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One-Step Conservative Surgery for Abnormal Invasive Placenta (Placenta Accreta–Increta–Percreta)

J. M. Palacios-Jaraquemada

INTRODUCTION

Fifty years ago, placenta accreta was an obstetric rarity. Today, however, placenta accreta and its variations represent one of the principal causes of maternal morbidity and mortality. That this is the case is often attributed to the increased number of cesarean deliveries, but close examination of the numbers involved suggests that other factors may also be in operation.

The potential for the compromise of neighboring organs, as well as the development of neovascularization, implies specific technical difficulties associated with the treatment of placenta accreta, all of which directly relate to morbidity and mortality secondary to hemorrhage. Placenta accreta, characterized by the abnormal adherence of the placenta to the myometrium, may present different degrees of invasion, which are categorized as placenta accreta, increta and percreta. Since these terms are all based on histological examination, their proper use should be postoperative as well as retrospective. From a clinical perspective, the different degrees of invasion are more appropriately termed placenta accreta or abnormal invasive placenta.

The most common location of placenta accreta is the anterior lower uterine wall, especially when associated with a prior cesarean scar[1–3]. This association implies difficulties from a technical surgical point of view including adherence to bladder, development of neovascularization, destruction of myometrial tissue and access to the pelvic subperitoneal spaces. The uteroplacental tissues of placenta accreta are noticeably fragile and tend to bleed excessively. Absence of surgical planes for dissection makes it difficult or nearly impossible to apply the usual hemostatic procedures if an accurate tissue dissection between invaded tissues cannot be made.

Understanding the behavior and development of placenta accreta is essential in order to plan an appropriate surgical approach. Various vascular occlusive mechanisms have been used to reduce the tendency for bleeding[4–6], but they have not always been effective and, in some cases, have been deficient. Issues such as these have led to the need for a detailed study of each aspect related to the treatment of placenta accreta. One of the most important is to understand how both the pelvic anastomotic system and the collateral uterine vascular anastomoses work (see Chapters 1 and 22).

Predicting the surgical difficulty, as well as understanding the specific invaded areas, is essential in order to know which vascular pedicles are involved in a given case. A combination of vascular control, fascial dissection and identification of specific pelvic elements (ureter or specific vessels) makes it possible to prevent injuries and to avoid complications. Once a primary diagnosis has been arrived at, the next priority is to know how and where the placenta invades the adjacent tissues.

Designing a one-time surgery implies solving all the problems caused by placenta accreta at one operation. This involves vascular disconnection of the invaded organs (uterus, placenta and bladder), correct compartment exposure of the pelvic organs (necessary for the hemostatic procedures), total resection of the invaded myometrium and, finally, uterine and vesical reconstruction[7].

One-step conservative surgery for abnormal placentation (OSCS) was first implemented in 1990 and, 20 years later, has been applied in more than 450 patients. This series includes the most diverse types and degrees of placental invasion, operated upon electively as well as in emergency circumstances. To date, 106 consecutive postrepair pregnancies have been reported; of these, only two cases of partial recurrence were noted. This number represents the lowest relapse rate reported for conservative treatments in abnormal invasive placenta.

PLACENTAL INVASION

For years, the absence of Nitabuch's layer was considered the main phenomenon that led to abnormal placental invasion. However, numerous cases have been reported where absence of Nitabuch's layer coexisted

with normal placentation. This observation suggests that the absence of this membrane might be a secondary process, rather than the primary cause of abnormal adherence[8]. In mammals, damage to the uterine collagen modifies decidualization; as such, it is logical to propose that a deformed collagen scar (repeated cesarean, postsurgical damage, radiation, etc.) may have a similar effect in the human being.

Placental invasion represents a highly complex phenomenon associated with numerous biochemical interactions. A series of myometrial mediators promote a physiological limit to trophoblastic invasion. In cases of extreme myometrial thinning (e.g. previous cesarean scar), the placenta might not find this physiological limitation and thus invade the myometrium excessively[9]. The absence of adequate vascular support would induce the secretion of vascular growth factors, the purpose of which would be to ensure sufficient placental flow to the fetus. This phenomenon would promote the opening and hypertrophy of microscopic anastomotic collaterals between the uteroplacental tissue, the bladder and the vagina. The vascular growth factors allow the newly formed vessels to grow rapidly and with a high flow. These characteristics are ideal to maintain an optimal placental blood supply; on the other hand, they also represent a surgical nightmare.

Fortunately, not all placental invasions have characteristic newly formed vessels. This is because the mechanism through which the placenta reaches the serosa is different. For placental advancement to occur through the myometrium there must be a prior tissue lesion. That is to say that placental advancement is a physical fact secondary to rupture of the myometrium. If, on the other hand, this fact depended on an invasive characteristic of the placenta, we would be able to see a similar percentage of invasions on all sides of the uterus, a phenomenon which has not been observed to date. All the evidence seems to indicate that if placental implantation is adequately supplied with blood, the placenta only advances through the myometrial defect. In contrast, if the placenta does not obtain an adequate vascular supply, the intense release of vascular growth factors promotes the thickening of the vascular microanastomoses (newly formed vessels, also called neovascularization).

