Chapter 12

Postpartum Hemorrhage, Subinvolution of the Placental Site, and Placenta Accreta

General Considerations

Postpartum hemorrhage, if severe, can be a major obstetrical emergency, which, if not treated promptly, may result in rapid exsanguination and demise of the mother. Specimens submitted to the pathology laboratory depend on the clinical situation and may include the *placenta, retroplacental curettings, other sampling from the endometrial cavity, the uterus, or, in some cases, no specimen at all*. Pathologic examination and diagnosis is facilitated by knowledge of the clinical situations and causes of postpartum hemorrhage such as:

- Injury from cervical lacerations or uterine rupture
- Coagulation defects
- Uterine atony
- Retained placental tissue
- Subinvolution of the placental site

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- Postpartum endometritis
- Placenta accreta
- Placental polyp

In the case of coagulation defects, specimens are rarely submitted. If they are, the findings usually consist only of hemorrhage and no specific pathologic lesion. If there is a rupture or laceration, whether or not it is repaired, with associated bleeding necessitating hysterectomy, documentation of the area of injury is essential. Since clinical history is sometimes missing, postpartum hysterectomy specimens should always be examined with the thought in mind that there might be this type of injury, and therefore documentation of any defect and whether there is surrounding hemorrhage should be done at minimum and sections submitted of the area. The remaining causes of postpartum hemorrhage usually result in pathologic lesions and will be discussed in the following sections.

Uterine Atony

Pathogenesis

After delivery of the placenta, cessation of blood flow through the endometrial vessels is largely accomplished by *contraction of the uterus*. **Uterine atony** is defined as the absence of normal uterine contraction. The most common underlying causes are:

- Overdistention from a large fetus, multiple pregnancy, or polyhydramnios
- Anesthetic agents
- Prolonged, augmented or rapid labor
- High parity

When the myometrium loses the ability to contract, the uterine vessels may bleed extensively and present a life-threatening situation necessitating hysterectomy. In general, uterine atony is a clinical diagnosis, but there are pathologic features that are seen in this setting that are relatively specific.

Pathologic Features

In normal circumstances, the postpartum uterus is *enlarged from myometrial hyperplasia and hypertrophy*. The uterine wall is usually markedly thickened but firm due to the contraction of the smooth muscle. If **atony** is present, the uterus will be *edematous and boggy*, and hemorrhage may be grossly evident. Usually this is a diffuse rather than focal change. Microscopically, the findings are relatively nonspecific and consist of typical hypertrophied myometrium with *diffuse, recent hemorrhage* often in the vicinity of large, open, dilated vessels. Groups of myometrial fibers will be *separated by edema fluid*, and these findings will be relatively diffuse throughout the uterus (Fig. 12.1). This is in contrast to what is

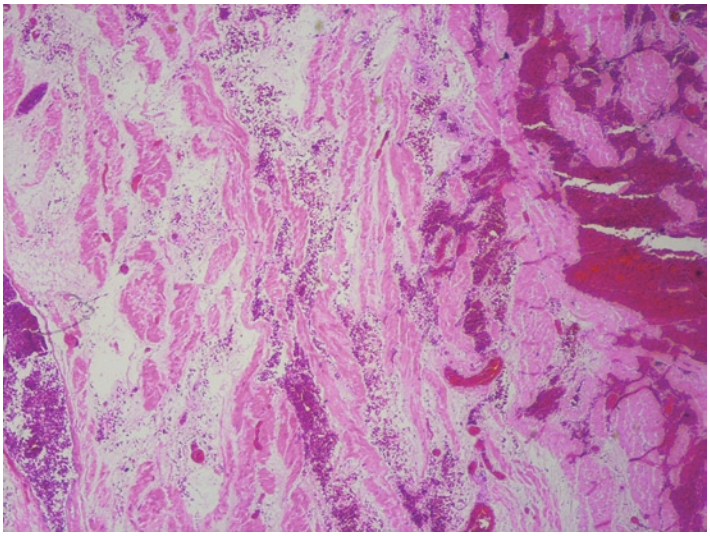

Figure 12.1. Microscopic appearance of the myometrium in a case of uterine atony. Muscle fibers are separated by edema fluid and there is focal recent hemorrhage. H&E ×20.

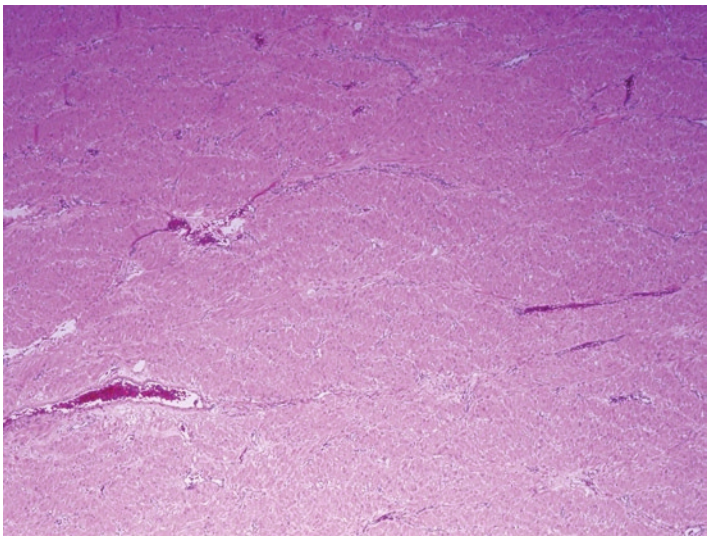

Figure 12.2. Normal postpartum uterus with hypertrophic but closely packed myometrial fibers, no significant hemorrhage and no edema. H&E ×20. Compare to Fig. 12.1.

present in a normally contracted postpartum uterus in which the muscle fibers appear tight against one another without separation by blood or fluid (Fig. 12.2).

