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CLINICAL CASE

CT angiography as a complementary diagnostic method for the planning of surgery in invasive placenta: case report and literature review

Jésser M. Herrera-Salgado[1], Elena Guzmán-Monteón[2], Gabriel Pérez-Morán[3], Ricardo M. Malagón-Reyes[4], Luis E. Reyes-Mendoza[1], and Jesús C. Briones Garduño[5]*

[1]Intensive Care Unit; [2]Emergency Room; [3]Radiology Service; [4]Acute Medicine; [5]Research Coordination. Hospital Materno Perinatal Mónica Pretelini, Toluca de Lerdo, State of Mexico, Mexico

Abstract

Abnormal placental invasion is a condition related to multiple conditions including previous elective cesarean delivery and its increased frequency. For the obstetrician, it is also increasingly common to face this clinical entity, which has a high morbidity due to organ injury both directly and indirectly during the surgical procedure and not being attended by a highly qualified medical staff. In this paper, we focussed mainly on the surgical approach and its multidisciplinary management and a clinical case on placental percretism attended at the HMPMPS using a complementary diagnostic method (CT angiography) to plan the vascular approach during surgery.

Keywords: *Invasive placenta. Surgical techniques in placental acretism. Critical hemorrhage due to placental acretism. CT angiography.*

Introduction

Placental acretism is a clinical entity that causes major obstetric hemorrhage and is associated with the need for massive transfusion in approximately 40% of treated cases and mortality that varies from 5 to 7% of cases treated[1]. Placenta accreta spectrum (PAS), formerly known as morbidly adherent placenta, refers to the range of pathologic adherence of the placenta, including placenta increta, placenta percreta, and placenta accreta. Maternal morbidity and mortality can occur because of severe and sometimes life-threatening hemorrhage, which often requires blood transfusion. Rates of maternal death are increased for women with PAS[2]. Several prenatal ultrasound signs of PAS were reported over the last 35 years, principally: loss of the clear zone (when the normal hypoechoic retroplacental zone in the myometrium under the placental bed is not visible on ultrasound); myometrial thinning (due to permanent damage of the uterine wall as far as the serosa, with placental tissue reaching the deep uterine circulation); placental lacunae as a numerous, large, irregular sonolucent intraplacental spaces often described on ultrasound giving the placenta a "moth-eaten" appearance in PAS in both transabdominal and transvaginal ultrasound; placental bulge describes the ballooning of the uterus containing the placenta away from its expected plane into the surrounding tissue, usually the bladder; subplacental and/or uterovesical hypervascularity results from excessive dilatation of the uteroplacental circulation beyond the spiral arteries,

***Correspondence:**
Jésser M. Herrera-Salgado
E-mail: drjherrera@gmail.com

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that is, including the radial and arcuate arteries, and is a prominent feature of PAS on prenatal ultrasound; placental lacunae feeder vessels These are seen as vessels with highvelocity blood flow arising from the deep arterial vasculature of the myometrium, that is, radial or arcuate arteries, and feeding the lacunae; and bridging vessels are seen as CD signals arising in the myometrium and appearing to travel beyond the uterine serosa and into the bladder before disappearing. Adherent and invasive placentation may coexist in the same placental bed and evolve with advancing gestation[2]. The purpose of this review is to demonstrate through images the usefulness of CT angiography in the study of the spectrum of placenta accreta, presenting a clinical case of invasive placenta to the bladder. The high morbidity and mortality of this pathology require diagnostic precision and rapidity at the time of having a patient with a known pathology or suspected. The advantages it offers are: more affordable cost for the patient, accessibility of CT scanners in hospital units, allows invasion recognition.