SURGICAL ANATOMY

Morphological classification

Although three types of adherent placenta are found throughout the obstetrics literature (accreta, increta and percreta), this classification is retrospective, histopathological in nature and of uncertain use in surgical practice. Moreover, efforts to find the corresponding diagnostic images of this histological classification have been elusive. Finally, not even pathologists consider the histological examination of placenta accreta as a diagnostic gold standard[10,11]. This apparent contradiction is supported by the fact that, in one specimen, all types and degrees of invasion may coexist. Therefore, histological diagnosis only provides a report of the site from which the sample was taken, and this area may or may not be representative of the remainder of the invaded area. Unfortunately, however, histological classification is relative to the degree of surgical difficulty; therefore, it is not always necessary to persist with presurgical studies. For example, countless cases describe simple surgery for placenta percreta, and extremely difficult surgery with bleeding for localized placenta accreta. In contrast, classification according to invasion areas bears a close correlation with the possibility of bleeding and surgical complexity; both features correspond to the origin of the blood supply and to the difficulty in pelvic dissection. The classification based on invaded areas makes it possible to know and plan how and on which vessels to perform vascular control[12,13].

From a surgical morphological point of view, three main types of anterior placental adherences may be distinguished[14]. In type 1 the anterior segment is noticeably thinner and the placenta reaches the serous surface, no newly formed placental–vesical or vesicouterine vessels are identified, and there is a lax dividing plane between the posterior bladder wall and the anterior surface of the uterine segment (Figure 1). In type 2 both the lower uterine segment and the posterior wall of the bladder are noticeably thinner, there is no lax plane between both organs and a fibrous scar connects them, and no newly formed placental–vesical or vesicouterine vessels are observed (Figure 2). Type 3 is characterized by a thinner uterine segment, vesical wall of variable thickness, presence of placental–vesical and vesicouterine neovascular circulation and vesicouterine plane with or without fibrous adherence (Figure 3).

This morphological, diagnostic and intrasurgical division establishes the type of approach advisable for each type. Type 1 usually constitutes the typical case labelled diagnostic false positive. In this type, both ultrasound and placental magnetic resonance imaging (pMRI) show dehiscence with placental advancement which reaches the serosa; therefore, and from an

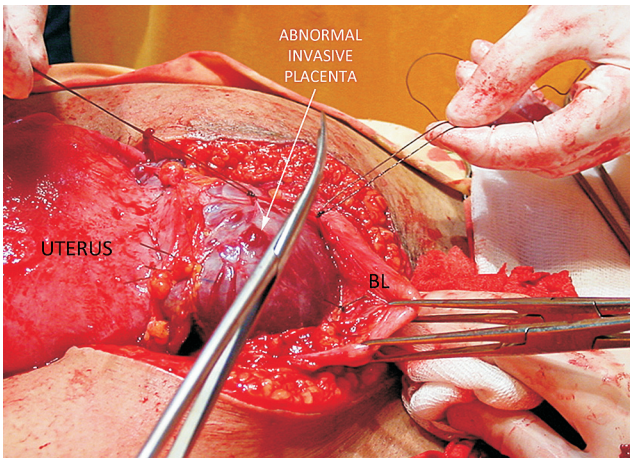

Figure 1 Superior view of anterior placental invasion. The placenta reaches to the uterine serosa, but there is a lax dissectible tissue between posterior bladder wall and the placenta. BL, bladder

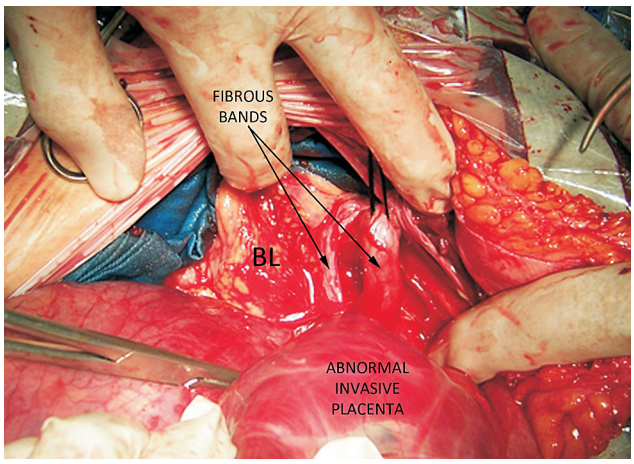

Figure 2 Superior view of anterior placental invasion. Notice the dense fibrous bands between the posterior bladder wall and the placenta. BL, bladder

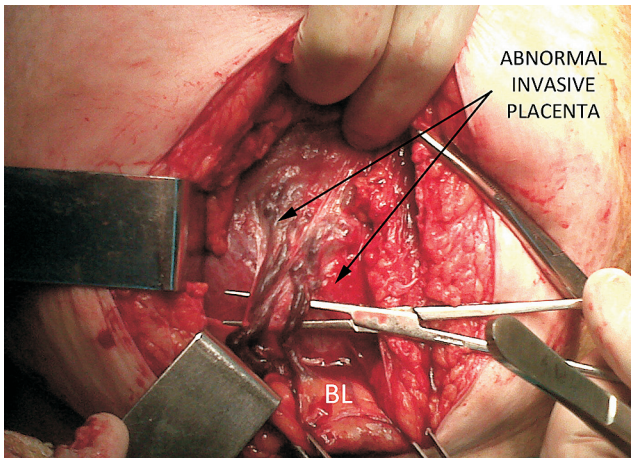

Figure 3 Superior view of anterior placental invasion. Newly formed vessels are present between posterior bladder wall and the abnormal placental invasion (black arrows) is evident. BL, bladder

exclusively diagnostic point of view, these are cases of placenta percreta. However, once the newborn is delivered, placental detachment can be performed without difficulty. There is no bleeding, and on direct examination no remains of adherent placenta are observed. A more detailed examination – which includes dissection of the retrovesical plane – would show a circular area of dehiscence hardly detectable if it is not explored specifically.