Suggestions for Examination and Report
(Uterine atony)

Gross Examination: Representative sections of the uterus should be submitted including the implantation site. The latter is usually a roughened, hemorrhagic area on the endometrial surface. Sections of the lower uterine segment and cervix, if present, should also be submitted. Attention should be given to the presence of lacerations, perforation or evidence of other injury, particularly in the cervix.

Comment: Marked edema and hemorrhage are present, consistent with the clinical history of uterine atony. A comment may also be made about the absence of other pathologic findings, specifically addressing any clinical differential diagnoses.

Retained Placental Tissue and Involution of the Placental Site

It is often assumed that evaluation of the completeness of the maternal surface of the placenta will uncover the presence of missing placenta tissue that has been retained in the uterus. It is therefore interesting that many cases exist in which the placenta was described by experienced observers as "intact" and retained placental tissue was later found to be present. Thus, practically, one cannot be reassured by the integrity of the placenta postpartum, regardless of the experience of the examiner. On the other hand, when the maternal surface of the placenta is **not** intact, the likelihood of retention of placental tissue is heightened. Placental tissue may be retained for a number of reasons. It may be merely due to *inadequate removal of the entire placenta at delivery*. It also may occur due to endometritis, subinvolution of the placental site, or placenta accreta. When there is associated **postpartum hemorrhage**, particularly delayed postpartum hemorrhage, it is more likely to be associated with a pathologic process. Delayed postpartum hemorrhage may occur days, weeks, or even months after delivery.

Normal Involution of the Placental Site

In order to understand subinvolution, one must find understand the complex process of normal placental site involution. Unfortunately, pathologists rarely receive normal postpartum uteri that would enable detailed study of the involutional changes at the former site of implantation. The following is an overview of the events at the placental site following normal delivery. They are also summarized in Table 12.1.

The separation of the placenta from the uterus takes place within the decidua basalis, largely due to the *shearing action of the myometrium as it contracts against the incompressible placenta*. **Immediately following delivery**, contraction of the uterus clamps the arteries, preventing uterine bleeding. The endometrial surface becomes covered with *blood clot and fibrin*. Within minutes to hours, the blood vessels start to undergo *thrombosis* and thus are no longer completely patent. **Within the first postpartum day**, the walls of the arteries and veins in the

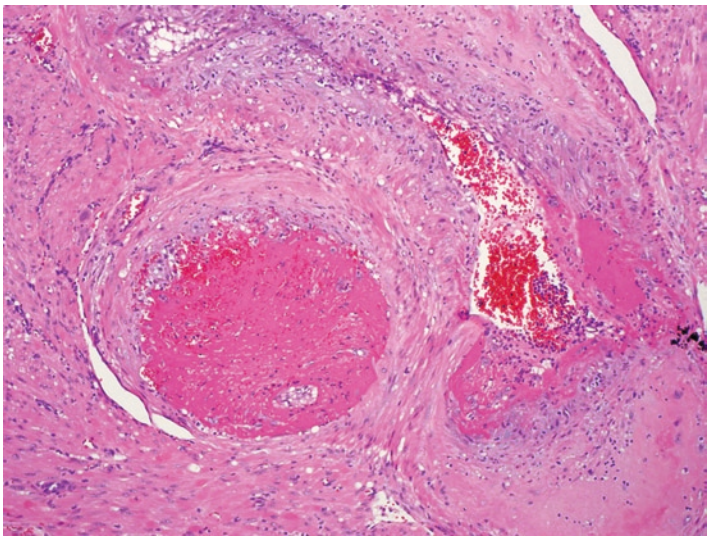

Figure 12.3. Normal involution of the placental site with thrombosed uterine vessels approximately 2 days after delivery showing thrombosis and early obliterative arteritis. H&E ×160.

implantation site *become hyalinized*. *Fibrinoid necrosis and inflammation* develop in the arteries. *The implantation site decreases in size*, from about 18 cm, the approximate diameter of a term placenta, to 9 cm. From **postpartum day 1 to day 3**, the *veins completely thrombose and the arteries develop obliterative endarteritis* (Fig. 12.3). There is early decidual necrosis and a modest neutrophilic and mononuclear infiltrate. From **day 3 to day 5**, *inflammation and necrosis* increase and reactive regenerating endometrial glands begin to appear. The thrombosed veins begin to organize, and the arteries show early intimal proliferation and continuing hyalinization. From **postpartum day 5 to day 8**, there is a *clear demarcation* of the necrotic decidua (which will be subsequently sloughed as the "lochia" or discharge) from the remaining endometrium. *Endometrial glands show pronounced reactive changes and are increased in number*. They regenerate by regrowth and extension of the adjacent endometrial glands and stroma. *Arteries are occluded* by endarteritis by this time. *Placental site giant cells are prominent* early in the involuting implantation site in the endometrium and superficial myometrium, but their numbers decrease over the ensuing weeks. **Three to four weeks** after delivery, *the endometrium at the implantation site is regenerated and inactive with scattered hemosiderophages*. Veins have mostly been recanalized, but *some residual vessels show hyalinization*, which may persist for many weeks even under normal circumstances.

The rapidity of the **involution of myometrial muscle mass** postpartum remains a mystery. *The average postpartum uterus weighs about 1,000 g and shrinks to less than 100 g in about 2 months*. The histologic changes are relatively minimal. Degenerative changes of the muscle occur within hours of delivery, and a mild chronic inflammatory infiltrate develops within the first 4 days and persists for up to 17 weeks. It is important to note that virtually *no myometrium repairs the incisional defect from cesarean*

sections and only a thin fibrous scar approximates the muscle layers. Thus, in subsequent pregnancies the probability of dehiscence exists with possible **uterine rupture** or **placenta accreta** (see below).