Physiopathology

It was formerly believed that abnormal placental invasion was based on a biological aberration of the trophoblast tending to invade the myometrium abnormally deeply during its placentation process. The current hypothesis is based on a secondary defect at the myometrium-endometrium interface that leads to abnormal decidualization in areas of uterine scar such as by previous cesarean section or instrumented curettages or uterine wall surgeries (myomectomy), which leads to abnormal invasion of the trophoblast. In normal pregnancy, a blastocyst implants into the endometrium, and after delivery, the placenta detaches from the uterus. In PAS, the placenta forms at a site of disruption between the endometrium and myometrium. Placental tissue implants onto the myometrium (accreta), into the myometrium (increta), or through the myometrium to surrounding organs (percreta)[3]. The invasive placenta is closely related to the history of previous surgical disruption which causes disruption of the integrity of the uterine wall (endometrium, myometrium, and perimetrium), the increase in cesarean section operation has a direct relationship with the greater frequency of cases of the invasive placenta, it is also worth mentioning that postpartum endometritis, the history of endometrial curettage are related to placental acretism but with cases of less severity. The most prevailing theory is that prior uterine surgery involving the endometrial–myometrial interface leads to defective decidualization in an area with a uterine scar, allowing the anchoring villi of the placenta to adhere to the myometrium abnormally and further trophoblast invasion. The most prevailing theory is that prior uterine surgery involving the endometrial–myometrial interface leads to defective decidualization in an area with a uterine scar, allowing the anchoring villi of the placenta to adhere to the myometrium abnormally and further trophoblast invasion. Other concepts ascribe PAS to the dysfunction of maternal vascular remodeling in the scarring areas or excessive invasion of the extravillous trophoblast (EVT), which may account for a small part of cases[4]. A new report found in more than 70% of samples, there were thick fibrinoid depositions between the tip of most anchoring villi and the underlying uterine wall and around all deeply implanted villi. The distortion of the uteroplacental interface by these dense depositions and the loss of the normal plane of separation are the main factors leading to abnormal placental attachment. These data challenged the classical concept that placenta accreta is simply owing to villous tissue sitting atop the superficial myometrium without interposed decidua. Moreover, there is no evidence in accreta placentation that the EVT is abnormally invasive or that villous tissue can cross the uterine serosa into the pelvis[5].

Extensive neovascularization is clearly evident in the majority of PAS cases. Tseng and Chou demonstrated upregulation of a number of angiogenic growth factors, including vascular endothelial growth factor (VEGF) and angiopoietin-2, in PAS lysates. Reduced expression of antiangiogenic proteins such as VEGF receptor-2, endothelial cell tyrosine kinase receptor Tie-2, and soluble fms-like tyrosine kinase 1 (sFlt-1) in syncytiotrophoblastic cells from PAS cases compared to normal placenta specimens suggests a proangiogenic phenotype. Severe, early-onset pre-eclampsia is associated with inefficient physiological placental invasion and hypoperfusion, leading to increased sFlt-1 expression and ultimately the clinical phenotype of proteinuria and hypertension. In contrast, invasive placentation results in deep implantation and hyperperfusion, along with suppressed local sFlt-1 expression as demonstrated by decreased expression of sFlt-1 in villous trophoblasts in PAS patients, specifically placenta increta and percreta. PAS-related angiogenesis may not be restricted to the trophoblast. Placental relaxin (RLN) and its receptor (RXFPI) play an important role in angiogenesis in the endometrium by stimulating the expression of VEGF. Increased expression of the RLN

gene and protein has been demonstrated in the PAS basal plate, whereas the receptor RFXP1 is overexpressed in both the basal plate and villous trophoblast in PAS specimens compared to controls suggesting that PAS may produce a number of autocrine and paracrine factors that promote the upregulation of angiogenic-stimulating factors combined with a suppression in antiangiogenic factors, leading to extensive neovascularization[6].

