

Massive Ascites – An Uncommon Presentation of Endometriosis

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ABSTRACT

Endometriosis as a cause of massive ascites is very rare. It is seldom possible to arrive at the diagnosis without surgical exploration. We describe a case of this entity which presented to us as a surgical problem. This proved to be a diagnostic dilemma to us and our colleagues in other disciplines, with the diagnosis being made based on histology after surgical exploration. The literature is reviewed and we hope our experience in this case will increase the awareness of this uncommon entity.

Keywords: Endometriosis, ascites

Singapore Med J 2003 Vol 44(2):098-100

CASE REPORT

SBS was a 41-year-old Malay female with a history of atrial septal defect repair at age of eight years. She was married with one child. She complained of worsening abdominal distension of more than six months' duration. There was no loss of appetite or weight. Her menstrual periods though heavy, were regular and were not associated with dysmenorrhoea. She did not experience dyspnoea. There was no recent overseas travel and she denied any family or past history of tuberculosis.

Clinically, she looked well. Vital signs were stable and jugular venous pressure was not raised. Lung auscultations were clear. Heart auscultation revealed a grade 3/6 early systolic murmur at the pulmonary area. Gross ascites was noted with no palpable abdominal mass. Pedal or sacral oedema was absent. Findings on digital rectal and vaginal examinations were unremarkable.

Evaluation of her ascites included normal results for complete blood count, electrolyte levels, renal and liver function tests, serum albumin and protein levels. Oesophagogastroduodenoscopy (OGD) showed antral gastritis and colonoscopy was normal. Serum lipase was normal and fecal fat was negative. Computed tomography (CT) of

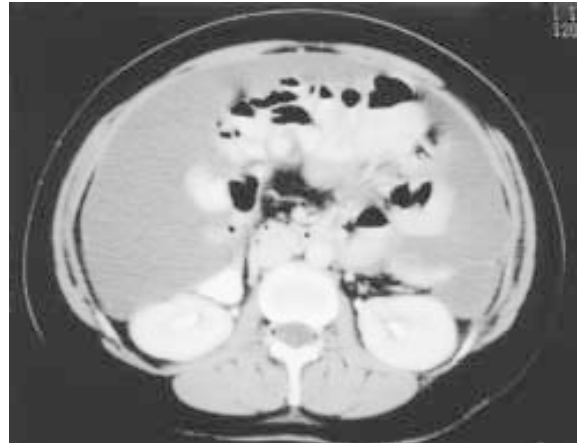


Fig. 1a CT scan of abdomen shows gross ascites with no other significant abnormality.

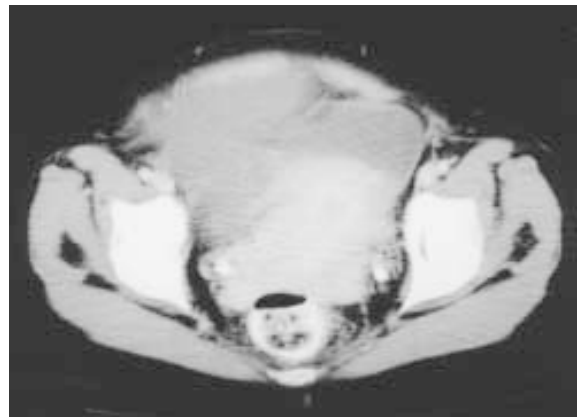


Fig. 1b CT scan of pelvis shows gross ascites, and enlarged and poorly-defined uterus.

abdomen and pelvis (Fig. 1a and 1b) showed gross loculated ascites with right pleural effusion and bilateral atelectases. The pancreatic tail and distal body were not visualised. Liver cysts were present. Spleen, kidneys, gallbladder and bowel loops all looked normal. No evident lymphadenopathy was present. The uterus appeared enlarged and poorly-defined. Findings at a gynaecological consult, including an ultrasonography of the pelvis were normal. Tumour markers including CA-125, CA 19-9 and carcinoembryonic antigen (CEA) were all normal. Diagnostic paracentesis yielded brownish fluid and cytology was that of inflammatory cells, with

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Fig. 2a Finding a cocoon during laparotomy, after 5.6L of ascitic fluid was drained.