Type 2 provides an apparent sensation of safety when the vesicouterine pouch is opened; the dissection plane is narrow though manageable. While dissection progresses, small and persistent hemorrhagic foci occur which are difficult to control. The dissection plane continues to be identifiable until the surgeon unexpectedly sees the vesical mucosa or the Foley catheter. Access inside the bladder is almost imperceptible because dissection is performed on a fibrosed plane that connects the posterior vesical wall with the uterine scar. If, during dissection, the anterior side of the placental invasion is injured, a severe high-pressure hemorrhage ensues, aggravated by the fetal content. Attempts to achieve hemostasis or suture of the placental invaded area (extremely thinning) usually aggravate the blood loss due to uterine tissue rupture, making it essential to evacuate the uterus in order to control the hemorrhage.

In type 3 the neovascular component must be ligated and divided between double ligatures so as to access the vesicouterine space; the ligature maneuver must always leave the main segment of the ligated vessel on the uterine side, because if the ligature is cut or released, the vessel which was cut off may easily be clamped with a hemostatic clamp. If this happens over the vesical sector, it is convenient to perform a vascular suture with polyglactin 000, which will include the vesical muscular tunic, to provide the mechanical support when suture is adjusted.

For the cases of fibrosis of the vesicouterine plane, it is advisable to open the anterior side of the parametrium and to dissect medially the cervicovesical space. This plane is only rarely invaded; however, if this occurs, it may be dissected through stepwise vascular ligatures and vascular section. Once inside this space, both fingers can be introduced laterally (Pelosi's maneuver) through the vesicocervical plane. After this, dissection and ligature can be performed on all the newly formed vessels towards the cephalic sector[15].

Induced neovascularization

Neovascularization is one of the main surgical problems in abnormal invasive placenta. These vessels are usually of high caliber and flow, and even though they may initially appear to have an anarchical pattern, they do not. From an embryological point of view, the vascular growth to the pelvis is formed by magma of interanastomosed vessels. Once organ differentiation has been established, many vessels develop whereas others regress until they become invisible. However, when the appropriate conditions are present (vascular growth factors), these vessels can develop and establish anatomical connections not described in normal conditions.

In cases of anterior placenta accreta, newly formed vessels can be observed between the uterine, vesical and vaginal arteries. Preoperative identification of the abnormal placental invasion enables planning of the approach and the technical tactics necessary to perform the specific vascular control of the pedicles involved.

Anatomy of newly formed vessels

It is usual to associate placenta accreta with newly formed vessels that communicate with the placenta, uterus, bladder and neighboring tissues. Newly formed vessels are generally of a large axial diameter, and they make adequate vascular exchange with the placenta possible. Once abnormal placentation has been established, the proliferation of angiogenic growth factors enables the development of high-volume

newly formed vessels with fragile and less developed medial muscular layers. Although these latter vessels initially appear to have an anarchic pattern, this specific arterial and venous vascular group has an organized distribution[14] at three anastomotic levels: (1) vesicouterine system (VUS), (2) placental–vesical system (PVS) and (3) colpouterine system (CUS). The VUS habitually involves vessels that connect the uterine artery with the posterior-superior bladder wall and also with the contralateral uterine artery. These are superficial vessels, and can be observed through the vesicouterine fold running transversally. The presence of direct anastomoses of considerable size between the uterine arteries and the bladder must be considered during embolization, because this communication can be a direct means to perform an undesired occlusion in the vesical parenchyma (Figure 4).

The PVS is probably the best known, as it establishes a connection between the placental vasculature and the vesicular muscular layer, and is perpendicular to the vesicouterine plane. Anastomoses between the bladder and placenta (PVS) could be observed as thin interconnected net (Figure 5) as well as easily identifiable thick cords (Figure 6). The PVS can send and receive vessels from the entire surface of the posterior bladder wall, and therefore make connections with branches of the upper and lower vesical arteries, although it frequently does so with the upper vesical pedicle.

Finally, the CUS is the anatomically most hidden (obscure) system, though probably it is the most important physiologically. The CUS is located in the thickness of the anterior bladder wall and is parallel to its long axis; thus, it can only be identified through deep dissection of the retrovesical space. Nevertheless, on occasions CUS may not be visible at first sight, because its macroscopic visualization depends on the degree of anastomotic vascular development, which may vary from imperceptible cords to the replacement of the vaginal muscular tunic for a noticeably developed vascular plexus. The CUS connects the lower, middle and upper vaginal pedicles to the caudal branches of the uterine artery, as well as the anastomotic intrauterine arcade, and is anatomically located between its muscular fibers.

