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CASE REPORT

PATHOLOGY/BIOLOGY

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A Death Due to Subinvolution of the Uteroplacental Arteries

ABSTRACT: A 19-year-old G1P0 Caucasian woman died 8 days postpartum because of the subinvolution of the uteroplacental arteries. Microscopic examination of the placental implantation site revealed large, dilated spiral arteries containing partially occluding thrombi in the superficial myometrium. The presence of cytotrophoblasts within and surrounding the spiral arteries was confirmed by low molecular weight cytokeratin immunohistochemistry. Infection of the Cesarean section incision site was demonstrated by the growth of *Staphylococcus aureus* and *Streptococcus agalactiae* Group B in the wound as well as the intrauterine blood clot. Although subinvolution of the placental site may be a cause of delayed postpartum hemorrhage and significant morbidity, the underlying pathophysiologic mechanism is unknown.

KEYWORDS: forensic science, subinvolution, postpartum hemorrhage, uteroplacental arteries

Case Report

A 19-year-old G1P0 Caucasian woman underwent a Cesarean section 8 days prior to her death. The estimated blood loss at delivery was 400–500 mL. At a follow-up postpartum appointment 7 days after delivery, she was discovered to be severely anemic with a hemoglobin of 6.7 and a hematocrit of 20.1 with a red blood cell (RBC) count of 2.4 M/μL, a mean corpuscular volume of 83.8, and an RBC distribution width of 15.3 (normocytic normochromic anemia). She received no transfusion and the following day collapsed at home. The husband brought her to the emergency room pulseless and without respirations, and advanced cardiac life support was unsuccessful.

An autopsy was performed and revealed a Caucasian woman measuring 62 inches and weighing 170 pounds with pallor of her conjunctivae, mucous membranes, and internal organs. The abdomen was protuberant, and a 17-cm horizontal C-section healing incision was present with dehiscence of the lateral margins but without erythema or purulent discharge. The uterus was enlarged weighing 520 g and measuring 19.0 × 12.0 × 3.5 cm, and a sutured surgical incision was present on the low anterior wall of the uterus with attached blood clot (Fig. 1). The placental implantation site was along the superoanterior wall with an attached 5.5 × 4.5 × 3.5 cm blood clot, and several small detached blood clots were present in the uterine cavity. The endometrium was sloughed, and the boggy myometrium contained prominent vasculature (Fig. 2).

Microscopic examination of the sections taken from the placental implantation site revealed dilated and thrombosed vessels within the upper (superficial) and mid myometrium with fibrinous material within vessel walls (Fig. 3). Scattered cytotrophoblasts were noted in the myometrium and myometrial vessels and highlighted by a low molecular weight cytokeratin immunohistochemistry stain (Fig. 4). There was also necrosis and acute inflammation of the superficial myometrium and endomyometrial junction with some attached inflamed decidua. No retained placenta or placenta accreta was noted on multiple sections. Microscopic sections of the blood clot attached to the placental implantation site and within the uterine cavity also contained inflamed necrotizing decidua.

The C-section incision on the skin revealed fibrosis with foci of acute inflammation, necrosis, and abscess formation. Cultures of this incision and the uterine blood clot were significant for *Staphylococcus aureus* and *Streptococcus agalactiae* Group B, with *Streptococcus agalactiae* Group B normally found in the female urogenital tract. Blood cultures revealed postmortem contaminants only making sepsis unlikely. Toxicology results performed on the postmortem femoral blood were negative. No blood cultures of the decedent were performed.

Given the above finding, the cause of death was determined to be complications of uterine subinvolution at the placental implantation site, the mechanism of death was postpartum hemorrhage, and the manner of death was ruled natural. Although the cause of the subinvolution was unknown, uterine infections have been associated with other cases with a uterine infection also noted in this individual.

Discussion

Postpartum hemorrhage remains one of the major causes of postpartum morbidity and mortality and defined as blood loss >500 mL in vaginal deliveries and >1000 mL for Cesarean births (1). Hemorrhage within the first 24 h after the birth is more common and referred to as primary or early postpartum hemorrhage. Primary and secondary postpartum hemorrhage share many of the same causes and can include uterine atony, retained placenta, placental accrete or percreta, endometrial infection, inherited coagulation disorders, consumptive coagulopathy, and lacerations of the perineum

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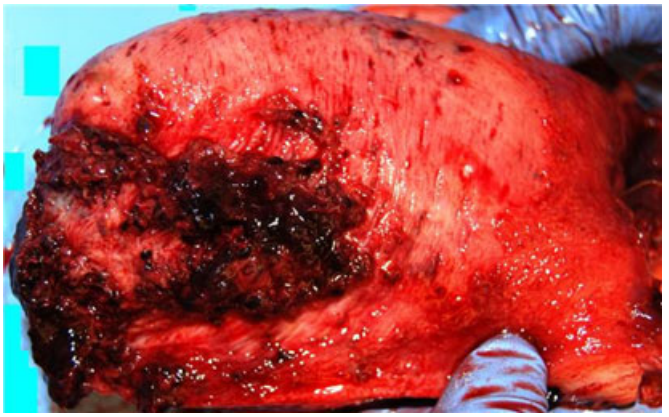

