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PLACENTA PERCRETA INVADING BLADDER

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The abnormal attachment of chorionic villi to myometrium with the absence of a normal cleavage plane is called placenta accreta and has 3 degrees of severity: 1) placenta accreta vera in which the villi are directly juxtaposed to the myometrium but do not actually invade, 2) placenta increta in which the villi invade the myometrium but not the serosa and 3) placenta percreta in which the villi penetrate the myometrium completely.

All degrees of placenta accreta are thought to be caused by a local absence of the decidua basalis which normally poses a barrier to placental invasion. This defect is frequently associated with previous cesarean sections or uterine trauma, endometritis or dilatation and curettage.[1-3]

Placenta accreta vera and placenta increta lead to incomplete placental removal after birth, often with persistent postpartum bleeding. Placenta percreta with rupture of the uterus at any time during the pregnancy is one of the most urgent obstetric catastrophes and results in massive blood loss and high mortality.[4-8]

Placenta percreta directly invading the bladder has never been reported in the urological literature and we know of only 2 documented cases in the English literature.[9, 10]

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CASE REPORT

S. B., 479926, a 29-year-old Para 4, gravida 5 woman, was first seen on July 2, 1971 for sudden gross hematuria in the twenty-sixth week of pregnancy. Her first pregnancy had resulted in a full-term vaginal delivery. The second pregnancy required an emergency cesarean section for fetal distress and the baby was born dead. The third and fourth pregnancies were delivered by cesarean section and were otherwise unremarkable. Following the fourth pregnancy she was placed on birth control pills until November 1970, after which she soon became pregnant. The pregnancy was completely unremarkable until the episode of hematuria. She had no previous urinary infections, flank pain, dysuria or voiding difficulties. There was no history of cyclic hematuria or urinary incontinence.

Physical examination was normal except for an enlarged uterine fundus consistent with a 26-week pregnancy and a well healed lower abdominal vertical scar from previous cesarean sections. Coagulation studies were normal except for a platelet count of 100,000 per cu. mm. and a somewhat shortened euglobulin lysis time (normal for pregnancy). Hematocrit was 22 per cent (last previous hematocrit was 36 per cent) and serum creatinine was 0.9 mg. per cent. An excretory urogram (IVP) revealed moderate right hydroureter consistent with a 7-month pregnancy but was otherwise normal (fig. 1). Cystoscopy on July 2 revealed massive clots. However, inspection of the bladder after thorough irrigation revealed no bleeding and no abnormal findings. The patient was given 4 units of blood and the urine remained clear for 5 days. When hematuria recurred, she received 2 more units of blood. An arterial bleeding point was cauterized on the posterior wall of the bladder slightly to the right and directly over the uterine fundus. The urine remained clear and she was discharged from the hospital. On July 18 intermittent hematuria recurred and she was rehospitalized. On July 19 cystoscopy revealed an arterial bleeder in the same area and there was a granular appearance to the bladder mucosa in this area. The bleeder was cauterized and the urine

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[10] Grabert, H., Mossa, A., Oliveira, S. F., Hutzler, I., Fainzielber, S. and Rodrigues de Lima, G.: Placenta percreta with penetration of the bladder. J. Obst. Gynaec. Brit. Comm., 77: 1142, 1970.

FIG. 1. IVP consistent with 7-month pregnancy.

remained clear until July 25 when massive hematuria recurred. More arterial bleeders were found in the same area and cauterization failed to stop the bleeding.

At emergency laparotomy the posterior bladder wall was found to be massively invaded by placenta. The scar from the previous section was ruptured and only the peritoneal reflection was holding the uterus together. A cesarean section was performed and a 3-pound boy was delivered who died 12 hours later. A total hysterectomy was performed. Bleeding occurred from nearly every point imaginable in the pelvis. Vascular clamps were applied to both internal iliac arteries. Despite the excellent collaterals below them, the maneuver seemed to slow the bleeding and accordingly, they were ligated. However, it was virtually impossible to control bleeding until the uterus was removed. For several significantly long periods during the procedure the blood pressure and pulse were unobtainable. A total of 46 units of blood was given. Ten of these units were not cross-matched.

There was considerable placental involvement of most of the bladder and hemostasis could be obtained here only by suturing the anterior edges of the bladder wall to the skin and packing the inside of the bladder. A similar hemostatic pack was placed in the vagina for counter pressure.

Microscopic examination of tissue removed from inside the bladder disclosed mature placenta with well formed chorionic villi and fragments of necrotic smooth muscle with decidua on 1 surface (fig. 2). Within the endometrium there was abundant degenerating decidua. Sections along the anterior uterine defect showed a layer of necrotic chorionic villi and entrapped decidual cells extending to the serosal surface of the uterus (fig. 3).