From a merely vascular point of view, the vesicouterine and placental–vesical anastomotic pedicles are likely to be controlled by endovascular occlusion of the uterine arteries (via transanastomotic flow). However, hemostasis of the colpouterine pedicle can be very difficult or nearly impossible, since it would represent a high-flow anastomotic arcade between the vaginal and uterine pedicles. Endovascular access to CUS is improved by catheterization of the internal pudendal artery, which is a branch of the posterior division of the iliac internal artery. On the other hand, CUS hemostasis can be performed in a simple, efficient and safe manner through square compression sutures, as described by Cho *et al.*[16]. This method stops bleeding from specific areas regardless of vessel origin.

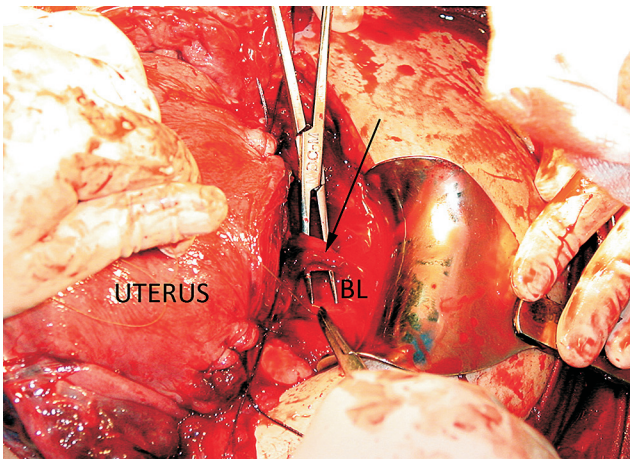

Figure 4 Superior view of lower uterus after reconstructive surgery for abnormal invasive placenta. Black arrow shows a direct anastomosis between transmedial interuterine anastomosis and the bladder

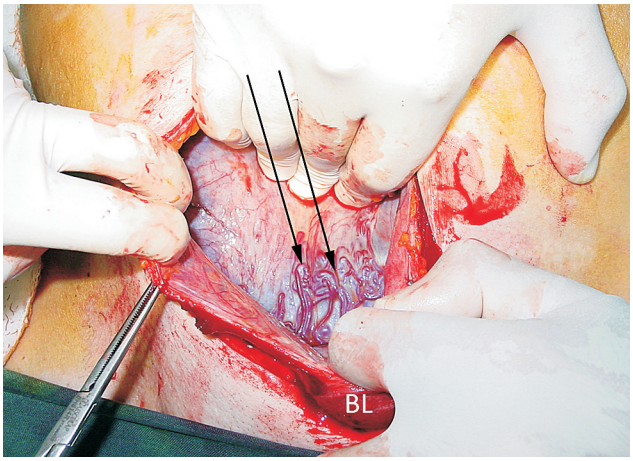

Figure 5 Superior viewing of anterior placental invasion. Black arrows show a group of thin newly formed vessels between the posterior bladder wall and the placenta. BL, bladder

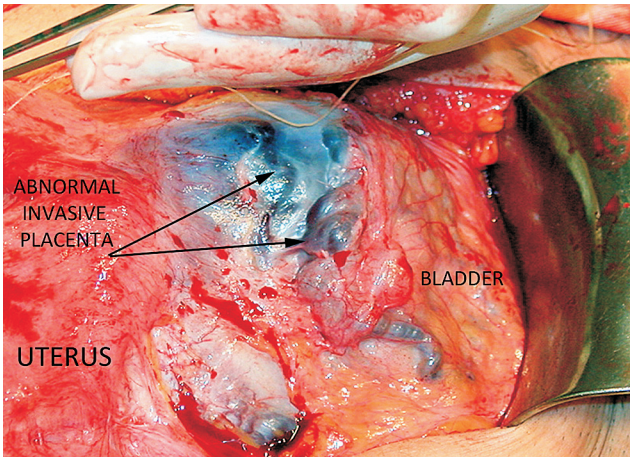

Figure 6 Superior view of anterior placental invasion. Black arrows show evident and thick newly formed vessels between the placenta and the bladder

Uterine anastomotic system

The uterus has two anastomotic systems: an upper one, supplied by the ovarian arteries; and a lower one, supplied by the vaginal arteries (see Chapter 1)[13]. Of the two, the lower or vaginal pedicle is the only one that can maintain uterine vascular integrity when both uterine arteries have been occluded or ligated. It is necessary to be aware, that simultaneous occlusion of the uterine arteries and the inferior vaginal artery (lower anastomotic pedicle) could produce uterine necrosis by over devascularization. This lower anastomotic system supplies collateral blood flow through vessels of a higher caliber than the uterine artery, and it has been shown that it is the major vascular supplementary system when the uterine arteries have been occluded.

The one-step surgery described below makes use of the occlusion of the uterine arteries, since most anterior invasions are not supplied by collaterals of the uterine artery.

ONE-STEP CONSERVATIVE SURGERY

Stage 1: invasion area

Even though ultrasound represents an accurate method to diagnose placenta accreta, pMRI is the most useful technique to establish the exact area (topography) of the invasion. Certain aspects related to the specific location of the invasion are essential when resective surgery is performed, such as parametrial invasion, and presence and location of newly formed vessels.

