Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Anesthesiology

Placental Abruption Complicated by Uterine Apoplexy, Puerperal Hematoma and Haemorragic Shock: A Case Report

Salim Chajai[1*], Hamza Zarouali[1], Nabil El Achhab[1], Meryem Abbouch[2], Sophia Lahbabi[1], Nezha Oudghiri[1], Rajae Tachinante[1]

[1]Maternal Critical Care and Anesthesiology Department, University Hospital Center Ibn Sina, Rabat
[2]Gynecology and Obstetrics Department, University Hospital Center Ibn Sina, Rabat

DOI: 10.36347/sjmcr.2024.v12i06.043 | **Received:** 05.05.2024 | **Accepted:** 13.06.2024 | **Published:** 15.06.2024

***Corresponding author:** Salim Chajai
Maternal Critical Care and Anesthesiology Department, University Hospital Center Ibn Sina, Rabat

Abstract **Case Report**

Placental abruption is a severe complication in pregnancy, causing third trimester haemorrhage. Placental tissue factor triggers coagulation activation with consumption of coagulation factors, and bleeding into the myometrium can cause uterine apoplexy, known as Couvelaire Uterus. We report the case of a patient who developed Uterine apoplexy after placental abruption, leading to coagulopathy and a puerperal hematoma. The patient was managed conservatively and had a favorable outcome, but the case report highlights the importance of early fibrinogen replacement in such patients.
Keywords: Postpartum Hemorrhage, Placenta Abruption, Hypofibrinogenemia, Puerperal Hematoma, Uterine Apoplexy.

INTRODUCTION

Placental abruption is a severe complication in pregnancy, causing third trimester haemorrage. The severity principally depends on the extent of abruption, the latter leading to bleeding, and coagulopathy by increased coagulation activation, coagulation factors consumption, fibrinolysis and dilution in the setting of resuscitation. Bleeding in the myometrium can lead to uterine apoplexy and further deteriorate the situation. We report the case of a patient in whom this sequence of events led to hemorrhagic shock, with a rare association of uterine apoplexy and puerperal hematoma.

CASE REPORT

We report the case of a 19 years old primigravida patient, at her 33th week of pregnancy, admitted to the obstetrical emergency department for a third trimester bleeding. She had no significant personal or family history, and her pregnancy follow up showed no anomaly. Physical examination revealed a conscious patient, with hypertension at 160/100mmHg and a heart rate of 90 beats per minutes. The patient was otherwise mildly pale. Fetal heart sounds were present, obstetrical ultrasound revealed placental abruption and positive fetal cardiac activity. Digital cervical examination found a two fingertips dilated cervix. Obstetricians indicated urgent cesarian section delivery.

Upon admission to the operating room, the patient presented with fetal crowning and imminent delivery. Vaginal delivery of a 1450 grams newborn was thus performed with an episiotomy. The newborn was transferred to neonatal intensive care with Apgar scores of 5-8-8. Artificial delivery with uterine revision was performed, finding an abruption of 50% of the placental surface. The patient received oxytocin for uterine atony prophylaxis, and the episiotomy was sutured.

Results of the blood work made on admission found hemoglobin at 9g/dl, Hematocrit at 35%, Platelets at 90 000/mm3, Urea 0,71g/l, Creatinine 10.3 mmol/l, Fibrinogen 1.4 g/l. The patients was transferred to a high dependency care unit with monitoring for bleeding, adequate uterine tone, blood pressure, heart rate, diuresis and consciousness. The patient presented after one hour a tachycardia at 130 beats per minutes, uterine atony and vaginal bleeding. She received 40 Units Oxytocin, 1 gram tranexamic acid early, and 2 packed red blood cells. Uterine revision showed an intra uterine bleeding, and a decision was made to proceed to surgery.

Patient received norepinephrine on a peripheral venous line initially, and rapid sequence intubation was performed using 150 mcg fentanyl, 60mg ketamine, 1mg midazolam and 80 mg rocuronium. A Ultrasound guided right jugular central venous line and left radial arterial line were taken. Patient received continuous infusion of

tranexamic acid, and anesthesia was maintained intravenously to avoid interference with uterine contraction. Active forced air heating was used to maintain normothermia. Surgical exploration found a Couvelaire's Uterus, with uterine ecchymosis and atony. The obstetric surgery team performed a triple ligation and a B-lynch procedure, which stopped the endouterine bleeding, incision closure was performed after checking for adequate hemostasis. At this stage, the patient received a total of 6 packed red blood cells, 6 fresh frozen plasmas, tranexamic acid and calcium.