The postoperative course was turbulent. Atelectasis and poor ventilation related to prolonged anesthesia were treated with a nasotracheal tube and ventilator for 4 days. The packs in the bladder and vagina were removed 3 days postoperatively. The urine remained relatively clear and was allowed to flow freely from the stoma. There was persistent sepsis and fever between 102 and 104F, right upper quadrant fullness and tenderness and an indurated large non-fluctuant pelvic mass. Blood culture yielded Escherichia coli and Pseudomonas. IVP demonstrated no change. An unusual form of disseminated intravascular coagulopathy developed with normal platelet counts but severely prolonged prothrombin time and partial thromboplastin time. However, this was controlled with intravenous heparin.

At 9 days postoperatively upper gastrointestinal bleeding developed that required 3 units of blood during 12 hours. The patient underwent pyloroplasty and vagotomy. Multiple eroded areas typical of a stress ulcer were noted. A thickened inflamed gallbladder was noted and a cholecystostomy was performed. Purulent collections were noted in the lower abdomen and pelvis and these were drained.

The patient did well and heparin was discontinued without recurrence of coagulopathy. The vesicostomy tract healed 4 weeks later and drains were slowly advanced and removed. The patient was moderately febrile until this time but eventually she remained afebrile without antibiotics. She voided well and the urine was uninfected. A cholecystogram revealed complete obstruction of the cystic duct by a 1 cm. non-opaque gallstone. Three months later she underwent an uneventful cholecystectomy.

DISCUSSION

The reported incidence of placenta accreta varies from 1 in 2,000 pregnancies to none in 70,000 pregnancies.[4, 5, 11] This discrepancy is probably owing to the fact that microscopic examination of the placenta after manual removal is not routinely done and the diagnosis is often overlooked.[6, 12]

[11] Novak, E. R. and Woodruff, J. D.: Gynecologic and Obstetric Pathology with Clinical and Endocrine Relations, 6th ed. Philadelphia: W. B. Saunders Co., p. 510, 1967.

[12] Eastman, N. J. and Hellman, L. M.: Williams' Obstetrics, 13th ed. New York: Appleton-Century-Crofts, Inc., p. 949, 1966.

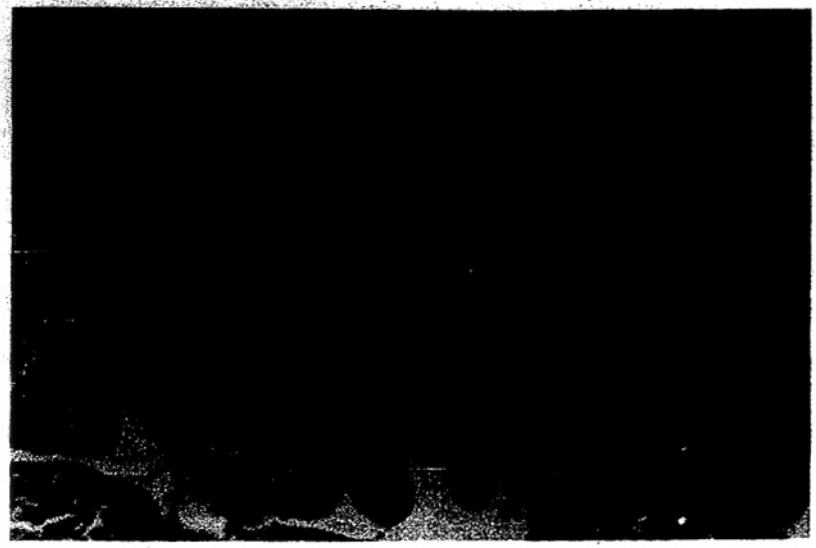

FIG. 2. Well-formed chorionic villi found to extensively invade bladder.

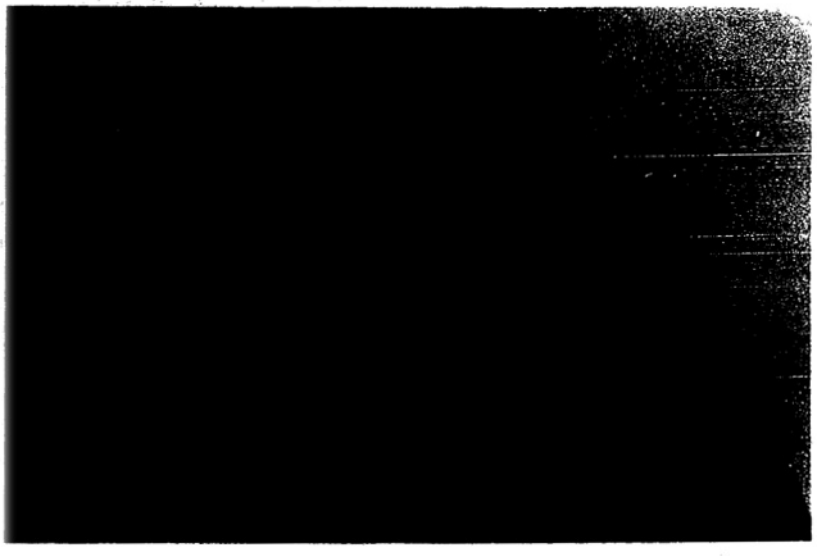

FIG. 3. Margin of defect in uterus shows normal layer of decidua basalis.