Subinvolution of the Placental Site

Pathogenesis

When the uterus does not undergo normal involution, **subinvolution of the placental site** is said to occur. Here, there is *failure of the normal physiologic obliteration of the blood vessels in the placental site as well as delayed myometrial involution*. The uterus is somewhat boggy and edematous, but not to the degree that is seen in uterine atony. There may be delayed postpartum hemorrhage, which typically occurs 1–2 weeks after delivery, but occasionally occurs several months postpartum. This is in contrast to uterine atony in which hemorrhage occurs immediately after delivery and tends to be much more acute and severe. Subinvolution is, in fact, the most common cause of "delayed" postpartum hemorrhage. It is more common in *multiparous women and tends to recur in subsequent pregnancies*. Causes include *retained placental tissue, infection, placenta accreta, and idiopathic causes*.

Pathologic Features

Most patients with subinvolution have normal placentas at delivery. Later, bleeding occurs and the specimen most commonly submitted to the pathology laboratory is uterine curettings. On histologic examination of the endometrial tissue, large *dilated arteries filled with blood and partially organized thrombi* are seen. The arteries are often found in groups of three or four, adjacent to normally involuting vessels (Fig. 12.4). The

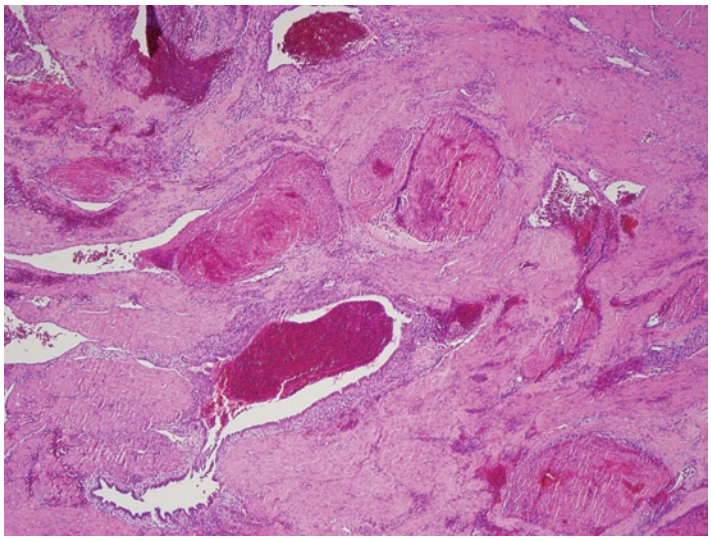

Figure 12.4. Subinvolution of the placental site. Note the enlarged, patent vessels with evidence of bleeding. The curettage was done 3 weeks after delivery and was accompanied by delayed postpartum hemorrhage. Involution is delayed, and the histologic picture is more consistent with what should be seen within the first day or two after delivery. H&E ×20.

histologic picture is often similar to normal involution, but the changes are delayed. Thus, clinical history is crucial in making the diagnosis, as the interval from delivery is necessary to evaluate whether normal involution has been delayed. Furthermore, in contrast to normal involution, where extravillous trophoblast is inconspicuous or absent, subinvolution is characterized by the *persistence of extravillous trophoblast*, particularly in a perivascular location. *Persistence of endovascular extravillous trophoblast* is also occasionally seen.

Suggestions for Examination and Report
(Subinvolution of the placental site)

Gross Examination: Subinvolution is most commonly seen in patients who present with postpartum bleeding. There are no specific gross lesions.

Comment: Subinvolution of the placental site is a common cause of postpartum hemorrhage, particularly delayed postpartum hemorrhage.

Postpartum Endometritis

Postpartum endometritis is an intrauterine infection that is classically caused by group A streptococci, but many other organisms, including anaerobes, have been implicated. It is an **acute endometritis** characterized by *pronounced acute inflammatory infiltrates within endometrial stroma and gland lumens* (Fig. 12.5) and may be associated with colonies of bacterial organisms. The inflammatory infiltrate should be in excess of the minimal acute inflammation associated with normal involution. Collections of neutrophils within gland lumina are an important diagnostic clue. Phlebothrombosis and a plasma cell infiltrate may also be present. Endometritis is often associated with subinvolution, and in this case the histologic features of subinvolution will also be present. Postpartum endometritis may lead to serious complications such as sepsis, pulmonary embolism, and even death. Treatment is usually curettage and antibiotics.

Suggestions for Examination and Report
(Postpartum endometritis)

Gross Examination: There is no specific gross appearance.

Comment: The diagnosis of acute endometritis postpartum, particularly if bacteria are present, may have serious clinical sequelae.

