

## RUPTURED UTERUS DUE TO PLACENTA PERCRETA

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INVASION of the uterine surface by the chorionic villi is limited by protective elements in the decidua basalis. Wislock<sup>1</sup> has shown the presence of a mucopolysaccharide in the decidua that serves to limit trophoblastic erosion of maternal tissue. Microscopic sections from most cases of placenta accreta show an absence of the decidua basalis and atrophy of the decidua vera. The degree of invasion of the chorionic villi whether superficial, deep or entirely through the myometrium has given rise to the terms accreta, increta and percreta. Irving and Hertig<sup>2</sup> in 1937 recorded the most complete survey of placenta accreta (eighty-six cases including eighteen personal cases). The authors' findings showed that in over three-fourths of the cases the decidua, which serves as a line of cleavage for the separation of the placenta, was compressed and contained few or no glands. In almost all cases the chorionic villi could be seen invading the myometrium. The first recorded case of rupture of the uterus due to placenta percreta was that by Alexandroff<sup>3</sup> in 1900. In his case a hysterectomy was performed and the patient lived. A review of the literature by Hertig and Irving up to 1937 covering eighty-six cases of placenta accreta showed only four instances of spontaneous rupture due to chorionic villi penetrating the visceral peritoneal surface of the uterus. The case reported herein illustrates such a condition.

## CASE REPORT

N. B., a white married female aged twenty-six years, gravida III, Para I, was admitted with a history of having been awakened at 6 A.M. on May 25, 1949, by a knife-like pain in the epigastrium and an inability to breathe.

The patient's previous history was relevant to the case. She had had a miscarriage after a pregnancy of three months in 1943, with retention of placental elements for which a curettage was done three months later. She had abnormal menses with intermenstrual bleeding thereafter and abdominal pains until

1945 when she became pregnant. This resulted in a normal delivery in 1946 with retention of a portion of the placenta. She bled for one month and a curettage was done. The record described the curettings as a "large amount of soft bloody material but no placental elements could be recognized." Diagnosis was subinvolution of the uterus with chronic infection.

In the present pregnancy her expected date of confinement was May 26, 1949. She was admitted in threatened premature labor on April 16, 1949. Her complaint at this time was upper abdominal pains but no contractions were observed. The fetal heart was found to be good. After morphine and bed rest she was discharged in thirty-six hours.

At her admission on May 25, 1949, she was seen within ten minutes of her arrival and within twenty minutes of the onset of symptoms. She presented a picture of increasing shock, cyanosis, blood pressure of 70/50 and pulse of 120 with poor volume. The abdomen was board-like above the bulge of the uterus. The fetal heart could be heard faintly. Glucose in saline and one unit of plasma were given and suitable donors were obtained within one hour. She was taken to the operating room at 7:40 A.M., one hour and twenty-five minutes after admission. In spite of the intravenous fluids, plasma and 400 cc. of blood her blood pressure never rose above 70/50. On opening the peritoneum pure unclotted blood not diluted with amniotic fluid was released under great pressure. The right cornual region of the fundus showed a clear punched out area larger than a silver dollar through which a lobe of the placenta was protruding. A recently deceased fetus was extracted through a low fundal incision and the placenta was peeled off easily except for an area about 1 inch wide around the perforation. This 1 inch border was excised to get back to thicker myometrium and both this excised portion and the cesarean incision were repaired with three rows of continuous chromic No. 0 catgut. Despite 4 pints of blood given under oxygen pressure for rapid delivery

the patient's condition on the operating table was considered too weak for hysterectomy. The tubes were hastily ligated and the peritoneum closed. Her condition, although shaky for forty-eight hours, steadily improved until her discharge on the eighth postoperative day. She was seen at home on June 10, 1949, feeling well.

Frozen section revealed fibroblastic proliferation and round cell infiltration in the margin of myometrium examined. The decidua basalis could not be seen.

#### ETIOLOGY AND TREATMENT

An etiologic background had been proven in only six of Irving's personal cases. Previous retained placentas, endometritis and old cesarean scars were thought to lead to an atrophied decidua basalis. Some authors have indicted the endocrines, particularly the corpus luteum, for the failure of the decidua. In the case presented there may well have been either penetration of the uterine wall by the curette or endometritis following the miscarriage in 1943. Also significant was the retention of a portion of the placenta after delivery in 1947. The frequent association of placenta accreta with placenta previa points toward previous endometritis as an etiologic factor.

In spite of the good results with hysterectomy recorded by some of the earlier authors manual removal still held sway as the treatment for placenta accreta up to 1925 with a high mortality (66 per cent in thirty cases), whereas hysterectomy employed in Irving and Hertig's eighteen cases showed less than 6 per cent mortality. Obviously the type of

case will dictate the treatment. In cases in which there is accreta partial separation is the rule and external bleeding is present. Failure to find a line of cleavage in attempted manual removal should lead to the diagnosis; hysterectomy is the surest cure. In placenta increta and total accreta which is rarer and when there is no external bleeding many obstetricians (after John O. Polak's memorable case in 1916) have let the placenta remain for days or even weeks to have it slough out. With the still rarer placenta percreta one has no choice but to operate.

#### SUMMARY

A discussion of placenta accreta, increta and percreta is presented. A case of the latter with spontaneous uterine rupture is given. An etiologic theory is offered through the experiments of Wislocki and the recorded data of Irving. The etiology of either infection or trauma causing endometrial damage seems logical in the case presented herein. Treatment of the various types is also summarized.

#### REFERENCES

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