

## Recurrent Rupture of Varicose Veins of Uterus in Two Successive Pregnancies

BY

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Rupture of uterine surface varicosities in pregnancy or puerperium with resultant haemoperitoneum, though well documented, is fortunately rare. The first such case was reported by Casaulon in 1778 (Whitridge Williams, 1904). Aetiology regarding venous rupture remains speculative. The vein may rupture spontaneously, without trauma or the association of labour. Pregnancy throws an increased functional demand upon the pelvic vascular system which dilates as regards the venous component in response to increased venous pressure. The co-existence of such raised venous pressure with inherent vein wall weakness may be important in predisposing to rupture. (Hodgkinson *et al.*, 1950; Finch, 1956).

The case of a patient who had intraperitoneal bleeding from ruptured uterine varicose veins in two successive pregnancies is presented.

### CASE HISTORY

The patient, a Cantonese aged 35 years, presented at the 30th week of her first pregnancy in good health. The last menstrual period began on June 14th, 1963. The examination found no significant abnormalities.

A fortnight later, on January 24th, 1964, intermittent colicky pain occurred in the right hypochondrium over a two-hour period. She felt well after resting throughout this episode. Early the following morning, pain recurred, sudden, acute and continuous with generalised spread. She became nauseated, felt faint and reached hospital gravely ill.

On admission she was anxious, pale, perspiring and in extreme pain. The blood pressure was 90/70 mm. Hg. and pulse rate 98 per minute and thready. The abdomen was distended and generally tender; maximal tenderness was in the epigastrium. Shifting dullness was present. The

uterus was soft and of the size expected. Foetal details were obscure, but the foetal heart was audible at the rapid rate of 170 per minute. There had been no vaginal bleeding and the cervix was closed.

Haemoperitoneum was suspected although the precise source of bleeding was in doubt.

After sedation with morphine and blood replacement, laparotomy was performed through a right paramedian incision. On opening the peritoneal cavity, 1,140 ml. of fresh and clotted blood was aspirated. Lower segment caesarean section was carried out and a living male child weighing four pounds was delivered. Evacuation of the uterus facilitated further exploration. An actively bleeding varicosity was present posterior to the meeting of Fallopian tube and right uterine cornu. A circular leash of varicose veins surrounded the bleeding vessel. The fundus adjacent to the right cornu contained a diverticulum where the myometrium was thin, and several veins coursed over this region. A widespread ectopic decidual reaction clothed the whole posterior uterine surface, binding the pelvic colon by adhesions to uterus, and passing laterally to involve the posterior peritoneal layer of the right broad ligament and entirely burying the right ovary in "goat's beard" adhesions. Venous oozing also occurred from numerous knots of dilated veins in the base of the right broad ligament. The placental attachment was at the fundus posteriorly but did not underlie the ruptured right cornual vein nor the sacculated portion of fundus.

A figure-of-eight chromic catgut stitch controlled the bleeding from the ruptured vein, and a continuous atraumatic stitch the area of generalised venous oozing in the right infundibulo-pelvic ligament.

The patient's condition necessitated transfusion of three pints of blood, but her post-operative progress was satisfactory. The infant died after two hours of respiratory difficulty.

A year later this patient was pregnant again. The last menstrual period began on January 17th, 1965. She remained well until the 29th week when she developed pain in the epigastrium. She was allowed home on improvement after several hours' observation in the Casualty Department. (It would have been better to have admitted her, but she doubtless did not stress her previous history.) Ten days later, on August 19th, 1965, at 31 weeks, the abdominal pain recurred and

became intense and continuous so that she was admitted to hospital.

Her general condition was poor and blood transfusion was immediately required. The abdomen was again generally distended with maximal tenderness above umbilicus. The uterus was clearly defined as a relaxed organ enlarged according to the dates. The foetal heart was heard indistinctly.

Previous experience of this patient allowed a confident diagnosis of spontaneous intraperitoneal haemorrhage from ruptured uterine varicosities. Laparotomy and Caesarean section was undertaken. A living female infant, weighing 3 pounds 14 ounces, was delivered. Free blood within the peritoneal cavity measured 560 ml. The pelvic decidual reaction and pelvic colon adherent to the posterior uterine surface persisted. The diverticulum near the right cornu was transparent, so that a foetal limb could be seen through it and the source of the acute intraperitoneal bleeding was a ruptured varicose vein in precisely the same area as previously—the right posterior cornual surface. (Fig. 1).

Partial placenta accreta existed. (Fig. 2). The attachment was once again in the posterior fundal region, and an area three inches in diameter was morbidly adherent. Continuous bleeding from the ruptured surface vein and added blood loss during attempts at placental removal occasioned deterioration of the patient and neces-

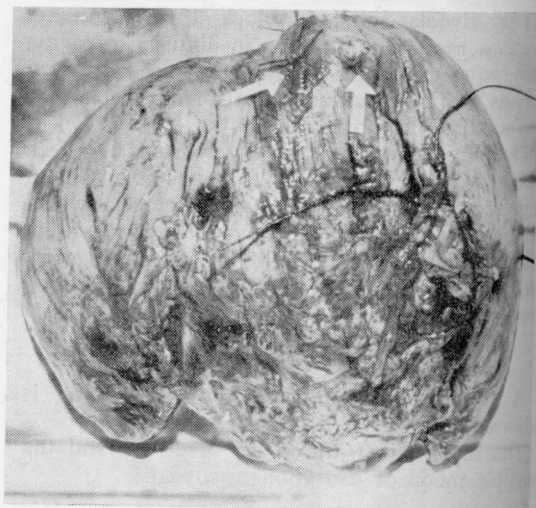


Fig. 1—Raw posterior serosal surface of uterus with diverticulum in right fundus (vertical arrow) and site of ruptured varicose vein (oblique arrow).

sitated transfusion of four pints of blood, and expeditious total hysterectomy was therefore performed.