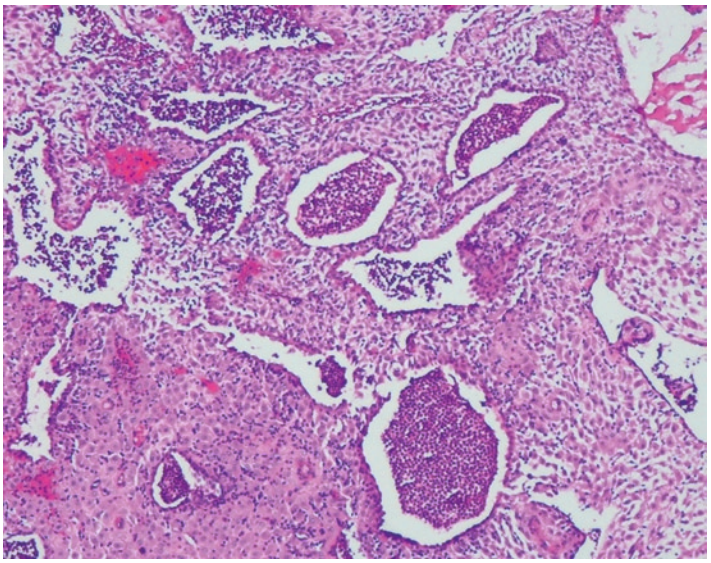

Figure 12.5. Postpartum endometritis showing an inflammatory infiltrate consisting predominantly of acute inflammatory cells. Note the large collections of neutrophils within the gland lumina H&E ×40.

Placenta Accreta, Placenta Increta, and Placenta Percreta

In normal implantation, the extravillous trophoblast invades the decidua in a controlled fashion and converts the spiral arterioles of the endometrium to uteroplacental vessels (see Chap. 8). In **placenta accreta**, there is a *failure of the normal decidua to form*, at least locally, because the endometrium is deficient and cannot decidualize. The trophoblast does not stop invading when it should, and chorionic villi penetrate into the myometrium. Traditionally, placenta accreta has been divided into **placenta accreta, placenta increta**, and **placenta percreta**, based on how deeply the trophoblastic tissues invade. In **placenta accreta**, *the chorionic villi are attached to myometrium without intervening decidua*, in **placenta increta** *the myometrium is invaded by the placental villous tissue*, and in **placenta percreta** *the villi penetrate through the entire uterine wall*. The underlying pathogenetic mechanisms and etiologies are likely to be the same, the only difference being a quantitative one, which, however, may be of considerable clinical importance, particularly in the case of placenta percreta.

Clinical Features and Implications

Placenta accreta is relatively rare, with an incidence of around one in 7,000 pregnancies. The incidence is higher in the setting of placenta previa, where it is estimated to be 1.18%. This has been termed "placental previa accreta" and develops due to deficient decidualization of the cervical stroma. The occurrence of placenta accreta has been steadily rising, and this is thought to be secondary to the increased

cesarean section rate. The type of surgical closure after cesarean section is also thought to have an impact on the development of future placenta accreta. It is often detected after delivery *when the placenta fails to separate or is incompletely delivered*. Increatas and percretas more frequently manifest antepartum and earlier in gestation because of hemorrhage or uterine rupture. In 45% of cases, there is an elevation of maternal serum α-fetoprotein levels. Diagnosis by ultrasonography and magnetic resonance imaging (MRI) is possible, and cases have been reported as early as 14 weeks. Sonography of placenta accreta often displays *irregular lucencies in the villous tissue*. These "lakes" presumably derive from the abnormal implantation and an abnormal disposition of maternal spiral arterioles to the intervillous space.

Placenta accreta may be associated with *life-threatening hemorrhage that can lead to maternal and/or fetal death*. Maternal deaths occur in approximately 9.5% of cases and fetal death in a similar percentage. Placenta percreta may lead to uterine rupture, or it may invade the bladder, causing hematuria. Massive hemorrhage from perforation has also been described. Thus, when a placenta percreta or a deep placenta increta is identified by radiologic studies, delivery by cesarean section with hysterectomy is usually undertaken even in cases where the fetus is significantly premature. Although the usual treatment is hysterectomy, microembolization through the internal iliac arteries has been used to treat less severe cases of placenta accreta. Embolization is performed, and the placenta may be left within the uterus, to be followed by spontaneous expulsion several days later. Occasionally the placenta may be retained in these circumstances for months. Pathologic changes of uterine retention of the placenta are discussed below.

Pathogenesis

In **placenta accreta**, the villous tissues are anchored to muscle fibers rather than to intervening decidual cells due to a *deficiency of decidua*. Normally, the placenta separates from the uterine musculature in a plane just peripheral to Nitabuch's fibrinoid layer, within the decidua basalis. It is accomplished by the *shearing action of contracting myometrium against the stationary, noncontracting placenta and occurs in irregular planes of friable decidual cells*. Without this layer, uterine contractions do not dislodge the placenta or portions of the placenta, or the entire placenta is retained. Sometimes the area of adherence may be quite small, and retention of placental tissue in the uterus may not be immediately noticed.

Placenta accreta is a nice example of the *importance of endometrial decidualization for proper control of trophoblast invasion*. This correlation is further underlined by the fact that *absence of decidualization in tubal pregnancy also coincides with increased trophoblastic invasiveness*, and thus ectopic pregnancies are essentially tubal placenta accretas. They usually perforate the wall, becoming placenta percretas. Tubal rupture does not occur from distention of the tube but rather the penetration of the relatively thin muscular wall. A similar situation arises in the lower uterine segment and endocervix as decidualization is not fully developed in these areas. At present, the specific decidual characteristics responsible for control of invasiveness are still unknown.

Any condition that leads to the development of **deficient decidua** predisposes the patient to placenta accreta. *The most frequent predisposing condition is a history of previous cesarean section and/or curettage*. The risk for development of placenta accreta increases with a history of multiple cesarean sections and multiple surgeries. Other predisposing conditions include *placenta previa (see Chap. 13), submucosal leiomyoma, cornual implantation, placenta membranacea (see Chap. 13), and uterine anomalies*. In all these cases, there is the potential for deficient decidualization. Placenta accretas and particularly percretas are said to be increasing in frequency and this undoubtedly relates to the greater frequency of cesarean sections. In a surgical incision, reconstitution of a *normal* uterine wall is not possible. Therefore, in the subsequent pregnancy, the expanding uterus may dehisce at the former incision site. If the placenta implants over this previous scar, uterine expansion will cause the placenta to be implanted on very thin scar tissue and/or peritoneum.