Prenatal diagnosis

The prenatal diagnosis of PAS requires a high index of suspicion. The first step is identifying maternal risk factors. The most significant risk factor for PAS is the combination of a prior cesarean delivery and a placenta previa. Other major risk factors include a prior history of PAS, cesarean scar pregnancy, uterine artery embolization, intrauterine adhesions (Asherman syndrome), and endometrial ablation. Ultrasound is the preferred imaging modality for the prenatal diagnosis of PAS and can be highly accurate when performed by a provider with expertise. PAS can be diagnosed on ultrasound as early as the first trimester. MRI may be considered an adjunct to ultrasound imaging but is not routinely recommended. Recent consensus guidelines outline the ultrasound and MRI markers of PAS[7]. The prenatal MRI is highly accurate at detecting the presence, depth, and topography of placental invasion. All the recorded MRI signs show an optimal diagnostic performance in identifying pregnancies with invasive placentation. MRI and ultrasound do not significantly differ in their ability to detect the presence of invasive placentation, although the difference between the two techniques with regard to assessment of the depth and topography of placental invasion requires further evaluation. Prenatal MRI is highly accurate in diagnosing disorders of invasive placentation. Ultrasound and MRI have comparable predictive accuracy[8].

Tomography has not been described as a complementary diagnostic method in placenta accreta, however, given the need to obtain images of the degree of vascular invasion and adequately plan surgery, it is considered a useful method. It is not contraindicated to have a CT scan during pregnancy. The iodine-based contrast medium has a high safety profile, being classified in category B according to the FDA, the main risk reported is alteration of the development of the fetal thyroid, administering a dose adjusted to 0.7 mL/kg, corresponding to 30% less than the standard dose, due to pregnancy[9].

Surgical techniques in invasive placenta

Retrograde radical hysterectomy

The woman is placed in the lithotomy position and the cesarean is performed by fundal hysterotomy away from the placenta. The ligated umbilical cord and attached placenta are left within the uterus and the hysterotomy is closed with a continuous suture (for hemostasis). The uterus is exteriorized and kept under upward traction so that uterine vascular constriction can diminish blood loss. Direct handling or dissection at the placental site is avoided. The round ligaments are divided and ligated, and the broad ligaments are incised laterally and parallel to the infundibulo-pelvic ligaments to expose the retroperitoneum. The loose areolar tissue encountered in this space is carefully dissected parallel to the ureters and the pelvic sidewall vessels. Stepwise, the devascularization procedure starts with ligature of the anterior divisions of the internal iliac arteries. Next, the utero-ovarian ligaments and tubes are divided and ligated bilaterally. The posterior vaginal fornix is exposed by placement of a sponge stick into the vagina, which is opened transversely, 1–2 cm below the cervicovaginal junction. Roger hysterectomy clamps are used to circumscribe the vagina, sequentially dividing and securing each pedicle with a suture ligature. This technique is similar to the radical retrograde approach used for *en bloc* resection of extensive pelvic disease, such as in women with ovarian cancer. Cesarean hysterectomy is performed using the posterior retrograde approach, in which the ureters are carefully identified, dissected, and preserved through the anterior bladder pillar to keep them out of the field of dissection. The cervix is seized by Museux forceps and pulled up behind the uterus. The retrograde approach is continued by retracting the uterus sharply upward, exposing the remaining cardinal ligament attachments (with uterine vessels) medial to the ureters, uterosacral ligaments, and bladder pillars, which are sequentially divided by clamps and secured with suture ligatures. The vesicouterine space is developed cephalad by blunt dissection until the bladder is completely detached from the anterior aspect of the uterus or the lowermost extent of bladder invasion (usually above the trigone level) has been reached. If the bladder is involved, cephalad blunt dissection of the bladder is stopped. Cystotomy is particularly helpful for defining the dissection planes and determining whether resection of the posterior bladder wall is required. The extent and type of reconstruction may require simple closure of the bladder defect or ureteroneocystostomy

followed by bladder repair. Aortic cross-clamping can be performed prophylactically in cases of suspected percreta or if the woman is hemodynamically unstable[10].

Malagón Reyes technique

This surgical technique is described in two steps:

– Fundic and arciform cesarean section, with fetal extraction, followed by the administration of 6% polidocanol sclerosing solution with the use of a 6 Fr feeding tube that causes sclerosis of the placental bed
– Abdominal hysterectomy with ligation of hypogastric (internal iliac) arteries. There is evidence that such a uterine incision technique greatly reduces trans-surgical blood losses[11].