Since Irving and Hertig's classic study of 18 cases, prompt hysterectomy has generally been considered the safest way to manage placenta accreta vera or placenta increta. They reported a 66 per cent mortality if manual extraction was performed or if the placenta was left in after delivery. They had no mortality with prompt hysterectomy.[4] However, McKeough advocated more conservative management of placenta accreta (as one would manage an abdominal pregnancy). This included leaving the placenta in the uterus after delivery, treatment with antibiotics and performing hysterectomy only if bleeding persisted.[13]

There is less controversy regarding aggressive

[13] McKeough, R. P. and D'Errico, E.: Placenta accreta, clinical manifestations and conservative management. New Engl. J. Med., 245: 159, 1951.

management of uterine rupture. Since 1900 fewer than 40 cases of uterine rupture from placenta percreta have been reported. In all cases of uterine rupture from placenta percreta not associated with bladder invasion, prompt laparotomy was performed for an acute abdomen and hysterectomy was not technically difficult. Decidua basalis was lacking and previous cesarean sections or dilatation and curettage were implicated as a cause in most instances.[2, 5, 7, 8, 14]

There are only 2 documented cases of placenta percreta with bladder invasion. Both cases manifested at a much earlier stage of pregnancy than our case and operative management was simpler. One case was reported as "an example of initially inadequate diagnostic pursuit of a rare involvement".[9] The patient had a history of 6 cesarean sections. She had gross hematuria and mild lower abdominal pain in the fourth month of pregnancy. Although she required 11 pints of blood, an IVP was normal and the urine cleared after all clots were removed from the bladder. Cystoscopy initially revealed no source of bleeding and the pelvic examination was normal for a 4-month pregnancy. Cystoscopy was done again for recurrent bleeding and biopsy of a granular lesion at the dome of the bladder produced massive bleeding. Only at laparotomy was the diagnosis made and the patient was managed with 10 units of blood, cesarean section, hysterectomy and segmental bladder resection. The placenta was attached to the old cesarean section scar in the lower uterine segment and invaded the bladder posteriorly.

The other case of placenta percreta with bladder invasion was heralded by dysuria and gross hematuria in the sixteenth week of pregnancy in a patient with 4 previous cesarean sections.[10] Cystoscopy revealed an "exophytic growth" on the posterior wall and trigone. Laparotomy revealed invasion of the placenta into the posterior bladder wall. A large blood clot (1,000 cc) was removed from the bladder but no active bleeding was found. A subtotal hysterectomy was performed with an uneventful recovery. Microscopic examination demonstrated invasion of uterine muscle by trophoblastic cells and chorionic villi in the bladder.

In our patient initial cystoscopy and IVP were normal. The advanced pregnancy of 7 months made it difficult to justify termination and hysterectomy although the correct diagnosis was suspected. The good results obtained initially by fulguration of the bleeding point on the posterior wall of the bladder caused us to underestimate the severity of uterine penetration and produced hope that cesarean section could be delayed until a viable infant was possible.

It is clear now that in these cases temporizing can be disastrous and that early interruption of a pregnancy is essential. Wilson and associates reported that surgical patients who require transfusion of more than 25 units of blood in 24 hours have a mortality rate of 95 per cent.[15] It is possible that at an earlier stage of pregnancy, the placenta could have been left in place and treated as in an abdominal pregnancy. Even in the absence of that alternative, with earlier operative intervention we might have found less massive placental attachment to surrounding structures and a simpler procedure with less morbidity would have been possible.

The presence of a normal decidual layer in our patient is contrary to other reports. The previous cesarean sections may have resulted in a defect in the entire uterine wall rather than in the decidua basalis only, especially if portions of bladder had been inadvertently included in the uterine suture line. However, the absence of incontinence or cyclic hematuria prior to this pregnancy weighs against a true vesicouterine fistula.[16, 17] The presence of intact decidua basalis weakens previous assumptions that placenta percreta is caused simply by absence of a barrier layer of decidua.

SUMMARY

A rare case of placenta percreta invading the bladder wall is reported. When hematuria occurs during pregnancy in a patient with a previous cesarean section, placenta percreta should be suspected. Aggressive management of this condition is recommended. Histologic data are presented to question the concept that placenta percreta is caused simply by a defect in the decidua basalis.

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[15] Wilson, R. F., Bassett, J. S. and Walt, A. J.: Five years of experience with massive blood transfusions. J.A.M.A., 194: 851, 1965.

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[17] Falk, H. C. and Tancer, M. L.: Management of vesical fistulas after cesarean section. Amer. J. Obst. Gynec., 71: 97, 1956.