FIG. 1—*Uterus showing the placental implant site with attached blood clot.*

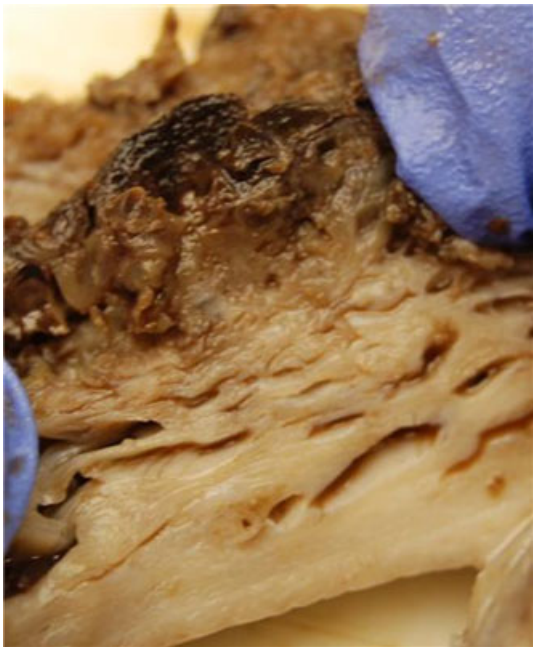

FIG. 2—*Myometrium showing prominent vasculature.*

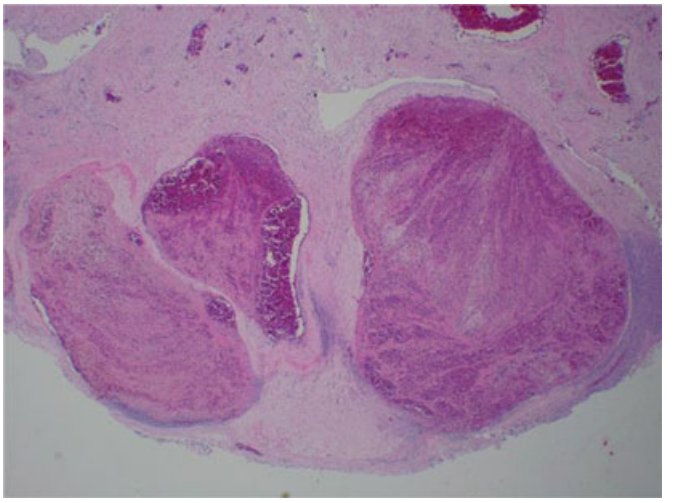

FIG. 3—*Photomicrograph of the placental implantation site with dilated and thrombosed vessels (H & E staining).*

FIG. 4—*Photomicrograph highlighting the cytotrophoblasts within the myometrium and myometrial vessels (low molecular weight cytokeratin immunohistochemistry staining).*

(2). Secondary postpartum hemorrhage, however, has received less attention, most likely because it complicates only about 1% of all pregnancies and is more frequently associated with maternal morbidity rather than mortality. However, secondary postpartum bleeding may be fatal, as is the case in this individual, because the increased uterine bleeding occurs between 1 and 2 weeks after delivery and the patient is often home and unaware that the hemorrhage is significant.

The physiologic and anatomic changes that occur in the uterine vessels during pregnancy and in the postpartum period are complex. In the beginning of pregnancy, the cytotrophoblasts derived from the placenta invade and surround the maternal spiral arteries, transforming them into large vessels that accommodate the increased blood flow needed by the placenta and fetus. The findings are most striking at the site where the placenta has inserted into the uterus. In the normal postpartum period, involution of the arteries occurs. Involution involves the modification of the arteries back to the nongestational state and eventual removal of the arteries from the uterus. The changes in the arteries include fibrointimal thickening, endarteritis, thrombosis, and replacement of the cytotrophoblasts within the vessels by maternal endothelial cells and regeneration of the internal elastic lamina. There is also a disappearance of the cytotrophoblasts from the myometrium interstitium. This process, in addition to the sloughing of the decidua in the superficial endomyometrium and the uterine smooth muscle contraction, is necessary to avoid abnormal postpartum bleeding (3).

In subinvolution of the placental site, the uterus is grossly enlarged and boggy. Multiple microscopic sections of the placental implantation site should be taken to determine the cause of the hemorrhage and to rule out other causes of secondary postpartum bleeding such as gestational trophoblastic disease, retained placenta, placenta accreta, and endometritis. Subinvolution of the placental site is an important cause of secondary postpartum bleeding and is defined by either a partial or complete lack of the normal involution of the superficial modified spiral arteries at the placental implantation site. Microscopically, the spiral arteries in the superficial myometrium are large and dilated and are partially occluded with thrombi (4). In addition, cytotrophoblasts are identified within

and surrounding the vessels and can be highlighted using low molecular cytokeratin immunohistochemistry staining. The clinical symptoms are delayed postpartum bleeding usually within 2 weeks of delivery. There is an abrupt onset of increased uterine bleeding that may require a hysterectomy in some cases.

The etiology of secondary postpartum bleeding often remains unknown if the patient can be treated conservatively; however, if bleeding is severe, a hysterectomy may be performed or the individual may not survive and require an autopsy to determine the cause of the bleeding. The exact pathophysiology of subinvolution is not known. Some suspect an immune component leading to abnormal interaction between the maternal and fetal tissues (5).

Conclusion

Subinvolution of the uterine arteries at the placental implantation site is the result of the modified spiral arteries refusing to convert to a nonpregnant state. This can lead to significant postpartum bleeding, and if not suspected, may result in death as in our case. The pathophysiology behind subinvolution is unknown but an immune etiology with miscommunication between the maternal and fetal tissues may be speculated. Although it is a common suspect in delayed postpartum bleeding and can cause significant morbidity, the mortality rate because of subinvolution is unknown.

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