From a vascular point of view, the blood supply of the female generative tract can be divided into two clearly defined areas. One involves the uterine body itself, labelled sector one (S1), and the other involves the lower uterine segment, cervix and vagina, labelled sector two (S2)[13] (see also Chapter 1). S1 receives its blood supply mainly from the respective bilateral uterine and ovarian arteries; S2, in contrast, is supplied by a series of subperitoneal vessels originating primarily from internal pudendal arteries, secondarily from collaterals of the internal iliac artery and, to a lesser extent, by the uterine arteries. Precise knowledge of the location of the area of invasion makes it possible to plan efficient vascular control. For example, the occlusion of the uterine arteries or ligature of the anterior branch of the internal iliac artery in invasions in the S2 area implies a high possibility of continued bleeding, because their branches arise from the posterior division of the internal iliac artery. This trunk connects the internal iliac system with collaterals of the external iliac and the femoral arteries; therefore, this circumstance leaves only two alternatives to control bleeding: a proximal vascular control at the level of the infrarenal aorta or a specific hemostatic control over tissue and prior to fascial dissection of the pelvis.

Both areas can be identified using images of a mediosagittal pMRI slice[12]. If a perpendicular line is drawn in the medial sector of the posterior side of the bladder, two areas can be delineated, an upper one corresponding to S1 invasions and a lower one, corresponding to S2 invasions. The sagittal plane also provides information on the healthy myometrium above the cervix. In general, abnormal placental invasion near the cesarean scar promotes an anterior bulge. This determines that healthy myometrium will move in a cephalic and caudal direction. The myometrium superior to the placental invasion is easily shown with ultrasound, but this is not the case with the lower myometrium. This detail is essential when planning a conservative procedure, since the absence of a healthy myometrium below the area of invasion – minimum 2 cm – technically reduces the possibility of resecting the invaded myometrium and of performing a safe reconstruction.

Stage 2: surgical scheduling

The literature on the ideal moment to perform elective surgery in placenta accreta is contradictory. Despite this, tacit consensus exists to operate between weeks 35 and 38. This time interval is governed by the possibility of additional fetal lung maturation. There is a statistical increase in complications after week 35 related to placenta percreta; however, it is not always clear whether the authors used the same criteria to define invasion (clinical or histological). The most common complication in placenta accreta is bleeding, which is related to the disruption of the invaded area (uterine distension) attached to the placenta. In cases of anterior invasions, from week 35 the upper edge of the invasion extends beyond the upper wall of bladder. This phenomenon causes a lack of anterior parietal support, producing (spontaneously or through uterine dynamics) an additional disruption in the myometrium with higher risk of bleeding. Also, the dynamic traction on the invaded myometrium could produce variable activation of the coagulation system and, therefore, activate fibrinolysis. This phenomenon may go completely unnoticed and cause marked hypofibrinogenemia during the cesarean surgery. If this alteration is not detected through the quantification of fibrinogen and its degradation products after removing the placenta or performing the hysterectomy, a capillary and continuous hemorrhage occurs. This type of bleeding is very hard to treat, by either compression sutures or endovascular treatment. For this reason, if plasma fibrinogen reaches levels near 200 or 250 mg/dl before surgery, 1 U of cryoprecipitate per 10 kg body weight must be provided, thawed and infused before the surgery begins. This precaution is vital, because both hysterectomy and placental removal generally cause a decrease in fibrinogen levels to between 100 and 200 mg/dl. If fibrinolysis already has begun, a further postpartum physiological reduction would bring the fibrinogen level below its minimum hemostatic level to maintain a stable clot in the placental bed[17]. Because lyophilized fibrinogen is not available in all centers, it is necessary to take into consideration that the time required to use cryoprecipitate

(request, defrost, transport and administer) is not usually less than 45 minutes to 1 hour. Under such circumstances, it would require an excessively and dangerously long time to correct the defibrination associated hemorrhage in conjunction with other technical problems during surgery (see Chapter 4).

The presence of a moderate, profuse or recurrent hemorrhage, as well as active labor, is an indication for termination of pregnancy. Nevertheless, and in spite of the urgency, the best clinical, hematologic and hemostatic control must be provided before surgery commences, as well as the most experienced surgical team.

Emergency

Whether in the presence of an ultrasound diagnosis or diagnostic suspicion due to clinical record and history, it is advisable to start the surgery with four or more units of red cells and plasma available 'in the surgical room'. Promises of immediate supply 'when called' are not acceptable, since the time to request, prepare and transport these agents may be excessive in the presence of bleeding of more than 500 ml per minute which is the norm in this type of abnormal placental invasion.

Basic hemostatic evaluation is recommended, as well as investigation of fibrinogen levels. However, factor-1 quantification is not always available, and in many centers it is only available after significant delays. In order to avoid this inconvenience, a 5 ml blood sample must be obtained prior to the start of surgery and placed in a dry test tube. The tube can be placed in a liquid bath at 37°C or under an assistant's armpit. The sample is examined after 15–20 min; if it produces a clot which remains firm when the tube is shaken, the fibrinogen level is correct. If the clot breaks up when moved, the fibrinogen level is near 100–150 mg%, and it is necessary to request cryoprecipitate immediately. On the other hand, if the blood does not coagulate, its level is below 50 mg%, a fact that indicates a severe fibrinolytic process. In such cases, it is advisable to administer lyophilized fibrinogen or cryoprecipitates and also to request immediately the supply of an equivalent dose to administer during surgery[19].

