Degenerated Retained Product of Conception Misdiagnosed as Invasive Trophoblastic Disease

Ai Peng Tan, MMED (Diag Rad)

Department of Diagnostic Radiology, National University Hospital, Singapore

SUMMARY

Retained products of conception (POC) complicates nearly 1% of all pregnancies, occurring with greater frequency after termination of pregnancy than after vaginal or caesarean delivery. The presenting symptoms of retained products of conception are similar to those of gestational trophoblastic disease and hence accurate differentiation is difficult based on clinical history and physical examination alone. The distinction between these two entities is extremely important as the treatment differs dramatically. These patients often need to be further evaluated with either ultrasound, computed tomography (CT) or magnetic resonance imaging (MRI) of the pelvis. Hence, radiologists play a vital role in clinching the diagnosis although at times it may be challenging to differentiate between these two entities. Herein, we discuss a case of degenerated retained products of conception which was initially misdiagnosed as invasive trophoblastic disease in a 41-year-old woman whom last known pregnancy was 10 years ago.

KEY WORDS:

Retained products of conception; Gestational trophoblastic disease; menorrhagia

INTRODUCTION

Retained products of conception (POC) complicates nearly 1% of all pregnancies, occurring with greater frequency after termination of pregnancy than after vaginal or caesarean delivery. The presenting symptoms of retained products of conception are similar to those of gestational trophoblastic disease and hence accurate differentiation is difficult based on clinical history and physical examination alone. The distinction between these two entities is extremely important as the treatment differs dramatically. These patients often need to be further evaluated with either ultrasound, computed tomography (CT) or magnetic resonance imaging (MRI) of the pelvis. Hence, radiologists play a vital role in clinching the diagnosis although at times it may be challenging to differentiate between these two entities. Herein, we discuss a case of degenerated retained products of conception which was initially misdiagnosed as invasive trophoblastic disease in a 41-year-old woman whom last known pregnancy was 10 years ago.

CASE REPORT

A 41-year-old gravida 6 para 4 woman presented to the emergency department with menorrhagia and intermittent abdominal pain for 2 months. There were no symptoms of

anaemia or significant weight loss. Patient has a regular menstrual cycle of 30 days but presented with menorrhagia with a period length of 7 days. Review of systems was otherwise unremarkable. Patient was hemodynamically stable. No conjunctival pallor was detected. Vaginal examination revealed a bulky cervix. Speculum examination demonstrated a foul-smelling uterine mass.

The serum β -HCG level was 2834 mIU/mL at presentation. Sonography revealed a 7.8-cm intracavitary mass of heterogeneous echogenicities. Peripheral vascularity was detected on Doppler interrogation. Computed tomography (CT) of the thorax, abdomen and pelvis revealed an enlarged uterus with thickened endometrium. Fluid and gas pockets were seen within the endometrial cavity. Magnetic resonance imaging of the pelvis which was performed one day later showed a large intracavitary mass lesion, measuring approximately 8.3 X 6.8 X 6.1 cm. This mass demonstrated heterogeneous signal intensities on both T1 and T2 weighted images with a central area of fluid signal, in keeping with necrosis. Distended serpiginous vessels were seen around the uterine mass. Post gadolinium administration, irregular peripheral enhancement was noted. There was also thinning of the myometrium and obliteration of the junctional zone. No pelvic lymphadenopathy was seen. The initial impression was that of invasive trophoblastic disease in view of the raised β -HCG level. Retained products of conception was deemed less likely in view of the absence of recent delivery or termination of pregnancy.

Patient was admitted and given intramuscular progesterone. Pap smear and endometrial biopsy were performed. Histological analysis revealed mainly blood and fibrinous exudates admixed with neutrophils. There were also scattered aggregates of infarcted chorionic villi and dystrophic calcifications. No vesicles or foetal parts were seen.

Patient subsequently underwent hysteroscopy dilatation and curettage. The endometrial cavity was noted to be irregular and contains a large amount of haemorrhagic tissue. There was massive intra-operative blood loss and the patient's haemoglobin dropped from 9 g/dL to 4g/dL. Intravenous oxytocin and intramuscular ergometrine were administered. Ribbon packing of the uterine cavity was performed with bimanual uterine massage for 20 minutes. The bleeding was eventually controlled. Patient was monitored in the intensive care unit for 3 days. On post-operative day 3, the ribbon packs were removed. Patient recovered well and was discharged with oral antibiotics.

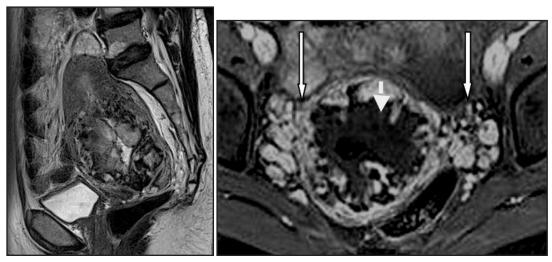


Fig. 1a, b: Sagittal T2-weighted (a) and axial T1-weighted post contrast (b) images of the pelvis demonstrate a mass of heterogeneous T2 signal with peripheral nodular enhancement and a central area of fluid signal, in keeping with necrosis (short white arrow). Distended serpiginous vessels (long white arrows) are seen around the uterine mass.

During follow up in the outpatient clinic, patient remained well with no further per vaginal bleeding or abdominal pain. Serum β -HCG level at follow up 2 weeks later remained slightly elevated at 18.6 mIU/mL.

DISCUSSION

Retained products of conception (POC) complicates nearly 1% of all pregnancies, occurring with greater frequency after termination of pregnancy than after vaginal or caesarean delivery.1 The presenting symptoms of retained products of conception are similar to those of gestational trophoblastic disease² and hence accurate differentiation is difficult based on clinical history and physical examination alone. The distinction between these two entities is extremely important as the treatment differs dramatically. Retained products of conception may be treated conservatively or with curettage whereas gestational trophoblastic disease may require chemotherapy.³ The serum β-HCG level characteristically remains elevated in patients with gestational trophoblastic disease while the hormone falls to an undetectable level over 2-3 weeks in patients with retained POC. In our case, the serum β -HCG level was markedly elevated, unusual for retained POC. The raised serum β -HCG level in our described case may be due to the presence of viable trophoblastic tissue, seen as the enhancing elements on MRI. On the other hand, there are histologic subtypes of gestational trophoblastic disease in which serum B-HCG levels are normal or only slightly elevated, including placental site trophoblastic disease. Hence, undue emphasis should not be placed on serum β -HCG level when attempting to differentiate between retained POC and gestational trophoblastic disease.

Retained POC appears on MR imaging as an intracavitary uterine soft tissue mass with variable T1 and T2 signal intensities, variable amounts of enhancing tissue and variable degree of myometrium thinning and obliteration of the junctional zone.³ These findings overlap with those of gestational trophoblastic disease, rendering it challenging for the radiologist to make a definitive distinction between retained POC and gestational trophoblastic disease. Abnormal uterine vasculature is a recognized feature of gestational trophoblastic disease but is noted in our case, one of the few reasons why an initial diagnosis of invasive trophoblastic disease was made.

In our case, the initial misdiagnosis of invasive trophoblastic disease can be attributed to several compounding factors such as the similar presenting symptoms and imaging findings as well as the markedly elevated serum β -HCG level and absence of recent delivery or termination of pregnancy.

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