Idiopathic ovarian vein thrombosis: A rare cause of pelvic pain – Case report and review of literature

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Ovarian vein thrombosis is a condition most commonly identified in the puerperium and in association with malignancy, pelvic infections, surgery and thrombophilia. We report a case of idiopathic ovarian vein thrombosis and therefore highlight the importance of considering the diagnosis in women presenting with lower abdominal pain, in whom more common diagnoses cannot be identified.

Key words: abdominal pain, idiopathic, ovary, venous thromboembolism.

Introduction

Ovarian vein thrombosis (OVT) is a rare condition, most commonly identified in the postpartum period.[1,2] Other common associations include gynaecological malignancy, surgery, pelvic inflammatory disease (PID), sepsis and hypercoagulable states.[1–5] Rarely, OVT has no attributed cause and there are only two reported cases worldwide in which OVT was apparently idiopathic.[6,7] Here, we discuss the first reported Australian case of an unprovoked OVT.

Given the potentially serious implications of this condition, prompt and accurate diagnosis is essential. Complications arising from OVT include extension and embolisation of the thrombus and rarely, death.[1,5,6,8] Anticoagulation is the mainstay of treatment.[2,5]

This case highlights the importance of considering OVT as a rare, but important potential diagnosis in women presenting with lower abdominal pain.

Case report

A healthy 42-year-old woman self presented to our emergency department with sudden onset constant central abdominal and right iliac fossa pain associated with nausea. There were no associated bowel or genitourinary symptoms. Her previous medical and surgical history was unremarkable and obstetric history entailed two term normal vaginal births, the last being two and a half years previously. She had no identifiable risk factors for PID and no personal or family history of malignancy or thrombophilia.

On physical examination, the patient was found to be afebrile, normotensive and not tachycardic. Examination revealed mild periumbilical tenderness and moderate tenderness in the right iliac fossa, with local rebound tenderness. There were no palpable masses or organomegaly and bowel sounds were present. Gynaecological examination was unremarkable.

Laboratory investigations including hepatic, renal and pancreatic function were normal. Beta-human chorionic gonadotropin (β-hCG) was negative and urinalysis was clear. Electrocardiogram (ECG), chest and abdominal X-rays were unremarkable. The only abnormalities from the initial investigations were a mild leucocytosis (white cell count 12.6; reference range 4.0–11.0) and an elevation of the C-reactive protein (CRP) to 103 (reference range <5).

Computed tomography (CT) imaging, performed to elucidate surgical causes for her pain, revealed the unexpected finding of right OVT, with no CT evidence of any other cause for the patient's presentation (Fig. 1). The diagnosis was later confirmed by pelvic duplex ultrasound (Figs 2,3).

The patient was admitted to hospital for anticoagulation and further investigations to identify a possible aetiology for the diagnosis of OVT. An intravenous unfractionated heparin infusion was commenced, as well as oral anticoagulation with warfarin. A thrombophilia screen showed no evidence of a coagulation disorder. Serum

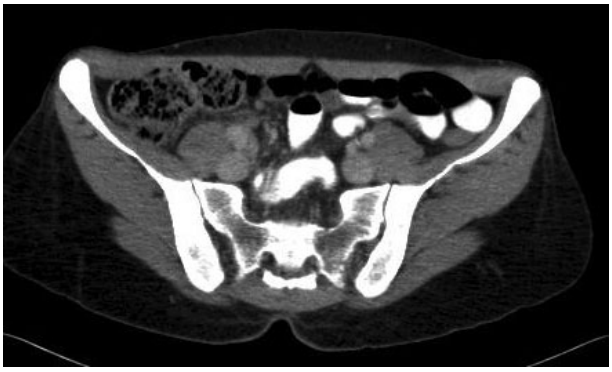

Figure 1 CT abdomen: The inferior aspect of the right gonadal vein is distended and thrombosed.

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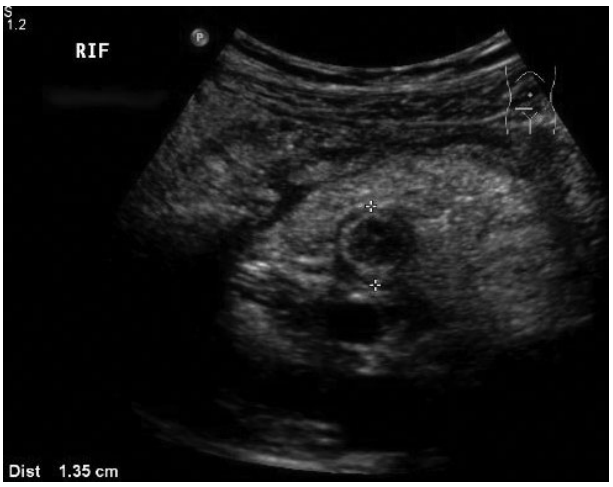

Figure 2 Pelvic ultrasound scan: Tubular hypoechoic right iliac fossa structure, in keeping with ovarian vein thrombosis.

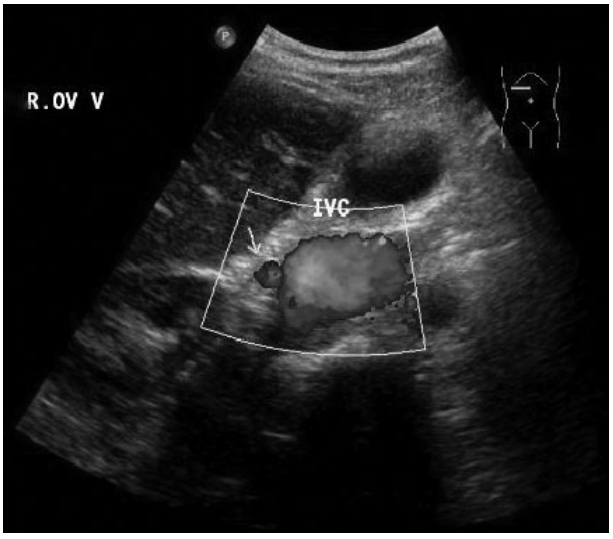

Figure 3 Pelvic duplex ultrasound scan: Colour Doppler imaging demonstrates non-occlusive thrombus in the right ovarian vein.

tumour markers, including carcinoembryonic antigen, CA 19.9, CA 125, and alpha fetoprotein, were within the normal range. Endocervical swabs were negative for *Chlamydia* and gonorrhoea and high vaginal swab culture was unremarkable.

