Involution of Implantation Site and Retained Placenta

Involution of the Placental Site

Pathologists rarely obtain a postpartum uterus to enable a detailed study of the involutional changes that take place at the former site of implantation. Therefore, involution of the normal placental site has been studied by only a few investigators. Normally, the postpartum lochia contain the decidual remnants, including perhaps some of the degenerating remnants of the vasculature that had previously undergone the so-called physiological changes of pregnancy. Only when significant postpartum hemorrhage occurs and hysterectomy then becomes necessary is the pathologist asked to seek the cause of the bleeding. He or she may then find remains of villi, incompletely thrombosed vessels, “placental polyps,” and some degree of inflammatory reaction. Frisoli (1981) suggested that retained placental tissue is found in about 50% of such cases at curettage. The curettings and postpartum uteri are difficult tissues to study objectively because most pathologists have little experience with the normal, complex process of placental site involution. Williams (1931), in a classical paper, attempted to rectify this situation. His study should be read before any interpretation of such a postpartum uterus is undertaken. It must also be recognized that in 85% of normal, delivered placentas the decidua basalis shows foci of polymorphonuclear leukocyte infiltration (Schneider, 1970). These cells are part of an apparently normal process of implantation and are not considered an expression of deciduitis or infection. Nevertheless, postpartum hemorrhage and subinvolution are nearly always associated with significant inflammation, if only because the cervix remains open and patulous.

The placenta separates from the uterus in the decidua basalis, deep to Nitabuch’s fibrin layer. This fibrin layer is usually present in the floor of the delivered placenta but, contrary to some opinions, it does not serve as the cleavage plane. Placental separation probably occurs largely because of the shearing action of the underlying myometrium against the noncontractile placenta. One may speculate that detachment might be easier when the placenta is more turgid and still filled with fetal blood, but this is apparently not the case. Walsh (1968) found that when the cord is clamped early and more fetal blood remains in the placenta, postpartum hemorrhage is significantly more common than when this blood was allowed to drain into the fetus.

Immediately following delivery, the uterine surface becomes covered with fibrin and blood clot; the contraction of the uterus clamps the maternal vessels and stops uterine bleeding. Ludwig (1971) and Ludwig and Metzger (1971) have made elegant electron microscopic observations of this area postpartum. In their scanning electron microscopic study, they speak of a “wallpapering” of the endometrial surface with a delicate meshwork of cross-linked fibrin that includes deformed erythrocytes. They had shown earlier that, with the first contraction after expulsion of the placenta, this fibrin is deposited and that it aids with hemostasis. There is pronounced furrowing of the inner surface of the uterus following delivery that is bridged by the “tapestry” of fibrin. Mukaida et al. (1975), who investigated this topic more recently, came to similar conclusions.

Friedländer (1870) found that most of the endometrium degenerates after birth and that regeneration takes place from the glands and stroma in the spongy layer of the endometrium; this process is largely completed by 4 weeks postpartum, at least in the area away from the implantation site. The endometrium of the implantation site itself is not reformed for several additional weeks after delivery. The controversy over what constitutes physiological changes after birth did not cease until Williams (1931) published his comprehensive study of normal uteri that had been removed up to 4 months after delivery. He showed that the detachment of the placental site proceeded with little inflammation or necrosis. The cessation of blood flow through the endometrial vessels was largely accomplished by contraction of the
uterus itself. In the absence of contraction, as in cases following former overdistension (e.g., in multiple pregnancy, hydramnios), these vessels may bleed extensively. This uterine atony presents a life-threatening situation that demands emergency attention. Normally then, these vessels are clamped by uterine contraction; they thrombose subsequently (Figure 10.1), and the vascular placental site eventually becomes a mass of hyaline plaques (Figure 10.2). The vessels become “organized” by the ingrowth of fibrocytes and endothelium, and they are later recanalized. This process takes many weeks. One can then readily identify former implantation sites by the presence of these unusual hyalinized vessels and by the remains of some placental site giant cells. These residua of implantation have been demonstrated particularly impressively in several monkey species, in which they form macroscopically visible plaques (Bronson et al., 1972). Although the changes in monkeys are in general similar, they differ by involving usually both uterine sides, as their placenta is commonly bidiscoid. Moreover, they are much more massive and persist much longer than the approximately 7 weeks considered to be normal for women. Also, some calcification occurs in the hyaline areas of simian placental site involution, which is not often the case in humans.

The placental site is then “exfoliated,” as Williams (1931) called it, rather than being absorbed into the myometrial tissue. At least the portions that are central to the myometrium are undermined. Williams (1931) considered that the decidual site is undermined by regrowth and by extension and downgrowth of endometrium that originates from remaining endometrial glands and stroma. He described it to be a “conservative” effort of nature to thus dispose of the thrombosed vessels. It must be borne in mind, however, that this applies only to the decidual portions of the placental site; the myometrial part has a much different regressive curve. One mystery of postpartum uterine involution is the rapidity with which the muscle mass is reduced. The average uterus weighs about 1,000 g after term delivery and, largely through the dissolution of cytoplasm, shrinks to less than 100 g usually within 2 months. An appreciation of this