Esperanza Bautista technique

This technique begins with an incision in the infra-umbilical midline covering an upper umbilical area inclusive, enters the abdominal cavity, externalizes the uterus, and hysterectomy begins, at the time of linking uterine vessels the body cesarean section is immediately performed with a classic technique extracting the fetus, leaving the placenta *in situ* closing the uterus and completing its hysterectomy[12].

Comprehensive and multidisciplinary management

Placenta accreta must be diagnosed antenatally to minimize risks. The American College of Obstetricians and Gynecologists has recommended delivery between 34 0/7 and 35 6/7 weeks of gestation through cesarean hysterectomy to optimize neonatal maturity and minimize the risk of maternal bleeding. It should be transferred to a Placenta Accreta Center of Excellence or a level three or four center for delivery with the aim of improving delivery at these facilities due to the availability of a large, interprofessional team. These teams should include perinatologists, pelvic surgeons, intensivists, general surgeons, urologists, and neonatologists. The patient's hemoglobin level should be optimized before delivery, and there should be coordination with the blood bank to ensure supplies if a massive transfusion should be needed[13,14].

The International Federation of Gynecology and Obstetrics (FIGO) proposed a nomenclature grading system under the umbrella diagnosis of PAS disorders, that replaced the old categorical terminology (placenta accreta, increta, and percreta) PAS Grade1 – non-invasive, PAS Grade 2 – superficial invasion, PAS Grade 3A – deep invasion, PAS Grade 3D – deep invasion with disruption of the serosa, PAS Grade 3E – deep invasion with adherent extrauterine structures[15].

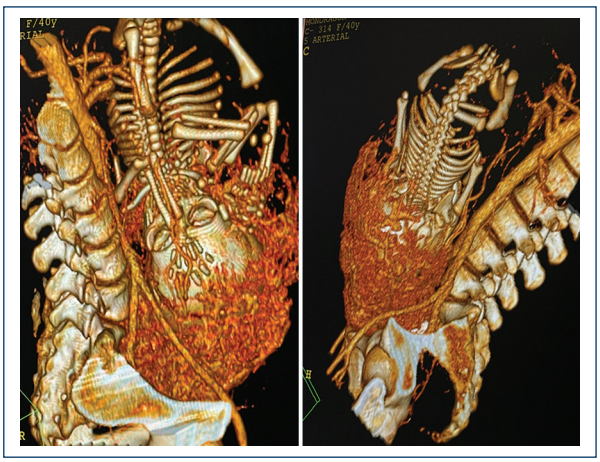

Figure 1. CT angiography of placenta accreta.

The degree of suspected invasion is transcendental for the surgical approach to be planned, the greater the depth of invasion there will be greater the probability of massive hemorrhage. After the procedure, is recommended admission to the intensive care unit to closely monitor for signs of bleeding, hypoperfusion, and fluid overload from resuscitation. Of note, some providers also offer delayed hysterectomy. In this practice, the placenta is left *in situ*, and the hysterectomy is performed at a later time. In the limited number of reported cases, it has been shown to decrease blood loss and decrease the need for transfusion[16].

Clinical case

Proposed Malagón – Pérez PAS Protocol

In this publication, we propose a protocol that includes the following steps:

– Early diagnosis performed by ultrasound maternal–fetal medicine
– Diagnostic cystoscopy when bladder involvement is suspected
– Once the diagnosis of an invasive placenta has been made, we propose CT angiography as a complementary method for the pre-surgical vascular anatomical approach.