Pathologic Features

In placenta accreta, the placenta is often disrupted during delivery and there may be *missing cotyledons*. However, completeness of the maternal surface cannot always be accurately evaluated. If the placenta is relatively intact, a **focal placenta accreta** may still be present. When histologic sections of such a placenta are made, the deficiency of endometrium that underlies placenta accreta is generally not evident. It may be possible to make the diagnosis of placenta accreta if curettings are done that include the myometrium, but it is very difficult as the tissue is often difficult to orient. If portions of the myometrium are removed with the placenta and remain attached to the floor (Fig. 12.6), the diagnosis may also be made.

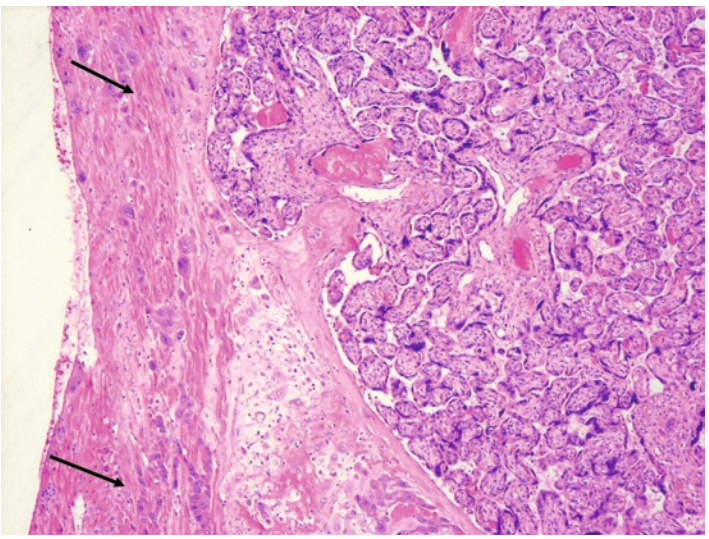

Figure 12.6. Section of the basal plate of a term placenta showing the presence of myometrial fibers (*arrows*) with intervening extravillous trophoblast but no decidua between the muscle and the chorionic villi indicative of at least focal placenta accreta. H&E ×40.

However, in the case of a placental specimen or curettings, the diagnosis of accreta can certainly not be ruled out. The diagnosis is much easier to accomplish when the entire uterus is available, which of course is the less acceptable outcome for the patient. Nevertheless, hysterectomy is a frequent sequel of placenta accreta.

The cesarean-hysterectomy specimen is often quite remarkable on gross examination (Fig. 12.7). If the diagnosis is known prior to delivery, the placenta may be left in situ in the uterus. Then, the true relationship of the placenta to the implantation site may be studied. The serosal surface of the uterus is often *congested and hemorrhagic, and may show nodular protrusions representing a thinned myometrium overlying placental tissue* (Fig. 12.7). As a cesarean section is performed prior to the hysterectomy, an anterior incision is usually evident, which may or may not have been sutured. Examination of the uterine cavity will show placenta implanted *over myometrium, which is markedly thinned, or even absent* (Fig. 12.7). At times, only a thin covering of peritoneum is present over the placenta. If the placenta is not left intact, retained placental tissue may still be visible firmly attached to the endometrium. In placenta percreta (Fig. 12.8), placental tissue may be visible perforating through the uterine serosa. Care must be taken to ensure that loss of

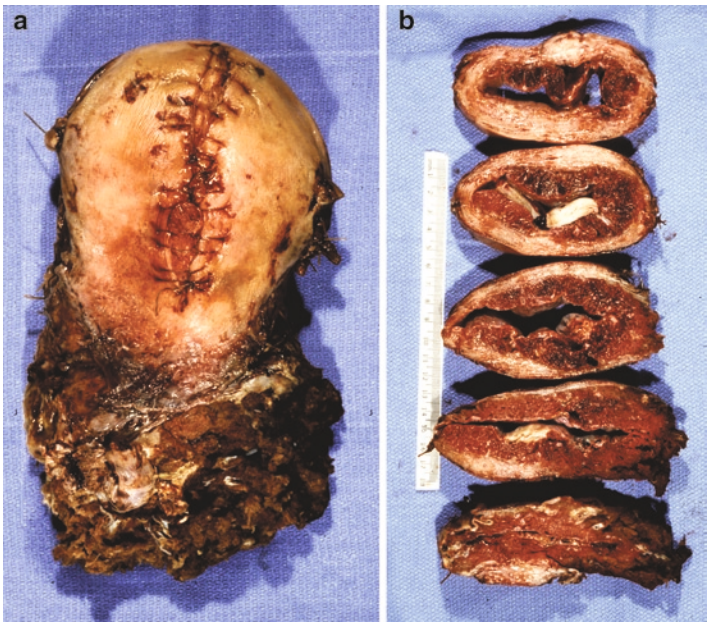

Figure 12.7. Cesarean-hysterectomy specimen with placental implantation over the cervical os leading to a placenta previa accreta. (**a**) Note the protrusion of hemorrhagic placental tissue in the lower segment. A *vertical* scar represents the incision made for the cesarean section, which was then sutured prior to the hysterectomy. (**b**) Same specimen as part (**a**). Serial transverse sections have been made with the most superior at the top and the most inferior at the bottom. Note that the myometrium becomes thinned to invisibility in the lower uterine segment. This was essentially a placenta previa percreta as it invaded through the lower uterine segment.