Fig. 2b Closer view of the peritoneal cavity and its contents.

no malignant cells seen. The pH of the ascitic fluid was 8.3, with specific gravity of 1.034, total protein of 58.0 g/L, albumin 31g/L, lactate dehydrogenase (LDH) 1470, glucose of 4.0 mmol/L and amylase was 26 U/L. These results were again inconclusive. No leucocytic count was possible, as the sample was heavily blood-stained. This specimen yielded no bacterial or fungal growth; acid-fast bacilli smear and polymerase chain reaction for mycobacteria were all negative. Autoimmune markers like anti-ds DNA antibody, ANCA antibody and anti-nuclear antibody were all negative. Magnetic Resonance Imaging (MRI) cholangiography did not yield any significant new information and 2D-echo of the heart showed moderate aortic stenosis with normal left ventricular dimension and a calculated left-ventricular ejection fraction of 56%.

A diagnostic laparotomy was subsequently performed. We found massive haemorrhagic ascites totaling 5.6 L. The liver was noted to be densely adherent to abdominal wall. The omentum, viscus and bowel were totally matted down forming a cocoon with no planes for any attempts at dissecting (Figs. 2a and 2b). In the pelvis, the patent fimbrial end of a fallopian tube was in communication with the peritoneal cavity, while the rest of the organs were coated with the same thickened peritoneum. The ascitic fluid was sent for analysis and peritoneal biopsy was taken. Histology of the peritoneal biopsy was reported as endometriosis. She recovered well from the laparotomy and was referred to the gynaecologist for medical therapy.

DISCUSSION

Endometriosis is diagnosed with the identification of endometrial glands and stroma in extra-uterine locations. Till date, the pathogenesis remains unelucidated. The classical explanations include the coelomic metaplasia theory postulating transformation

of coelomic epithelial lining of peritoneal cavity and the transplant theory citing retrograde menstruation as the causation for the condition. Newer findings of genetic predisposition⁽¹⁾, immune dysfunctions with increased level of auto-antibodies⁽²⁾ in patients with endometriosis, the possible role of environmental toxins like dioxin⁽³⁾ and plausible molecular aberrations with expression of aromatase P450 from endometrium with endometriosis continue to add new variables to the yet unsolved equation.

First described by Brews in 1954⁽⁴⁾, large bloody ascites associated with endometriosis remained a rare condition. Till 1995, only 22 cases were reported in the English literature⁽⁵⁾. Endometriosis associated with ascites was found to be more common in black, nulliparous women⁽⁶⁾. Our patient was married with one child and had regular menses with no dysmenorrhea or pelvic pain, without any suggestion of gynaecological disease. Her previous cardiac surgery served as a confounding factor, although clinically she was not in cardiac failure. She was thus presented to us as a surgical problem. It became a diagnostic puzzle when investigations performed for all possible differentials were all non-conclusive. At laparotomy, dense adhesions were encountered and the bowel was essentially in a cocoon prohibiting further dissections or adhesiolysis, both of which were not warranted as patient did not have intestinal obstruction. There has been a report from Milan of a case of idiopathic sclerosing peritonitis associated with florid mesothelial hyperplasia, ovarian fibromatosis, and endometriosis with ascites – an association not previously described⁽⁷⁾. A 35-year-old women with massive ascites and intestinal obstruction underwent laparotomy with small bowel resection, hysterectomy and bilateral salpingo-oophorectomy. The diagnosis was made post-operatively on histology. This condition resembles the cocoon we encountered at laparotomy. Unfortunately, no bowel

wall specimen was obtained to allow for histological comparison. We did not aspirate the right pleural effusion for diagnostic evaluation. The occurrence of right-sided pleural effusion is thought to be due to a small communication between pleural and peritoneal cavities and/or overload of the lymphatic drainage of the peritoneal cavity through the right thoracic duct⁽⁸⁾.


The first-line treatment of endometriosis is medical therapy, directed towards the oestrogen responsiveness of endometriosis⁽⁹⁾. A state of pseudopregnancy (using oral contraceptives or progestins) or menopause (using gonadotropin-releasing hormone agonists) is induced. Alternatively, danazol which directly inhibits steroidogenic enzymes, endometriotic implant growth, pituitary gonadotropin secretion, and interacts with androgen and progesterone receptors could be used for the treatment. Surgery may be indicated only if patients failed medical therapy or have extensive endometriosis such as endometriomas.

This is an interesting case of endometriosis presenting to us as a surgical problem of ascites, which challenged and baffled us and our colleagues in other disciplines. Many non-gynaecologists may not be

aware that endometriosis can be a very rare cause of ascites. It is our hope that this case report will be useful to our colleagues.

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