Medical history: a 40 years old female patient with no history of personal or family chronic diseases, three previous pregnancies with elective cesarean sections,

Table 1. Test blood results

Day	1	1.1	2	3	4	5	6
Ph (Acidity Index)	7.32	7.38	7.41	7.42	7.43	7.44	7.45
HCO^{3-} (mmoL/L)	17	20	19	21	21	22	23
PCO^2 (mmHg)	31	32	30	31	30	29	28
PO^2 (mmHg)	102	95	90	95	79	79	74
Base ecf (mmoL/L)	–10	–4	–4	–2	–2	–1	–1
Lactate (mmol/L)	4	1.8	1.2	1	1.1	1.1	1
INR	2.1	1	1.2	1	1.1	1.2	1.1
PT (seconds)	16	11.5	12	12	11.5	11.5	11.5
PTT (seconds)	65	29	29	30	31	33	30
Hematocrit (%)	20	25	26	27	28	28	29
Platelets (x $10^3/mm^3$)	107	110	109	118	125	151	154
White blood cells (x $10^3/mm^3$)	16.8	15	15	19	14	14.5	15
Glucose (mg/dL)	80	91	90	89	96	82	87
Creatinine (mg/dL)	0.79	0.7	0.69	0.66	0.9	0.7	0.65
BUN (mg/dL)	12	11	13	14	17	15	13
Urea (mg/mL)	29	27	25	25	24	23	24
Total bilirubin (mg/mL)	1	0.8	0.7	0.45	0.5	0.4	0.41
AST (UI/L)	85	80	78	76	74	53	41
ALT (UI/L)	49	44	41	43	42	41	39
LDH (UI/L)	423	325	335	495	329	291	300
Sodium (meq/L)	137	139	140	141	142	139	140
Potassium (meq/L)	4.6	3.8	3.8	4	4.1	4.1	4.2
Chlorine (meq/L)	110	109	109	108	108	107	108
Calcium (mg/dL)	8.1	8.9	8.9	8.7	8.6	8.3	8.9

HCO^{3-}: denotes Bicarbonate; PCO^2: CO2 partial pressure; PO^2: O2 partial pressure; INR: International Normalized Ratio; PT: prothrombin time; PTT: partial thromboplastin time; BUN: blood urea nitrogen; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase. Source: clinical record.

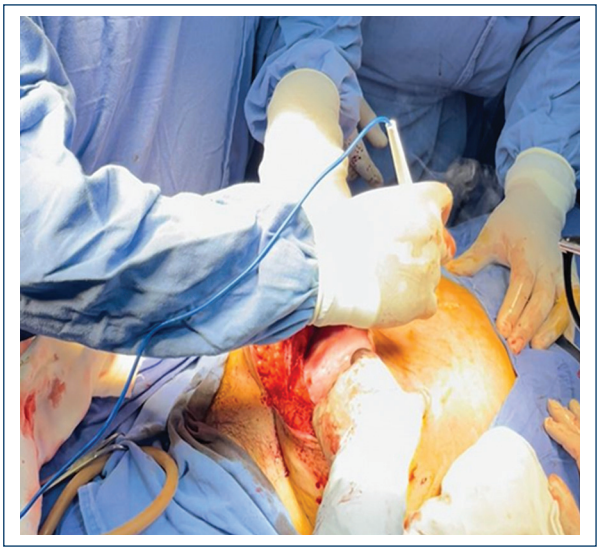

Figure 2. A longitudinal incision in abdominal wall source clinical record.

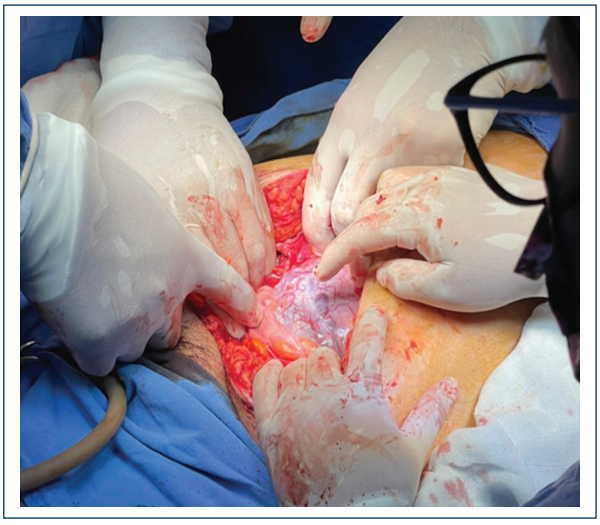

Figure 3. Identification of the area of placental percretism.