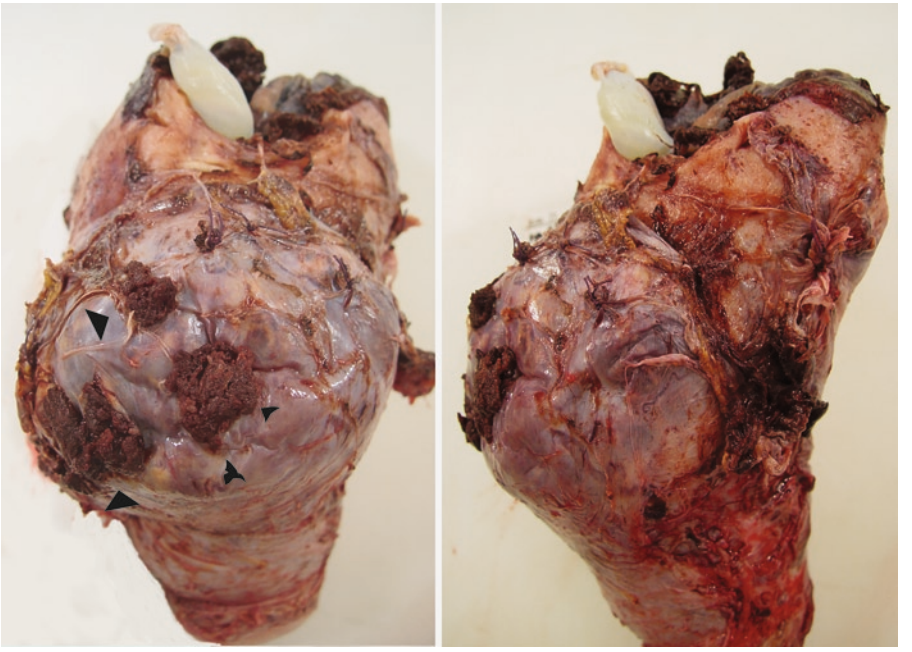

Figure 12.8. Photograph of cesarean-hysterectomy specimen with placenta percreta in which placental tissue can be seen protruding through the serosal surface (*arrowheads* at *left*).

integrity of the serosa is not due to rough handling of the specimen prior to examination. Correlation with clinical history may be helpful in these cases. On microscopic examination, one sees *villous tissue that has grown onto or into the myometrium without intervening decidua*. It is important to note that it is the **lack of decidua** that is diagnostic of this entity (Fig. 12.9). This point is discussed more fully in the next section.

There are several associated pathologic findings seen with placenta accreta. First, the normal *physiologic conversion of maternal vessels may be focally deficient*. This may be related to the abnormal invasiveness of trophoblast or to the general lack of availability of decidual vessels for implantation. There is also usually a *deficiency of placental septum formation*. When septa are present in a placenta accreta, they are composed of uterine muscle rather than decidua, extravillous trophoblast, and fibrinoid. This leads to abnormal flow patterns in the intervillous space, which may be appreciated on antepartum imaging.

Pitfalls in Diagnosis: There are several important pitfalls in the diagnosis of placenta accreta, partly due to confusion in distinguishing the populations of cells that make up the placental floor. The first difficulty lies in the fact that although the presence of chorionic villi attached to the myometrium is diagnostic of placenta accreta, *rarely are the chorionic villi present* **directly** *on the myometrium*. This is unfortunate because this is the most common definition of placenta accreta in textbooks and journal articles, and it is technically not quite correct. Villi implanted on the myometrium are really a fortuitous finding and are not required for

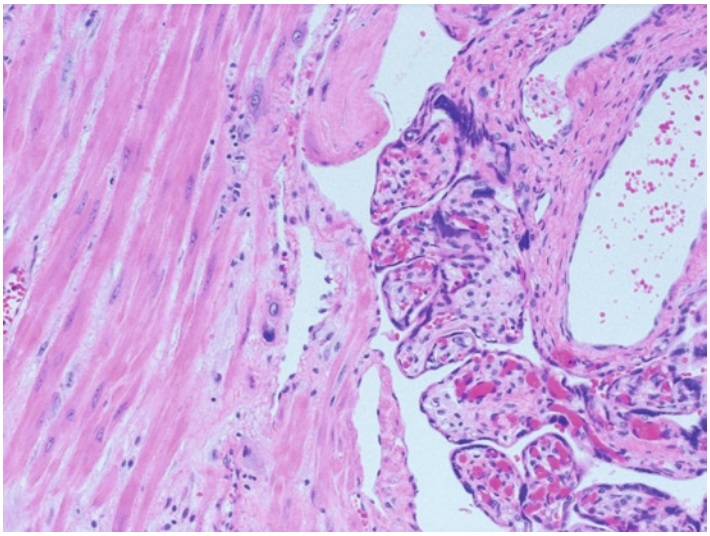

Figure 12.9. Placenta accreta showing "classic" picture with chorionic villi attached directly to the myometrium. H&E ×200.

diagnosis. The main defect in placenta accreta is the *deficiency of decidua*, and that is generally not discussed. In most cases, there is *fibrinoid and extravillous trophoblast in between* the myometrium and the villous tissue (Fig. 12.10a). The crucial point here is that the diagnostic feature of placenta accreta is the **lack of decidua and not implantation onto the myometrium**. Therefore, if villi are present adjacent to fibrinoid or extravillous trophoblast, which is **then** adjacent to myometrium, and there is no intervening decidua, the diagnosis of placenta accreta is made. Insistence on the demonstration of villous implantation on the myometrium will result in *underdiagnosis*.