Although better fluid and blood replacement is under current discussion, we prefer to use two well placed large-bore venous accesses to administer crystalloids or Ringer's lactate solution on a 3:1 ratio with respect to estimated blood loss. Volumetric replacement maintains peripheral oxygenation, protects the microcirculation and avoids multisystemic damage. Administration of fluids and blood replacement must be accomplished early, according to the basic clinical signs and before the development of arterial hypotension, because the compensating mechanisms of the pregnant woman could maintain acceptable levels with blood losses of up to 30–40% of total volume. Since the contents of the circulatory system represent approximately 7% of body weight, the risk of shock is higher in women with a small body mass and in those with previous anemia or bleeding. Common mistakes associated with management of bleeding from placenta accreta include deficiency in recognizing bleeding severity, insufficient fluid administration during resuscitation and delay in stopping the bleeding[19].

Besides the clinical signs of shock, it is absolutely necessary to check the patient's acid–base status using an arterial sample in order to assess the efficacy of resuscitation.

Presurgical measures

Standard blood tests are recommended, including a complete coagulation profile. In normal conditions, this type of surgery is performed under epidural or spinal anesthesia; however, the presence of clinical or subclinical coagulation disorders contraindicates either technique.

Two large-caliber venous accesses are recommended, and the blood bank and laboratory service must be warned of the singular characteristic of this surgery. Because abnormal placentation is a possible cause of exsanguinating hemorrhage, before starting surgery four cross-matched units of packed red blood cells (pRBC) and four bags of fresh frozen plasma (FFP) should be on hand in the surgical room. An additional amount of pRBC, FFP, cryoprecipitate and platelets should be on reserve and ready for immediate use[20]. During surgery, communication between the anesthesiologists and the surgeon must be directed to assessing the estimated volume, the speed of blood loss and avoiding any predictable hemodynamic or hemostatic deterioration.

Stage 3: laparotomy and initial evaluation

Pfannenstiel incision may provide sufficient access if the location of the invasive adherence is known and the surgeon is experienced. However, when vascular control either is or may be required at the aortic level, the essential incision must be median infraumbilical with cephalic extension. Regardless of incision type, the primary objective at this stage will consist of widely exposing the retrovesical space. When this area is apparently adherent to and fused with the placenta, it is not as technically difficult as might be thought (Figure 7). In order to identify the correct place for dissection, the bladder must be elevated with two Allis clamps, a maneuver which will noticeably simplify dissection, repair, ligature and section of the newly formed vessels between bladder-invaded uterus and the placenta. Dissection should start immediately inside the round ligament, from which a small buttonhole can be made, which will include the peritoneum and newly formed vessels, which always must be ligated between double ligatures. The poor muscular layer of the newly formed vessels often allows them to go unnoticed during the pulling maneuvers, since they collapse as veins. If they are inadvertently cut, this can be a cause of postsurgery rebleeding and secondary morbidity.

This stage is slow and requires care, and dissection of the new vessels must be made meticulously, since they are fragile and have high blood flow. On occasions, there is not much tissue between the two ligatures; in this case, an incision on the vesical side is preferred. If the ligature moves or if bleeding occurs, a stitch can be made, which includes the vesical muscular layer with polyglactin 000. On occasions, dissection may be hindered by tissue fibrosis, which makes it difficult to identify a well-defined anatomical plane. In these circumstances, dissecting the vesical surgical plane makes it possible to make a bridge, pass a finger and then exert upward traction. This maneuver, described as a retrovesical bypass, is of great use and allows access to the posterior-lower sector of bladder.

Retrovesical dissection is finished when all newly formed vessels between bladder, uterus and placenta are ligated, and the upper portion of the vagina is accessible. At this moment, hysterectomy must be considered if repair is not considered possible, as might occur when there is segmental tissue destruction of greater than 50% of the organ's axial circumference, or when tissue loss in the distal uterine segment leaves less than 2 cm of healthy segment above the uterine cervix.

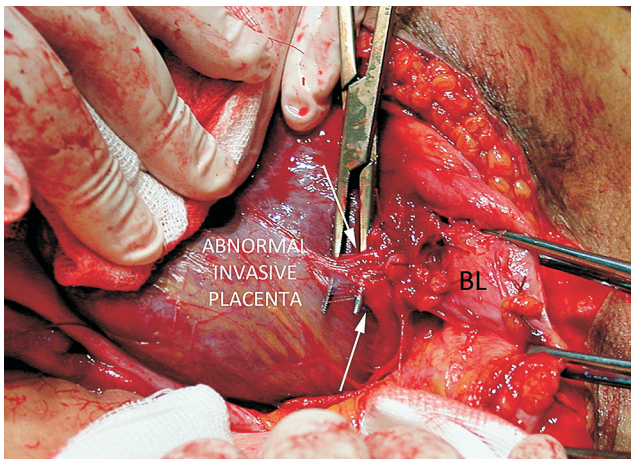

Figure 7 Superior view of anterior placental invasion. After ligature of newly formed vessels the posterior bladder wall is easily dissected. BL, bladder