The patient remained clinically stable and was discharged on warfarin once her international normalised ratio (INR) reached a therapeutic level of anticoagulation. Follow-up pelvic ultrasound scan 2 months later revealed complete resolution of the OVT.

Discussion

Ovarian vein thrombosis is a rare condition affecting predominantly women in the puerperium, and especially the early postnatal period.[1,2] The incidence of postpartum OVT ranges from 1 in 600 to 1 in 2000 deliveries.[2,4,9] Ovarian vein thrombosis is less commonly associated with other conditions, including gynaecological malignancy, abdominal and pelvic surgery (especially hysterectomy and salpingo-oophorectomy), PID and thrombophilia.[1–5,10] No risk factors for the development of OVT were identified in our patient.

Patients with OVT often present with non-specific symptomatology or a clinical picture suggestive of alternative, more common diagnoses. Eighty percent of patients with OVT present with fever and 55% have right iliac fossa pain.[9] There may be a palpable abdominal mass on examination.[2,4,6,9] Frequently, these patients are misdiagnosed as having acute appendicitis, particularly given the preponderance for thrombosis to occur in the right ovarian vein.[1,11]

Interestingly, the three reported cases of idiopathic OVT presented with right lower abdominal pain and nausea only. Onset of symptoms ranged from 2 days to 2 weeks. The first reported case was from Turkey in 2005.[6] In this report, a 36-year-old woman presented with a 2-day history of right lower quadrant pain and nausea. In the second case report from the United States of America in 2006, a 27-year-old woman presented with a history of right lower abdominal pain, radiating to the flank, gradually worsening over a 2-week period.[7] All three reported cases of idiopathic OVT were somewhat atypical, given that the patients were apyrexial, and in no case, was there a palpable mass on abdominal examination. Duration of symptoms once therapy was initiated was not well documented in the previous case reports; in our patient, symptoms resolved once anticoagulation was initiated.

There are no firm guidelines for duration of anticoagulation therapy in OVT. An arbitrary period of 6 months seems to be standard in all reported cases, regardless of aetiology. In our patient, repeat ultrasound scan 2 months after diagnosis revealed resolution of thrombus. The decision was made to cease warfarin therapy, with a plan to repeat imaging of the ovarian veins if there were any recurrent symptoms. In the 2005 case report of idiopathic OVT, repeat CT at 40 days post diagnosis confirmed persistent thrombus. Warfarin was continued for 6 months, at which stage, further imaging showed that the thrombus had calcified and therapy was ceased.

It is important to identify OVT promptly and accurately, as the potential sequelae can be severe. Recognised complications include pulmonary embolism, which is reported to occur in 25% of patients with postpartum OVT and thrombus extension into the inferior vena cava and renal veins.[1,5,6,8] Ovarian infarction, sepsis, and death have also been reported in cases of OVT.[1,6]

Although extremely rare, idiopathic OVT does occur. This is the first Australian case report of idiopathic OVT and there are only two other case reports worldwide. However, given the potentially serious complications that can arise as a result of OVT, it is a condition that should be recognised and treated. It is thus imperative that clinicians consider this condition as a differential

diagnosis in women presenting with lower abdominal pain.

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Fetal demise despite normalisation of serum potassium in Gitelman syndrome
Case report and literature review

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Key words: Bartter syndrome, Gitelman syndrome, hypokalaemia, pregnancy.

Case

A 22-year-old nullipara presented at 24 + 5 weeks with gastroenteritis. Booking investigations were normal. Electrolytes were Na^+: 131 mmol/L, K^+: 2.5 mmol/L, HCO_3^-: 31 mmol/L, Mg^{2+}: 1.0 mmol/L and Cl^-: 92 mmol/L. Diuretic or anti-emetic use was denied. Fetal ultrasound was normal. Intravenous (IV) KCl and oral magnesium oxide were supplemented. Normalisation of K^+ took 7 days despite cessation of vomiting.

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Scan at 28 weeks revealed oligohydramnios (Amniotic Fluid Index (AFI): 4.6 cm), growth on third centile and no cardiac activity. She denied membrane rupture. A 1031-g stillborn male was delivered. Autopsy and placental karyotyping were normal.

Despite IV KCl and oral magnesium, K+ failed to normalise (range 2.2–3.3 mmol/L). Serum aldosterone was 72 (pregnancy range 40–60 ng/dL), renin 25 (N: 10–18 ng/mL/h) and morning cortisol 21 (range 4–22 μg/dL). Thyroid functions, brain CT and MRI of the abdomen were normal. Twenty-four-hour urine electrolytes showed normal Cl^- (<20 mmol/24 h) and K^+ 89, (N: 25–150 mmol/24 h) and low Ca^{2+} 40.6 (N: 50–250 mmol/24 h). A diagnosis of Gitelman syndrome (GS) was made.

Daily KCl (120–160 mmol) was given. By day 12 K^+ normalised, but she developed peripheral paraesthesia, decreased sensation and severe proximal muscle weakness. Cranial nerve examination was normal. ECG on day 19

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