The patient then presented a bleeding from the vaginal wall, with a puerperal hematoma on physical examination (Figure1A). She required less norepinephrine but her laboratory work up revealed thrombocytopenia at 28 000/mm3 and hypofibrinogenemia at 1.3g/. Her arterial blood gas showed a pH at 7.35, HCO3- 19.7mmol/l, Base excess -6mmol/l and pCO2 37 mmHg.The patient received 3g fibrinogen concentrate, 4 fresh frozen plasmas, 8 platelet units and continued tranexamic acid. Vaginal packing was performed, as interventional radiology wasn't available for an arteriography and selective embolization.

The patients condition improved and she was weaned from norepinephrine after two extra red blood cells transfusion, the control laboratory work up revealed a fibrinogen at 2.9g/l and platelet count at 98 000/mm3. The packing was removed after 24 hours without additional bleeding and the patient was extubated. On follow up at 14 days the puerperal hematoma showed favorable evolution (Figure 1B).

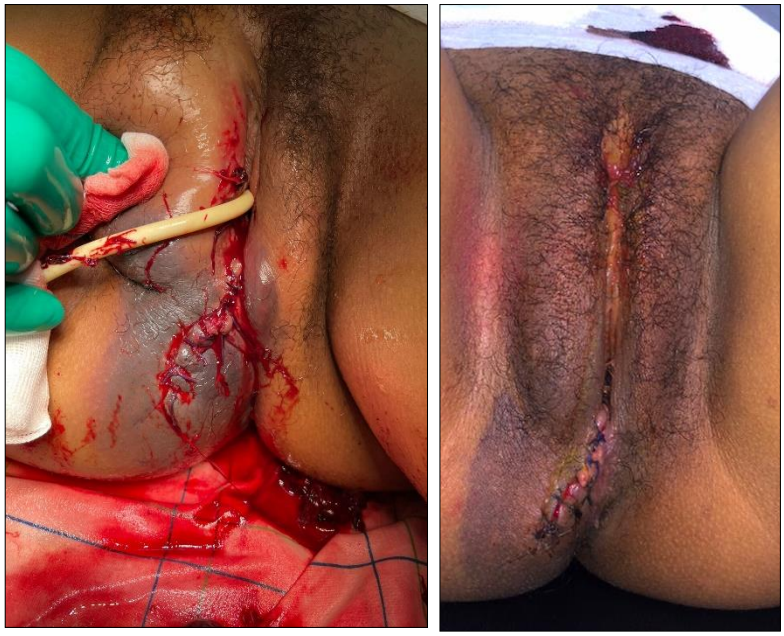

Figure 1 : Puerperal Hematoma. A : operative field. B : At day 14

DISCUSSION

Placental abruption is a serious complication of pregnancy. It involves the premature detachment of a normally inserted placenta. Placental abruption complicates 0.4% to 1% of pregnancies [1]. Two often associated mechanisms are responsible for the onset of placental abruption: the rupture of a decidual artery (vasospasm) or the necrosis of a decidual vein (decidual vessel thrombosis). Page's classification allows for the severity of placental abruption to be classified based on the surface area of placental detachment [2]. The main risk factors favoring the occurrence of placental abruption are preeclampsia, chorioamnionitis, pelvic trauma, smoking, drug abuse, as well as multiparity.

Placental abruption is a complication leading to an increased consumption of coagulation factors as well as fibrinolysis. The intensity of these disturbances is correlated with the extent of placental detachment and the presence of intrauterine fetal death [3]. Tissue factor originating from the trophoblast and decidua, after passage to the maternal circulation, triggers coagulation activation during placental detachment. During pregnancy, the fibrinogen level progressively increases from the first to the third trimester, reaching values of 4-6 g/l, leading to a prothrombotic state aiming at reducing bleeding risk during delivery. Thus, normal levels in the general population should be considered pathological in pregnant women. A fibrinogen level between 2 and 3g/l indicates a high-risk hemorrhagic situation, with the risk of severe post partum hemorrhage being multiplied by 12 when fibrinogen level is lower than 2g/l [4]. Coagulation factors dilution in the setting of fluid resuscitation and blood transfusion also contributes to coagulopathy, which highlights the importance of triggering an early massive transfusion protocol. Moreoever, tranexamic acid, an antifibrinolytic, should be started early and not later than three hours to be efficient. In our patient, the

fibrinogen level was 1.4 then 1.3 g/l, which is explained by increased factors consumption, blood loss secondary to placental abruption and hemorrhage, and dilution. These levels triggered a close monitoring of the patient, and fibrinogen levels lower than 2g/l in the setting of postpartum hemorrhage should be replaced [5].