Stage 4: hysterotomy

Once the anterior side of the uterus has been exposed and the newly formed vesico–uterine–placental vessels have been interrupted, control of the colpouterine pedicle remains when the placenta has been removed. This is the reason why there is no risk of hemorrhage during hysterotomy. Several authors are very careful not to make an incision in the placenta during hysterotomy, and many use intraoperative ultrasound to this aim. Hysterotomy can be performed in the upper sector of the invasive area in a completely safe manner. Even though there is placenta in this area, its insertion is normal and the newly formed vessels have been ligated; therefore, bleeding is not a problem. At this stage, it is advisable to apply the hysterotomy method described for placenta previa[21]. When hysterotomy is performed, only the muscular layer is cut (Figure 8). Next, the hand is introduced between the myometrium and the placenta until the sac is reached. After that, the baby is gently extracted and the uterus exteriorized for better handling.

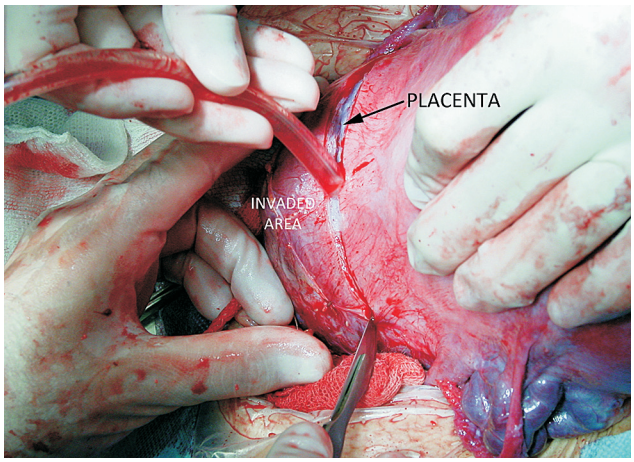

Figure 8 Cutting of the myometrium above the abnormal invasive placenta. Notice that the hysterotomy is performed over the placenta, but not over the newly formed vessels, which were ligated in a previous step

Stage 5: resection and hemostasis

Once the uterus has been exteriorized, the dissection of the posterior side of the bladder can be completed if necessary. This maneuver allows clean access to the upper side of the vagina and uterine cervix, and is essential to obtain hemostasis of the colpouterine vessels. The inferior edge of the invasion can be trimmed with scissors, in order manually to remove the placenta with the invaded myometrium in one piece. Next, the cavity is cleaned with a gauze pad or a Pinard curette (Figure 9). After fetal delivery and placental

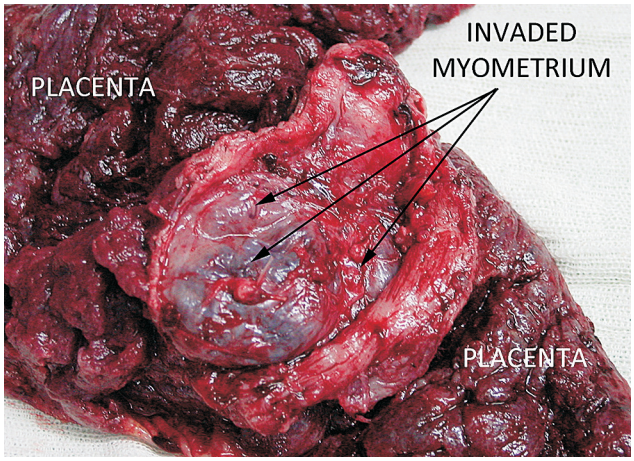

Figure 9 The entire placenta and invaded myometrium are removed in one surgical piece. In this way, uterine reconstruction is performed using healthy tissues

extraction, uterotonic drugs are administered, such as oxytocin (Syntocinon™, Novartis, Brazil) 20 IU i.v. after fetal extraction, plus 20 IU i.v. within the first 24 h, or carbetocin (Duratocin™, Ferring, Argentina) 100 mg i.v. in a single dose.

The colpouterine hemostasis component of the operation is performed with a pair of square stitches following the technique described by Cho *et al.*[16]. With the aim of preventing hematometra, a Hegar's bougy dilator #10 is placed in the uterine cervix, which will move towards one of the sides (Figure 10). In this manner the application of a square stitch is easier, and the potential for accidental closure of the internal cervical os is minimized. The advantage of using Cho's suture relies on the fact that the surgeon works on a surface instead of a specific pedicle. In addition, this procedure prevents inadvertent ureteral injury, since it is applied on uterine tissue. When the hemostatic suture has been placed, any additional bleeding can be checked. Due to the fragility of the newly formed vessels, it is preferable to apply U-stitches in order to minimize the suture cutting effect.