The patient thereafter progressed satisfactorily. The premature baby died on the third day.

#### DISCUSSION

The present patient is interesting in having had a rare complication in two successive pregnancies. In the first pregnancy she also had bleeding from a vascular decidual reaction covering the right infundibulo-pelvic ligament. Precisely the same site of uterine vein rupture was recorded by Marrow (1960) but the posterior uterine wall is not the commonest site of venous haemorrhage in pregnancy.

Finch's review in 1956 found 78 per cent. of such venous bleeding to be from broad ligament varicosities, 19 per cent. from the posterior uterine surface and three per cent. from the anterior surface of uterus.

Fatal haemorrhage near term has also occurred from an area of isolated decidual tissue on the lateral pelvic wall in proximity to the pouch of Douglas (Doyle *et al.*, 1957), from decidual tissue in the ovary (Reid, 1965) and from a vascular decidual reaction involving greater omentum (Melody, 1950). The ectopic decidual reaction seemed a likely recurring factor in the aetiology of bleeding in this patient—its macroscopic extent and obvious vascularity were outstanding.

In both this patient's pregnancies, warning abdominal pain occurred before final pain,

haemoperitoneum and collapse. Minor bleeding from the culprit vein probably heralded vein wall rupture and free intraperitoneal flow. The view that Braxton-Hicks contractions control bleeding from ruptured uterine surface veins temporarily may explain initial pain as being due to small subperitoneal uterine haematomata. Final pain follows the breakdown of covering visceral peritoneum and resultant parietal peritoneal irritation by blood contamination (Jurishica *et al.*, 1955).

At the first emergency in this patient, although haemoperitoneum was diagnosed, the precise cause of the bleeding was in doubt. The pain she experienced was upper abdominal and tenderness maximal in the epigastrium. Recent authors stress rupture of a splenic artery aneurysm as the best-known source of such bleeding in pregnancy (Furler *et al.*, 1962; Hanna *et al.*, 1964).

The sacculated diverticulum in the patient's uterus and the placenta accreta in the second pregnancy, both rare occurrences, contributed to making this patient something of an obstetrical curiosity.

#### SUMMARY

1. Spontaneous intraperitoneal bleeding from ruptured surface uterine veins complicated two successive pregnancies in a Cantonese patient.
2. Management in the first pregnancy was by caesarean section and vein ligation and in the second by caesarean-hysterectomy for the added complications of partial placenta accreta and weakening of the fundus by an excessively thin walled sacculatum.
3. The role of the ectopic decidual reaction in the causation of intraperitoneal bleeding in pregnancy is stressed.
4. A possible pathological course of events is described for rupture of uterine surface varicosities to explain initial minor abdominal pain and final generalised pain and collapse.

#### REFERENCES

- DOYLE, G. B. & PHILLIPS, D. L. (1957). *J. Obstet. Gynaec. Brit. Emp.*, **64**, 270.
- FINCH, T. V. (1956). *Amer. J. Obstet. Gynec.*, **72**, 1,189.
- FURLER, I. K., ROBERTSON, D. N. S., HARRIS, H. J. & PRYER, R. P. L. (1962). *Lancet*, *ii*, 588.
- HANNA, W. A., & MYLES, T. J. M. (1964). *Brit. med. J.*, *i*, 1,024.
- HODGKINSON, C. P. & CHRISTENSEN, R. C. (1950). *Amer. J. Obstet. Gynec.*, **59**, 1,112.
- JURISHICA, A. J. & GUTGLASS, M. (1955). *Obstet. and Gynec.*, **6**, 315.
- MARROW, A. E. (1960). *J. Obstet. and Gynaec. Brit. Emp.*, **67**, 792.

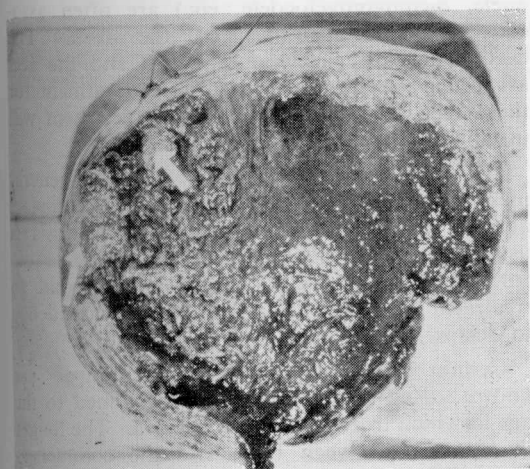


Fig. 2—Interior of uterus, with diverticulum (upper arrow) and partial placenta accreta (peripheral arrow). Haemostatic catgut sutures are seen at site of ruptured surface varicosity near right cornu.

- MELODY, G. F. (1950). *West. J. Surg. Obstet. Gynec.*,  
58, 460.  
REID, S. M. (1965). *J. Obstet. Gynaec. Brit. Cwlth.*,  
72, 634.  
WILLIAMS, J. WHITRIDGE (1904). *Amer. J. Obstet.*, N.Y.,  
1, 442.

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