The second cause of underdiagnosis is *confusion of extravillous trophoblast with decidua*. Extravillous trophoblasts are always present in the implantation site and are normally present adjacent to the myometrium and villous tissue. *If these trophoblastic cells are misinterpreted as decidual cells, the diagnosis will be missed* (Fig. 12.10a) and also result in underdiagnosis. If there is doubt about the true nature of cells in the implantation site, immunohistochemistry for cytokeratin can be extremely helpful, as trophoblastic cells are epithelial and are strongly positive for cytokeratins, while decidual cells are not.

Overdiagnosis of placenta accreta may also occur. In the normal implantation site, extravillous trophoblast and placental-site giant cells (see Chap. 8) are present in the basal portion of the placenta, the decidua, *and the myometrium*. Often, the presence of placental-site giant cells within the myometrium is interpreted as evidence of placenta accreta. However, the presence of these trophoblastic cells within the myometrium is *a normal finding and is not diagnostic of placenta accreta* (Fig. 12.10b).

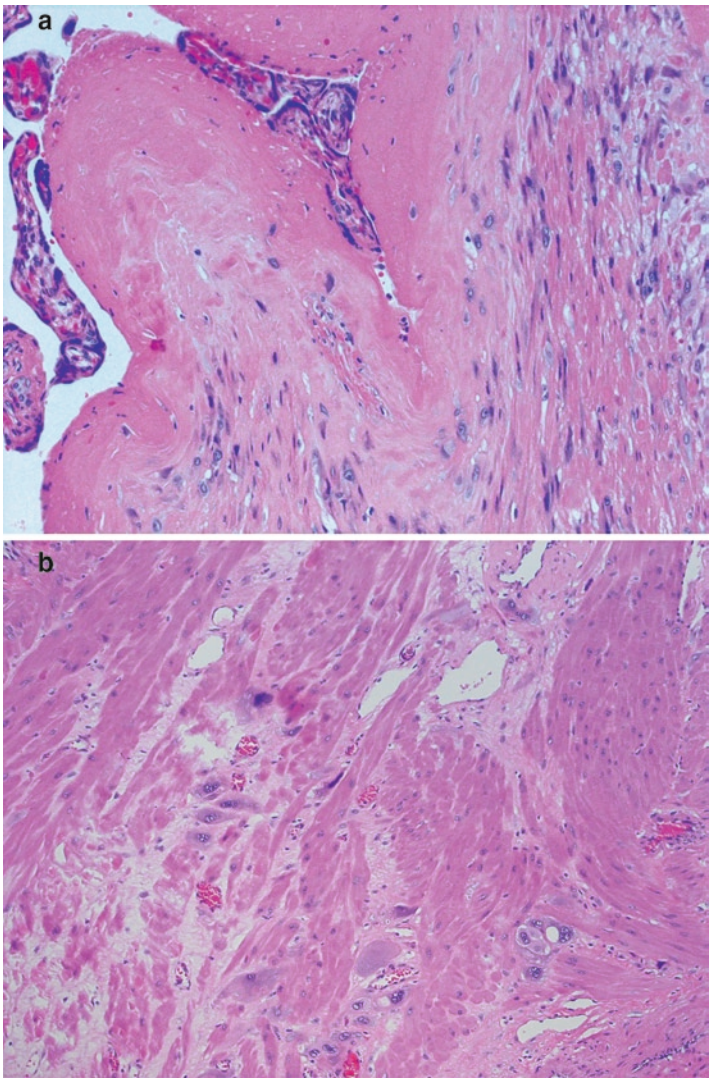

Figure 12.10. (**a**) Placenta accreta. Here, the chorionic villi implant on fibrinoid and extravillous trophoblast and not directly on myometrium (at the *right*), but with the absence of decidua is still diagnostic of placenta accreta. The extravillous trophoblast may be confused with decidual cells. H&E ×100. (**b**) Placental site giant cells present within myometrium, a normal finding of the implantation site that is not diagnostic of placenta accreta. H&E ×20.

Suggestions for Examination and Report
(Placenta accreta, placenta increta, and placenta percreta)

Gross Examination: If only the placenta is submitted, examination should involve careful inspection of the maternal surface for completeness and the presence of firm white tissue, which may represent attached myometrium. Retroplacental curettings should be completely submitted for microscopic examination. In a hysterectomy

(continued)

specimen, the area of accreta is often obvious, particularly if the placenta is left in situ. The myometrium will appear thinned on cut section. Sections should be taken to include placenta and myometrium in areas where *the myometrium is thinned or where there is firm placental attachment*. The anterior lower uterine segment is the most common place for placenta accretas associated with previous cesarean section. If the site of accreta is not obvious, or the placenta is not included, multiple sections should be submitted from the most likely areas to show accreta anteriorly – the lower uterine segment and cervix. The most hemorrhagic and roughened areas are the most likely to represent implantation site or retained placental tissue. One or two sections of normal implantation site should also be submitted along with a section without implantation site.

Comment: Comments should be directed to the location where the accreta was found and the extent of the accreta, e.g., depth and breadth. Usually a measurement is not necessary, but clinicians appreciate knowing whether it is only focal or extensive, where it is, and which side it involves. Other pathology that may be associated with increased risk of accretas should also be commented on, such as uterine scar, bicornuate uterus, etc.

Placental Polyp

Placental polyps are polypoid fragments of tissue consisting of degenerated chorionic villi that have become encased in fibrinoid and layered clot. They represent **focal placentas accretas**. Because of the degenerative changes associated with intrauterine retention of this tissue, the diagnosis may be difficult to verify. At times, however, some myometrial tissue is also present and one finds *villi directly attached to myometrium*. Placental polyps may be *seen in endometrial curettings for postpartum bleeding or may be spontaneously passed weeks or months after delivery* (Fig. 12.11). They are seen in up to 45% of women who present with delayed postpartum hemorrhage. When they are removed or spontaneously passed, the symptoms of bleeding usually abate. Rarely, failure to remove placental polyps has resulted in potentially life-threatening hemorrhage.