no allergies, no previous transfusions or other surgeries, 12 prenatal cares, no genitourinary infections, normal growing fetus, maternal weight gain 9 kg at the beginning of pregnancy, 60 kg at the end of pregnancy. She was admitted to hospitalization for a PAS study protocol, with no general or obstetric symptoms. During her hospital stay, an evaluation was requested by the gynecological urology service, which performed a cystoscopy with a finding of placental percretism. CT angiography was performed (administering infusion of 75.5% iopamirole contrast medium 370 mg/100 mL at

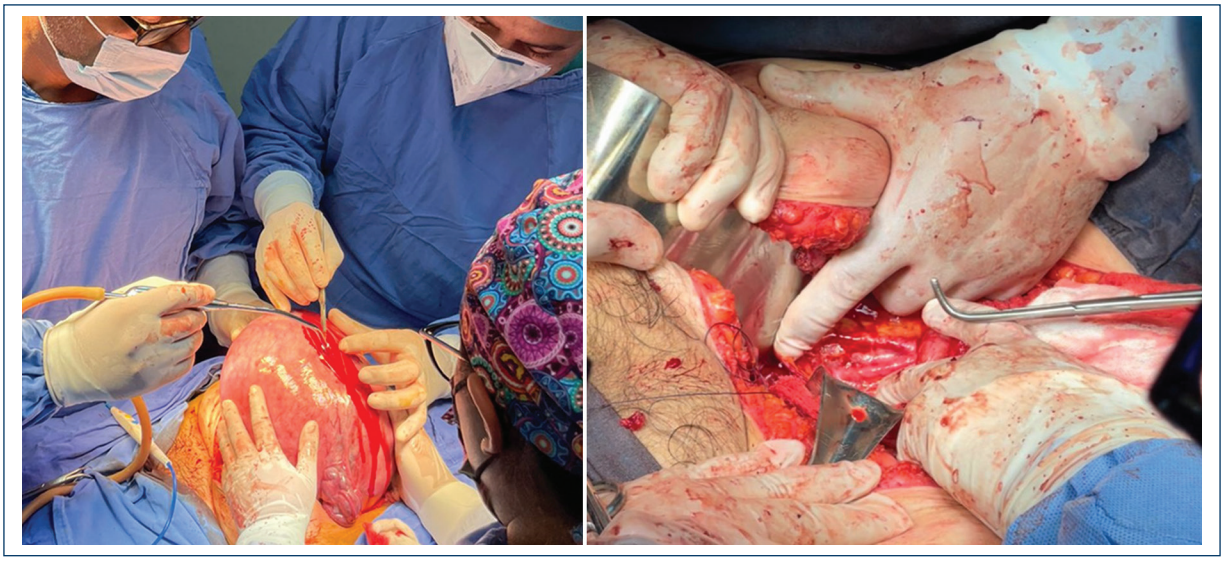

Figure 4. An incision in the fundus avoiding the placenta and large blood vessels, and exposure and ligation of hypogastric arteries.

a dose of 0.7 mL/kg body weight) with the next findings: Placenta that completely occludes the internal cervical orifice, irregularity in its edges that have contact with the bladder, with diminished placental uterine interface and that after intravenous contrast medium, tortuous vessels are identified from the placenta entering the bladder wall (Fig. 1).

Clinic for invasive placentas scheduled cesarean section hysterectomy after coordination with the team of blood bank, obstetric intensive care unit, anesthesiology, and neonatology.

Physical examination summary height 1.55 m, weight 60 kg, body mass index 25 Kg/m^2, body surface area 1.61 m^2. Neurological examination: Glasgow Coma Scale 15 puntos: bilateral pupillary diameter 3 mm, brainstem reflexes and no cranial nerve alterations, unaltered motor sensitivity, and response, preserved mental functions, grade II osteotendinous reflexes. Blood pressure on admission 102/72 mm Hg, mean arterial pressure (MAP) 84 mm Hg, heart rate 62 beats/min, respiratory rate 18 cycles/min, maintained blood pressure in ranges MAP 84-90 mmHg, without requiring antihypertensives nor vasopressors during hospitalization. Without any hemodynamic, respiratory, metabolic, hematologic, or renal abnormality.

