SPONTANEOUS RUPTURE OF THE UTERUS DUE TO PLACENTA PERCRETA

BY

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IN the normal process of implantation of the ovum, the invasion of the trophoblast is limited by the response of the decidua. Wislocki, Dempsey and Fawcett (1948) have shown that the decidua contains a muco-polysaccharide by virtue of which the trophoblastic erosion is checked, the villi penetrating only as far as the spongy layer. It is through this layer that the placenta separates during the third stage of labour. Placenta accreta is the pathological condition in which this line of cleavage is absent—the villi being attached to the deep compact layer of the decidua in the case of placenta accreta vera and to the muscular layer of the uterine wall in the case of placenta increta, or have penetrated to the serosal coat in placenta percreta. Aaberg and Reid (1945) further subdivided these types into complete, partial or focal, according to whether the whole surface of the placenta, one or several cotyledons or only part of one cotyledon is adherent respectively.

The literature of spontaneous rupture of the uterus due to placenta percreta is here reviewed and a case report is presented of rupture due to partial placenta percreta.

INCIDENCE

Spontaneous rupture of the uterus due to placenta percreta is very rare. The case here presented is the only one that has occurred at Edgware General Hospital and Bushey Maternity Hospital, London in 40,249 deliveries between 1934 and 1954. No case has been recorded at Queen Charlotte's Hospital, London in 103,356 deliveries, between 1908 and 1954. Fitzgerald, Webster and Fields (1949) reported 1 case in 92,232 in Cook County Hospital, Illinois, between 1928 and 1948.

REVIEW OF THE LITERATURE

The first description that I have been able to find of spontaneous rupture of the uterus, proved microscopically to be due solely to placenta percreta, was published by Alexandroff in 1900, and since that time only 8 other cases have been reported (Bortkewitsch, 1911; Kratochvil, 1922; Dietrich, 1922; Bakanow, 1928; Pettit and Mitchell, 1949; Fitzgerald, Webster and Fields, 1949; McCarthy and Nichols, 1950; Schuyler, 1952). In addition, Krahulik (1952) and Lehmann (1925) each reported one case of spontaneous rupture of the uterus due to placenta percreta in congenitally abnormal uteri. Mr. Gibberd had under his care at Queen Charlotte's Hospital in 1948 a case of uterine rupture where the placenta had penetrated an old Caesarean scar, and a similar case was reported by Stone, Donnenfeld and Tanz (1954). These cases, however, are not included in the discussion, as the spontaneous rupture cannot be claimed as solely due to placenta percreta.

CASE REPORT

Mrs. E. Q., aged 43 years, was pregnant for the second time. Her first child was delivered in 1948 by lower segment Caesarean section after a trial of labour lasting 22 hours, because of uterine inertia and extended breech presentation. External cephalic version at 34 and 36 weeks had failed. The child weighed 7 pounds 6 ounces at birth and is alive and well. The temperature rose to 100·4° F. on the second and third day after operation, and at that time a heavy growth of *Bacterium coli* was obtained from vaginal swabs. A course of Sulphathiazole was given. The abdominal wound healed well. She was discharged from hospital with the child 14 days after delivery, and told that any future confinements should be in hospital.

On 10th January, 1953, the emergency obstetric service was called to her home by her doctor who had found her semi-conscious. Her last menstrual period had

been in August, 1952. She had been well until 2 days before, when she had complained of abdominal discomfort, with tenderness especially in the umbilical region. Four hours before her doctor had seen her she complained of colicky abdominal pain and a desire to defaecate. At stool she obtained some relief. A further attack of colicky abdominal pain occurred ¾ hour later; it became more severe until finally 1½ hours later she had severe and continuous pain. She then complained of nausea and vomited twice. She complained of faintness and was noticed to be pale.

The doctor was called, but unfortunately could not be reached for an hour. He found the patient semi-conscious, extremely pale with a rapid feeble pulse and called the emergency obstetric service immediately.

When the obstetric service arrived the patient was cold, pale and restless. The radial pulse was hardly discernible and the blood pressure unrecordable. She groaned feebly when the abdomen was touched, but there was no guarding. The uterine outline could not be felt. There was no vaginal bleeding.