Suggestions for Examination and Report
(Placental polyp)

Gross Examination: Placental polyps are usually submitted as curettings or as an endometrial polyp in women with delayed postpartum bleeding. Unless unusually large, the specimen should be entirely submitted.

Comment: Placental polyps are usually indicative of a focal placenta accreta.

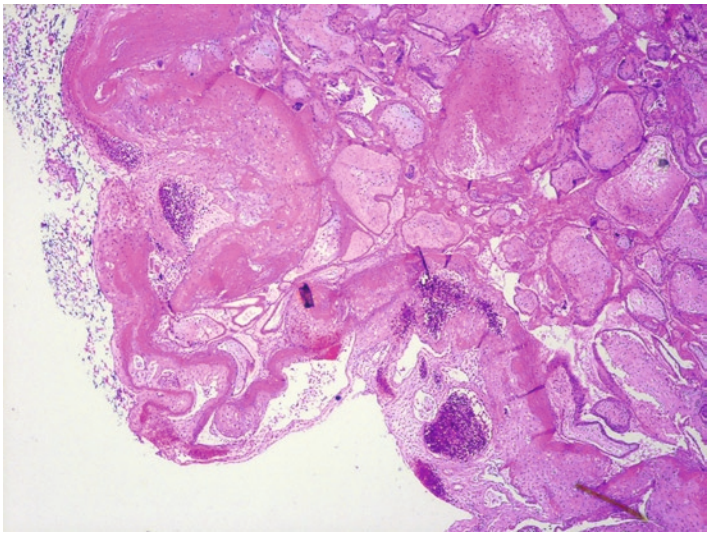

Figure 12.11. Placental polyp. Spontaneously passed tissue consisting of predominantly of degenerating chorionic villi enmeshed in fibrinoid and extravillous trophoblast. Although the implantation of this placental fragment is not present, the diagnosis of placenta accreta is presumed. H&E ×20.

Involution of a Retained Placenta

Placentas may be retained in utero after a fetal demise, when only one of a set of twins survives or when the placenta is not removed after delivery because of a placenta accreta. Since there is continued perfusion by maternal blood, the placental *tissue remains structurally intact for a long time*, particularly the trophoblastic cells. Initially there is *increased syncytial knotting*, followed by involution of the fetal vasculature, resulting in *avascular villi*. *Fibrinoid also accumulates in the intervillous space*. Eventually, the placenta atrophies and comes to resemble an infarct with marked calcification and villous hyalinization (Fig. 12.12). The more remote the fetal demise, the more likely the degenerative changes will mask any other pathologic lesions present.

Suggestions for Examination and Report
(Involution of a retained placenta)

Gross Examination: The placenta may appear grossly infarcted and is usually quite firm. The cord and membranes often are discolored red due to hemolysis. Routine sections should be submitted.
Comment: Increased syncytial knots, fibrinoid deposition, calcification, and degenerative changes are consistent with retention of placental tissue after delivery or fetal death.

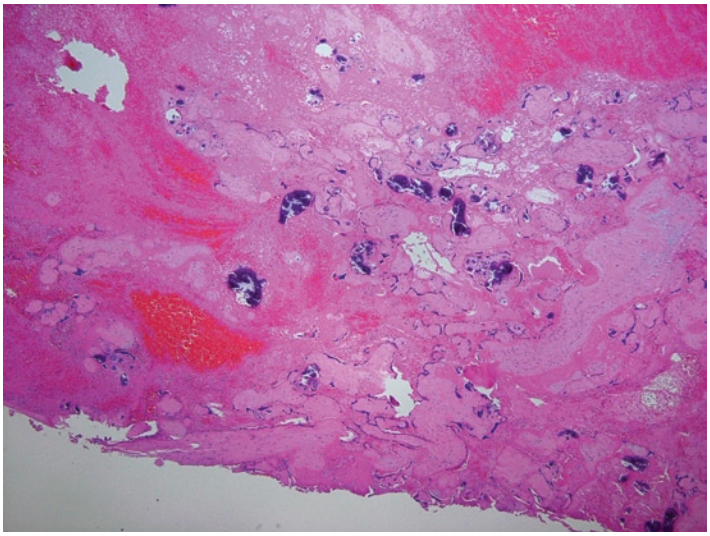

Figure 12.12. Involuting placenta in case of intrauterine fetal demise many weeks previously. Note the avascular, hyaline villi, fibrinoid and hemorrhage. H&E ×20.

Table 12.1. Histologic changes of normal placental site involution.

Time postpartum	Gross size (cm)	"Slough"	Glands	Decidua	Veins	Arteries
<1 day	From 18 to 9	Hemorrhage	Few, inactive	Viable	Hyalinized	Fibrinoid necrosis, minimal inflammation
1–3 days	7–8	Early necrosis	Mild reactive change	Necrosis and inflammation	Thrombosed	Obliterative endarteritis, hyalinization
3–5 days	6	Necrosis with inflammation	Regenerating glands, moderate reactive change	Increased necrosis and inflammation	Organizing	Hyalinization, intimal proliferation
5–8 days	4.5	Well demarcated	Marked reactive change, increased numbers of glands, placental site giant cells	Necrosis and inflammation	Organizing thrombi	Hyalinization
4–20 weeks	2.0	None	Inactive glands, hemosiderophages	None	Recanalized, hyalinized	Remnants of hyalinized vessels

Selected References

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