Cesarean section was performed under balanced general anesthesia, estimating blood losses of 2000 mL, during surgery, it was necessary to administer norepinephrine at doses of up to 0.8 mcg/kg/min, in addition to transfusing 2 erythrocyte concentrates and 2 plasmas, 1 g of tranexamic acid, 3 g of fibrinogen. Transoperative findings placenta with bladder percretism, abundant neoformation vessels, jellyfish head in bladder plica, vasa previa. Cesarean section hysterectomy was performed with the Malagón Reyes technique, as well as the Gala technique for bilateral ligation of hypogastric arteries (Figs. 2-4). After verification of coagulation, temperature, bicarbonate, and calcium in normal ranges, without evidence of bleeding and no vasopressor infusion needed, the abdominal wall was closed, leaving Penrose drainage. The patient was admitted to the obstetric intensive care unit for surveillance of high-risk surgical puerperium, presenting favorable evolution (Table 1 that summarizes biochemical evolution) with no bleeding greater than usual, no needed vasopressor support nor mechanical ventilation, then was discharged on the 3rd day of puerperium to the obstetrics ward, discharged home on the sixth postoperative day without complications. Treatment received: Enoxaparin prophylactically for 7 days, ceftriaxone 2 g/day for 7 days, analgesic with tramadol for 3 days, then paracetamol for 6 days. Obstetric outcome: Delivery by cesarean section, with estimated bleeding of 2000 mL, product of gestation with 36 weeks calculated by Capurro 2570 grams, feminine with Apgar 8/9, without neonatal neurological impact. The newborn was hospitalized for 2 days in intermediate care and subsequently discharged without complications.

Discussion

Placental acretism is a clinical entity that causes major obstetric hemorrhage and is associated with the need for massive transfusion[1,2]. Moreover, there is no evidence in accreta placentation that the EVT is abnormally invasive or that villous tissue can cross the uterine serosa into the pelvis[5], however, in the experience we have at HMPMPS, cases of pathology have been documented that demonstrate bladder involvement as well as myometrial increstism. In the case presented it was not necessarily massive transfusion as described in numerous serial cases[2,7,8], it was due to a planned surgery achieved with an adequate evaluation of placental invasion.

The usefulness of CT angiography in the study of the spectrum of placenta accreta is not described in medical literature, but it can be considered safe for the fetus and mother[9]. No maternal or fetal renal injury was documented, nor any type of adverse reaction associated with the use of contrast dye during the implementation of the aforementioned diagnostic method.

Many techniques may be performed to treat the spectrum of placenta acreta[10-12], in our care unit, the "Malagón-Reyes" technique is practiced with which favorable results have been documented, as was the case presented.

Considering the above, it is inferred that implementing CT angiography as a complementary diagnostic tool is very useful mainly to understand the invasive vascular anatomical alterations that may occur, achieving the opportunity to plan a safe surgery for the maternal-fetal binomial.

Conclusion

Currently, CT angiography is not recognized in the literature as a diagnostic method for placental accretism spectrum. However, it would be important to introduce it as a complementary diagnostic means, considering that it is safe for the mother and the fetus. In this clinical case, it was shown that it facilitated the planning of the surgical approach, allowed to understand the depth of the placental invasion with greater radiological and angiographic detail, achieving an anticipation of bleeding and allowing a favorable evolution in the patient. Regardless of the technique to be used for the surgical approach, knowing in detail the angiographic placental invasion is a transcendental piece of information for the team that treats patients with placental accretism spectrum.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical disclosures

Protection of human and animal subjects. The authors declare that the procedures followed were in accordance with the regulations of the relevant clinical research ethics committee and with those of the Code of Ethics of the World Medical Association (Declaration of Helsinki).

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

Use of artificial intelligence for generating text. The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript, nor for the creation of images, graphics, tables, or their corresponding captions.

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