

Update on ovarian vein thrombosis

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ABSTRACT

Rare conditions are always challenging due to the difficulty of diagnosis and the fact that there are no guidelines or protocol. As ovarian vein thrombosis (OVT) has an incidence 60 times lower than deep venous thrombosis (DVT), open-mindedness and thinking outside the box is necessary. This short article, looks into the pathophysiology of postpartum ovarian vein thrombosis (PVOT), diagnosis, complications and management.

Keywords: ovarian vein thrombosis, sepsis, pulmonaty embolism

INTRODUCTION

Postpartum ovarian vein thrombosis (POVT) has first been published in 1909. It can remain asymptomatic for many years; during pregnancy, puerperium is an important factor in septic miscarriage, terminations, and extrauterine pregnancies [1].

Causes of maternal mortality are sepsis and pulmonary embolism. POVT presents clinically with abdominal pain, fever, and malaises. Pelvic MRI is the gold standard diagnosis in PVOT which is located in 80% of the cases in the right side. According to Dinnuho [1,2] pulmonary embolism was noticed in 13% of the cases of PVOT and maternal mortality occurred in 4%. As with any rare condition, is difficult to know the exact incidence of PVOT, but different authors reports incidence between 1/2000 to 1/5800, being more prominent in patients with twins and Caesarean Section. As alluded to, OVT (ovarian vein thrombosis) has been diagnosed outside the pregnancy [2,3]. What makes the situation different in pregnancy and puerperium is pyrexia and inflammation in an unwell woman. There are two names that are interchangeable thrombosis or thrombophlebitis. As there is no clinical definition, some authors, enclose PVOT as part of septic pelvic thrombophlebitis (SPT), where thrombosis occur in more pelvic vessels. Recently, OVT has been reported after Coronavirus infection as well [4].

Pregnancy increases the risk of a thromboembolic event four to six times during pregnancy and even more in the puerperium. Virchow's Triade is the driven force behind PVOT [5]. The mean diameter of the ovarian vein at the end of pregnancy is three times bigger than that of a non-pregnant lady, and blood flow is 60 times than the non-pregnant state. After delivery, the sudden flow drop causes stasis which associated with the longer length of the right ovarian vein and multiple valves generates an increased risk of thrombosis. Dextro-deviation of the enlarged uterus, compresses the ovarian vein at the junction with inferior vena cava [6,7]. Left ovarian vein merges with the left renal vein, its length is shorter with fewer valves, so retrograde blood flow is possible postpartum. In different articles, infections have been documented as preceding the PVOT, but it is basically impossible to say, if the infection was genuinely there or PVOT was not diagnosed at the time of infection. Alongside the aforementioned factors, those who are involved in VTE contributes to PVOT as well (Table 1) [8].

PVOT can cause serious morbidity and mortality through sepsis, ovarian infarction, pulmonary embolism clot in the renal vein, ureteral obstruction and hydronephrosis, ultimately with renal failure.

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Article History:

Received: 18 April 2022

Accepted: 28 April 2022

TABLE 1. Risk factors for VTE

Age over 35
Hormonal changes in pregnancy
Instrumental delivery (mid cavity)
Multiple pregnancy
Preeclampsia
Antepartum haemorrhage
Caesarean Section
Stillbirth
Puerperal infection

DIAGNOSIS

Sign and symptoms that appear in PVOT are overlapping a multitude of conditions. As a guide, a patient on antibiotics for sepsis and not improving in the first 48 to 72 hours should undergo imaging diagnosis, which could rule out PVOT. Patients usually present with, abdominal pain, fever, raised PCR and White cell count. Ultrasound has limited benefits as it is patient and operator dependant and bowels distended [9]. Dilated patent veins are normal, in postpartum period. According to one study, specificity, sensitivity and accuracy are rather poor with figures of 55,6%: 41,2% 46,2%. CT has a sensitivity of 77% in diagnosis PVOT while MRI has a sensitivity close to 100%. In MRI and CT, both machines are looking at similar features in multiplane such as Para uterine mass, low attenuation with the lumen, and heightened blood vessel contrast [10,11].

TREATMENT

Surgical treatment has a mortality of 53% and involves the removal of the ovarian thrombi. This was

the most common form of treatment until 1970 [12]. Today heparin and different antibiotics regims are being used, regims that varies from hospital to hospital. Antibiotics should be prescribed for 7-10 days or, 48 hours after clinical improvement. Piperacillin with Tazobactam and clindamycin covers a large spectrum and is suitable in sepsis [12]. LMWH or warfarin is the best treatment prenatally and postpartum. With LMWH, the risk of osteoporosis, heparin induced thrombocytopenia, and bleeding is smaller. In women who do not accept daily injection, warfarin is an alternative and can be administered during breastfeeding. Reverse warfarin effects are more complicated than fraxiparin/clexane (etc). This thing should be kept in mind in case that any type of surgery is necessary. According to other authors, there is no consensus regarding the length of the treatment, ranging from 7-10 days to 3-6 months [13]. IVC Filter is a complex decision and should be made after careful consideration and multidisciplinary team meeting. Randomised study showed steep reduction of pulmonary embolism following IVC insertion, but no change regarding mortality [14].

Risk of recurrence is similar in OVT with other cases of DVT postpartum women should avoid using hormonal contraceptives and in future pregnancies heparin prophylaxis is recommended.

CONCLUSIONS

As we can see, PVOT has multiple and lifelong consequences. Therefore, a high index of suspicion is necessary particularly in cases that do not improve after 72 hours of antibiotics. Furthermore, as obesity becomes endemic and caesarean section is rising across developed countries, PVOT will become more common than it is now.

Conflict of interest: none declared
Financial support: none declared

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