A diagnosis of intraperitoneal bleeding was made, morphia gr. 1/6 intramuscularly was given at once and a transfusion of one pint of Group O Rhesus negative blood was given in about 20 minutes. Little improvement resulted and a second pint of blood was given during transference to hospital. On reaching hospital an additional blood drip was started and endotracheal oxygen given. There was no improvement and death occurred 20 minutes after admission.

Post-mortem Findings

The peritoneal cavity contained 3 pints of blood. The uterus was of a size consistent with 20-weeks pregnancy. At the fundus was a large subperitoneal haematoma, diameter 5½ inches, in the centre of which was a perforation 3 inches in diameter through which placental tissue was protruding. The scar of the previous lower segment Caesarean operation was not identifiable. The uterus was opened anteriorly by a mid-line incision, and was found to contain a 20-weeks mature female foetus. The placenta was large, and symmetrically attached over the upper half of the uterus, with the cord arising centrally. At its lower edge the placenta was easily separated, but as the area of rupture was approached there was no apparent plane of cleavage. Histological examination of sections taken near the edge of the perforation showed in some areas no evidence of decidual tissue or myometrium, the chorionic villi having penetrated to the peritoneal coat. In other areas there was no decidual tissue and here the chorionic villi were seen to have invaded the myometrium, which showed hyaline degeneration. In some areas near the perforation a thin layer of decidua separated the villi from the myometrium.

The condition was therefore one of partial placenta percreta.

Discussion

All the cases that have been described of spontaneous rupture of the uterus due to placenta percreta have been multigravidae of widely differing ages, the youngest being 21 years (Fitzgerald, Webster and Fields, 1949), the oldest 43 years (the case here recorded) and except for these 2 cases the child was viable. No indication of the possible aetiological factor was given in 2 instances (Fitzgerald, Webster and Fields, 1949; Alexandroff, 1900) but in 8 cases there was a history of previous uterine sepsis or features, such as manual removal of the placenta or curettage, which were almost certainly accompanied by some degree of infection. In 2 cases (Dietrich, 1922; Bortkewitsch, 1911) no history was given of abnormal symptoms during the pregnancy, in 8 the presence of abdominal pain was recorded. Pettit and Mitchell (1949) and Schuyler (1952) record that their patients had menorrhagia prior to the pregnancy. No menstrual abnormality is recorded in the remaining 8 cases. Rupture occurred at the fundus uteri in 7 out of 10 cases, the exceptions being those of Bortkewitsch (1911), where the site was the posterior wall of the corpus, Bakanow (1928), the posterior wall of the lower segment, and Kratochvil (1922), who does not indicate the exact situation in the corpus. Five cases were complete placenta percreta and 5 partial, there being no apparent relationship of the degree of involvement to age, parity, stage of gestation, situation of the placenta or the aetiological factors concerned. In 2 instances, only, was the condition fatal to the mother, the case here described, who was moribund on admission to hospital, and that of Dietrich (1922) on whom vaginal hysterectomy was performed. The remaining 8 cases were treated by laparotomy, 7 by hysterectomy and 1 (McCarthy and Nichols, 1950) by local excision of the perfora-

tion and ligation of the Fallopian tubes with conservation of the uterus. In both fatal cases, however, there was some delay before treatment could be instituted. The child survived in 2 cases only (Bakanow, 1928; McCarthy and Nichols, 1950). In 8 cases the decidua was found microscopically to be either absent or deficient, in 2 (Fitzgerald, Webster and Fields, 1949; Schuyler, 1952) there was no mention of the decidua. Hyaline degeneration of the myometrium was noted in 4 cases (Dietrich, 1922; Bakanow, 1928; Schuyler, 1952; present report).

Conclusions

(1) Multiparity and intra-uterine sepsis, rather than trauma alone, are the main aetiological factors of placenta percreta.

(2) The depth of penetration of the chorionic villi and degree of involvement of the placenta depends neither on the age of the patient nor the stage of the pregnancy.

(3) Decidual deficiency is the basic lesion in all cases of placenta accreta.

(4) Abdominal pain during pregnancy associated with a history of uterine sepsis should raise in the obstetrician's mind the possibility of placenta accreta and hospitalization should be insisted upon.

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