Stage 6: repair

Once hemostasis has been achieved, it is necessary to check that the fibrinogen level is higher than 200 mg%. The repair is performed in two stages. In the first, U-stitches are made with polyglactin suture 1 cm from the myometrial border. The aim with these stitches is to coapt the borders and reduces tension on the primary suture. In the first years of performing this surgery, a reabsorbable net was applied with excellent results; however, the net was not always available. For this reason, another anti-tension mechanism was designed (Vicryl™ mesh (polyglactin 910) Ethicon, USA), which yielded identical results. The second stage is to use a continuous suture with polyglactin 1, which closes the borders and provides secure hemostasis. On occasion, small muscular defects are observed in some areas of the posterior wall of bladder. Such defects as well as any residual focal bleeding are sutured with polyglactin 000 (Figure 11).

When the repair has been completed, the incision and dissection surfaces are inspected one more time, and a sheet of regenerated cellulose is placed as an anti-adhesion barrier between the bladder and the uterine repair.

Stage 7: postoperative care

Special attention must be paid to pain after one-step surgeries, as this type of surgery involves far more tissue disruption than is the case with a cesarean delivery. A useful plan includes morphine administration together with other painkillers starting in the immediate postsurgery period, modified depending on demand, and reduced on the second or third postoperative day.

One additional but important aspect of postoperative care is deep venous thrombosis prophylaxis.

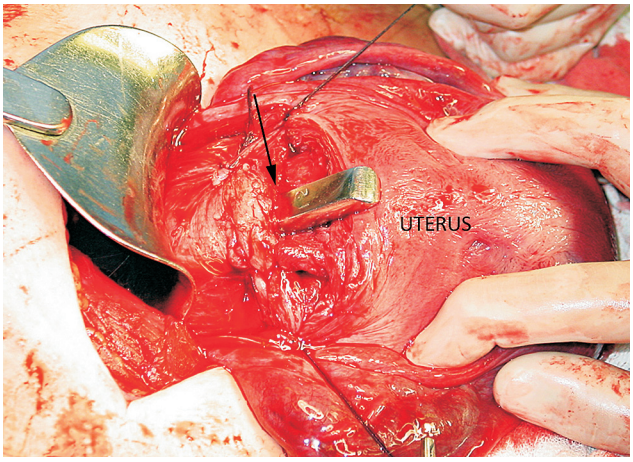

Figure 10 Superior viewing of lower uterus, a Hegar bougy (black arrow) is placed in cervical canal to avoid unwanted lochia occlusion during placement of Cho's hemostatic sutures

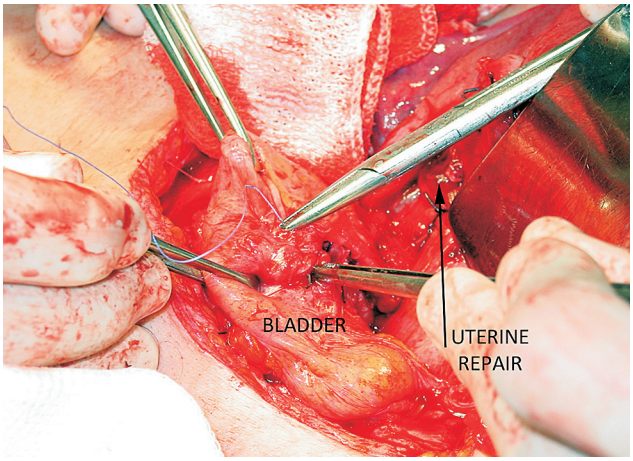

Figure 11 Superior viewing of lower uterus, on the left, the muscular bladder defects is sutured with 000 polyglactin. Repaired uterus (black arrow)

Pregnancy, pelvic surgery and bed rest should be regarded as risk factors[22]. From an ideal point of view, it is advisable to use intermittent pneumatic compressors[23]. Low-molecular weight heparin can be administered when the platelet count is over 100,000/mm^3, and until there is effective ambulation.

FOLLOW-UP

Control uterine studies were performed between 5 and 10 months after repair. The first 100 patients who underwent one-step conservative surgeries subsequently had hysteroscopy to explore the cavity and scar characteristics. The study showed only two cases of uterine synechiae, which were related to the use of chromic catgut. This follow-up procedure was later replaced by uterine T2-weighted MRI performed between 5 and 6 days before patient menses. Uterine MRI makes it possible to observe the repaired area clearly. Prior to the study, moderate urinary retention is requested, which renders a whitish color on T2. This contrast, added to the whitish color of the

premenstrual endometrium, makes it possible to define precisely the repaired myometrium.

SUBSEQUENT PREGNANCIES

To date, 106 subsequent pregnancies have been reported following this type of surgery, two cases of anterior placenta previa coincided with both cases of partial recurrence.

GENERAL OVERVIEW

Placenta accreta is a leading cause of emergency postpartum hysterectomy. Although hysterectomy can be a life-saving operation, it is associated with high maternal mortality and subsequent morbidities[24]. On other hand, conservative treatment leaving the placenta *in situ* has fewer complications of hemorrhage but more of an infectious nature including septic shock, sepsis, infection, endometritis, wound infection, peritonitis, pyelonephritis, vesicouterine fistula and uterine necrosis[25] among other medical complications.

One-step conservative surgery eliminates uterine damage and the entire placenta in the same surgical act. Its technical complexity can be learned with supervised instruction, as was the case with laparoscopy some years ago. Collaboration between countries could help to introduce this procedure as standard treatment for abnormal invasive placenta in the years to come.

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