Original Research Paper

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DELAYED PPH DUE TO PLACENTAL SITE VASCULAR SUBINVOLUTION- A CHALLENGE OF UTERUS PRESERVATION

Dr. Tanupriya 3rd year PG resident, Darbhanga medical college, Darbhanga.

Dr. Vandita Thakur 3rd year PG resident, Darbhanga medical college, Darbhanga.

ABSTRACT **Background:** PPH is an important reason for maternal morbidity and mortality. It is directly responsible for one sixth of the maternal deaths. PPH occurring in 24 hrs of delivery is primary, if within 24 hrs to 6 weeks after delivery is secondary. The incidence of secondary PPH is 2% . Placental site Vascular subinvolution is the rarest cause of secondary postpartum haemorrhage. **Methods:** On the basis of histopathological examination and by excluding other differentials the case was confirmed as placental site vascular subinvolution. **Case report:** Here is a rare case of placental site vascular subinvolution (VSI) in a P1L1 woman after uncomplicated spontaneous delivery. Her fertility was desired thus uterus preservation was mandatory. She was treated conservatively including triple uterotonics. A revision curettage was done and material was sent for HPE. **Results:** On H&E dilated "cluster"-shaped myometrial arteries in the placental area partially occluded by thrombi of variable "age" together with the presence of endovascular extra villous trophoblasts confirms the diagnosis. **Conclusion:** Post-delivery, the physiological mechanism of uteroplacental arterial involution is crucial for eliminating the remodelled changes in the vessel and for preventing major blood loss. In placental site vsi there is persistence of interstitial extra villous trophoblastic cells leading to low-resistance , dilated vessels with increased flow. The conservative treatment with triple uterotonics resulted in contraction of placental bed and vessels and reduction in bleeding.

KEYWORDS : secondary postpartum haemorrhage, puerperium, vascular subinvolution, vaginal bleeding ,placental site, uterus

INTRODUCTION

PPH is an important reason for maternal morbidity and mortality. It is directly responsible for one sixth of the maternal deaths. PPH occurring in 24 hrs of delivery is primary, if within 24 hrs to 6 weeks after delivery is secondary. Secondary PPH accounts only for 1% of all labours and its aetiology includes severe endometritis, retained products of conception, gestational trophoblastic disease, uterine artery pseudoaneurysm. Placental site VSI is one of the rare causes and accounts for approx. 13% of all cases of secondary PPH.

MATERIALS AND METHODS

Here is a rare case of placental site vascular subinvolution (VSI) in a woman after spontaneous vaginal delivery where female desired further fertility, hence hysterectomy was to be avoided.

CASE REPORT

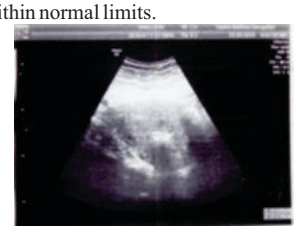
30 yrs female , P1L1 presented with heavy vaginal bleeding on 18th postpartum day. She had spontaneous vaginal delivery of live female baby at 39th gestational weeks, the delivery was uncomplicated. The bleeding was paroxysmal, heavy and profuse and she soaked>10 pads per day. She gives history of her mother having same kind of bleeding on her 20th day postpartum 30 years ago.

Examination

Pt is conscious and well oriented and afebrile. She has BMI of 23.4, pt had PR of 106/min and BP 90/60mmhg .On P/A uterus was subinvoluted, felt above the symphysis pubis(16-18wks) and boggy. Speculum examination shows per vaginal bleeding . No lacerations could be seen, episiotomy wound was healthy.

INVESTIGATIONS

The blood count showed WBC 7000/mcl, Hb 9.1g/l, HCT 32% and PLT 300,000/mcl. On USG uterus was enlarged and showed dialated tortous vessels along inner third of myometrium. (image 1).Pulsed-wave doppler sonography used to confirm the vascular characteristics of an elevated optimum systolic velocity using a low-resistance waveform On doppler PSV >0.83m/s. Bhcg, coagulation profile, LFT and KFT were within normal limits.

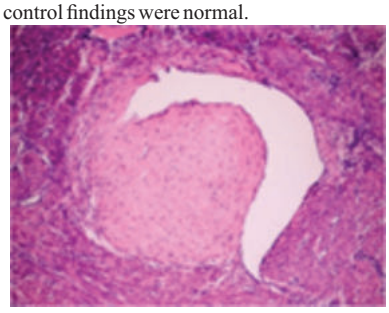


(image 1)

MANAGEMENT

We tried conservative therapy with triple uterotonics (Syntocinone 20IU,Methylergometrin 0.2mg/ml, and prostin 0.25mg).

A revision curettage was done and material was sent for HPE. On H&E dilated clustered myometrial arteries partially occluded by thrombi of variable age, together with the presence of endovascular extravillous trophoblasts which confirms the diagnosis (image 2). One week later USG showed anteverted, involuted ,empty uterus, clear endometrial line. All the control findings were normal.



(image 2)

DISCUSSION

Complex anatomic and physiologic changes occur in the uterine vessels at the time of pregnancy and during the postpartum period. These changes are generally prominent at the placental implantation site. Early in pregnancy (6-10 weeks), placental-derived Extravillous trophoblasts migrate in a retrograde manner and attack the decidual part of the maternal spiral arteries, thereby resulting in the development of endovascular “plugs”. The musculoelastic medial tissues of the arteries are destroyed, whereas hyaline fibrinoid material is deposited. The overall physiologic result of this colonization and remodelling activities is the development of large-calibre vessels characterized by high flow but low resistance, which are enough to meet the needs of the developing placenta and growing foetus. Post-delivery, the physiological mechanism of uteroplacental arterial involution is crucial for eliminating the remodelled changes in the vessel and for preventing major blood loss. In placental site vsi there is persistence of interstitial extravillous trophoblastic cells leading to low-resistance ,dilated vessels with the increased flow. Another crucial mechanism of subinvolution is based on EVT apoptosis, i.e., EVTs seem to lose Bcl-2 expression with decreased apoptosis. Absence of deposition of immunoglobulins (IgG, IgA, IgM) and complement proteins (C1q, C3d, C4) is reported in the walls of subinvoluted vessels. . The review of the literature also revealed the involvement of some nuclear factors identified in controlling human

trophoblast invasion, such as Signal Transducer and Activator of Transcription factors (STATs), Peroxisome Proliferator-Activated Receptor gamma (PPAR-γ), homeobox genes or Wingless and Int-1 (WNT)-dependent transcription factors.17 These factors may contribute to the pathogenesis of pregnancy diseases with abnormal placentation or failed trophoblast differentiation. Thus placental site VSI may be regarded as a condition of spectrum of abnormal placentation, hypertensive disorders of pregnancy being on the opposite end of this spectrum. It is important to note that lesions cannot be differentiated easily from congenital arteriovenous malformations. However, arteriovenous malformations are still defined as high-flow uterine vascular malformations, rather than as subinvolution, which is defined by low-flow uterine vascular malformation. Treatment of patients with SPS includes conservative medical therapy, hysterectomy and fertility-sparing percutaneous embolotherapy. Conservative treatment with triple uterotonics resulted in contraction of placental bed and vessels and thereby reduction in bleeding.

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