In the case of our patient, after vaginal delivery, the patient experienced uterine atony despite oxytocin administration. One important causative factor was the acute anemia the patient experienced, as the myometrium needs an adequate oxygen transport to contract efficiently. Uterine revision revealed uterine bleeding, and surgical exploration found an ecchymotic and atonic uterus consistent with uterine apoplexy. Uterine apoplexy, also known as Couvelaire uterus, is a severe form of retroplacental hematomas. It involves extravasation of blood into the myometrium. This clinical entity was first described by Couvelaire in 1911 [6]. However, its incidence remains difficult to estimate since the diagnosis is often clinical, showing a bluish appearance during cesarean section or histological examination during biopsy. It is a diagnostic emergency that complicates 5% of retroplacental hematomas [7]. Outside of a hemorrhagic context jeopardizing maternal prognosis, the management of Couvelaire uterus relies on a conservative approach [8]. In our patient, triple ligation and the B-Lynch technique ensured hemostasis. Avoiding hysterectomy allowed to maintain fertility in our young patient.

The evolution of our patient's case was marked by the onset of vaginal bleeding, and exploration revealed a puerperal hematoma. Puerperal hematoma is among the unusual causes of postpartum hemorrhage, with an estimated frequency of 1 in 1000 deliveries [9]. It involves bleeding from vascular rupture, most often venous, favored by the use of rotational maneuvers or instruments during fetal extraction. Diagnosis is often easier when it involves a vulvovaginal location, while deeper locations may require radiological exploration. Risk factors favoring the occurrence of puerperal hematoma include instrument use, macrosomia, drug abuse, multiple pregnancy, primiparity, the presence of vulvovaginal varices, preeclampsia, and coagulation abnormalities. The role of episiotomy is controversial, some arguing it is a risk factor, while others argue it is protective [10]. Our patient presented with imminent vaginal delivery and this may have contributed to the vaginal trauma. Hemostasis can be achieved either by mechanical compression or by surgical incision with hemostatic suture. In case of failure, selective embolization of the pudendal and inferior gluteal arteries represents the ultimate therapeutic recourse [11]. In our patient, mechanical compression and correction of hemostasis disorders stopped the bleeding.

CONCLUSION

Placental abruption complicated by uterine apoplexy can lead to a dangerous sequence of events. Uterine atony, bleeding and coagulopathy can favor bleeding in other sites, as it was the case in our patient who also developed puerperal hematoma. In any case of postpartum hemorrhage and hypofibrinogenemia (lower than 2g/l), fibrinogen replacement and early hemostatic resuscitation should be intiated.

REFERENCES

1. Baumann, P., Blackwell, S. C., Schild, C., Berry, S. M., & Friedrich, H. J. (2000). Mathematic modeling to predict abruptio placentae. *American journal of obstetrics and gynecology*, *183*(4), 815-822.
2. PAGE, E. W., KING, E. B., & MERRILL, J. A. (1954). Abruptio placentae: dangers of delay in delivery. *Obstetrics & Gynecology*, *3*(4), 385-393.
3. Sher, G., & Statland, B. E. (1985). Abruptio placentae with coagulopathy: a rational basis for management. *Clinical obstetrics and gynecology*, *28*(1), 15-23.
4. Cortet, M., Deneux-Tharaux, C., Dupont, C., Colin, C., Rudigoz, R. C., Bouvier-Colle, M. H., & Huissoud, C. (2012). Association between fibrinogen level and severity of postpartum haemorrhage: secondary analysis of a prospective trial. *British journal of anaesthesia*, *108*(6), 984-989.
5. Vermeulen, T., & Van de Velde, M. (2022). The role of fibrinogen in postpartum hemorrhage. *Best Practice & Research Clinical Anaesthesiology*, *36*(3-4), 399-410.
6. Couvelaire, A. (1912). *Deux nouvelles observations d'apoplexie utéro-placentaire (hémorragies rétro-placentaires avec infiltration sanguine de la paroi musculaire de l'utérus)*. G. Steinheil, éditeur.
7. Habek, D., Selthofer, R., & Kulaš, T. (2008). Uteroplacental apoplexy (Couvelaire syndrome). *Wiener klinische Wochenschrift*, *120*, 88-88.
8. Hubbard, J. L., & Hosmer, S. B. (1997). USA. Couvelaire Uterus. *J Am Osteopath Assoc*, *97*(9), 536-7.
9. Riethmuller, D., Pequegnot-Jeannin, C., Rabenja, C. A., Koeberle, P., Schaal, J. P., & Maillet, R. (1997). A rare cause of postpartum hemorrhage: a genital thrombus. *Journal de gynecologie, obstetrique et biologie de la reproduction*, *26*(2), 154-158.
10. Bienstman-Pailleux, J., Huissoud, C., Dubernard, G., & Rudigoz, RC (2009). Management of puerperal hematomas. *Journal of Obstetrics-Gynecology and Reproductive Biology* , *38* (3), 203-208.
11. Kehila, M., Khephren, S. B., Zeghal, D., & Mahjoub, S. (2013). Prise en charge conservatrice des hématomes puerpéraux de gros volume: à propos de 3 cas. *The Pan African Medical Journal*, 16, 9.