overlap of the signs and symptoms produced by abruptio placentae and by uterine rupture,[2] as is well illustrated by this unusual case.

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