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ENDOMETRIOTIC ASCITES: A VERY RARE PRESENTATION OF PELVIC ENDOMETRIOSIS

G. Obajimi and O. Awolude

Department of Obstetrics and Gynaecology, College of Medicine, University of Ibadan, Nigeria

Correspondence:
Dr. G. Obajimi
Dept. of Obstetrics and Gynaecology,
College of Medicine,
University of Ibadan,
Nigeria.
E-mail gbolahanobajimi@gmail.com

ABSTRACT

A 30 year-old P0^{+1} lady who was referred to the gynaecology clinic on account of inability to conceive for 8 years duration and progressive abdominal distension of 2 years duration. She had a history of severe cyclical dysmenorrhoea warranting occasional hospitalization. An abdomino-pelvic ultrasound revealed marked intra-abdominal collection. The uterus and ovaries appeared normal. She subsequently had laparoscopy and drainage of 6 litres of endometriotic ascites. Both fallopian tubes were diseased. She was followed up on an out-patient basis with sub-cutaneous goserelin injections and referred for assisted reproduction.

Keywords: Endometriosis, Ascites, Infertility, Laparoscopy

CASE

A 30 year-old P0^{+1} lady who was referred to the gynaecology clinic, whose complaints were inability to conceive for 8 years duration and progressive abdominal distension of 2 years duration. She had a history of severe, chronic cyclical dysmenorrhoea warranting occasional hospitalization. There was no history suggestive of weight loss, nausea, vomiting or change in bowel habit. However, she experienced early satiety and occasional bloating. She attained menarche at 14 years and menstruated for 5 days in a regular 30 days cycle. Her sexual debut was at 17 years. She had been on combined oral contraceptive pills (microgynon) in the preceding 12 months. She was the 2nd wife of a polygamous union. The other two wives had two children each. There was a history of voluntary termination of pregnancy about 8 years earlier.

Clinical examination revealed a healthy young lady with a distended abdomen. Fluid thrill was positive. Digital rectal examination was essentially normal. Vaginal examination was difficult and unremarkable due to the distension, limiting access to the uterus and adnexae. Her vital signs were normal. Laboratory investigations revealed a packed cell volume of 38%, white blood cell count of 14,600/mm^3, (Polymorphonuclear neutrophils were 90%, lymphocytes were 7% and monocytes 3%). Liver function tests, Electrolytes & Urea were within normal limits. Her Carcinoma antigen -125 was markedly elevated at 118u/ml (Reference <35). A chest X-ray was performed to exclude possible pleural effusion and was essentially normal.

Abdominopelvic ultrasound revealed marked abdominal collection with normal looking uterus and ovaries. There were no pelvic masses and other abdominal organs were unremarkable. A diagnosis of massive ascites probably due to an intra-abdominal malignancy was made in a patient with background history of infertility. In view of her stable clinical condition and reassuring abdomino-pelvic ultrasound, a decision was taken to further evaluate the peritoneal cavity. She subsequently had laparoscopy and drainage of 6 litres of endometriotic ascites. Findings were massive chocolatey ascites, dense pelvic adhesions, multiple endometriotic deposits along the anterior abdominal wall, pelvic side wall, large bowel, ovaries, uterus and the Pouch of Douglas. The ascitic fluid was sent for cytology and she was commenced on medical management for endometriosis with subcutaneous goserelin injection (zoladex) 10.8mg every 13 weeks for 6 months.

Cytology revealed sheets of epithelial cells and fragments of loosely arranged spindled stroma. There was no atypia. She was followed up at the gynaecology clinic for 12 months and she demonstrated sustained clinical improvement and was referred for assisted conception in view of the tubal disease.

DISCUSSION

The occurrence of endometriotic ascites is extremely rare[1] and the first documented case was by Brews in 1954[1]. Few studies have been reported since then. Appleby *et al.* in 2014[2] reported a case of intestinal endometriosis occurring in the presence of haemorrhagic ascites. A similar finding of haemorrhagic ascites managed by drainage, gonadotrophin releasing hormone analogues (GnRH) and

subsequent successful assisted reproduction was reported in 2014 by Bignall and colleagues[3].

Various theories have been propounded for this rare presentation and has been suggested to include the continuous release of endometrial cells from a ruptured chocolate cyst into the peritoneal cavity. Pelvic endometriosis is a recognised cause of low grade pelvic inflammation with varied manifestations[4]. Acute abdomen may result from chronic peritoneal irritation via chemical inflammation induced by the endometrioma[5]. The chemical irritation caused by these deposits leads to increased white blood cell count, elevated C-reactive protein and carcinoma antigen - 125 levels. This may mimic the presence of an intra-abdominal malignancy and may pose a challenge to clinical diagnosis. The most common endometriotic sites, in decreasing order, are the ovaries, anterior/ posterior cul-de-sac, broad ligaments, uterosacral ligaments, uterus, fallopian tubes, sigmoid colon and appendix. The symptoms of endometriosis are often correlated with the site of the implant and an unusual presentation of hemoperitoneum has been described in literature[6,7].

It is estimated that about 10-15% of reproductive aged women suffer from pelvic endometriosis[8]. Dysmenorrhoea, deep dyspareunia, dyschezia and dysuria are the most frequently reported symptoms. Despite its prevalence, the disease is still poorly understood, evidenced by lack of diagnostic blood tests and inconclusive evidence of a relationship between the extent of the disease and its symptomatology[8]. Several pathogenic theories have been proposed and none of these theories have been able to entirely explain the clinical presentation of the various types of endometriosis[9]. The retrograde menstruation theory provides the most lucid explanation of the aetiopathogenesis of pelvic endometriosis, suggesting a transtubal retrograde flow of endometrial fragments onto the peritoneum and abdominal organs. It has been suggested that the number and amount of menstrual flow in conjunction with both genetic and environmental factors, determine the degree of phenotypic expression of the disease[9].

The association between infertility and endometriosis is well established in literature, but a definite cause-effect relationship remains controversial[10]. The prevalence of endometriosis increases to as high as 25%–50% in women with infertility and 30–50% of women with endometriosis have infertility[11,12]. Severe pelvic endometriosis is known to distort pelvic anatomy, impair oocyte release and pick-up, alter sperm motility as well as fertilization and embryo transport[13]. The role of mild disease however remains elusive and may be related to the expression of inflammatory cytokines, growth and angiogenic factors as well as the expression of aberrant genes[13].

Our patient had secondary infertility and had never been investigated. She only presented because of the discomfort associated with the progressive abdominal swelling. Late presentation is not an uncommon finding in the tropics where health insurance is limited. She had a history of chronic pelvic pain and this should have been an early warning sign of endometriosis. The presence of gross ascites and elevated carcinoma antigen-125 values made it imperative to screen for the possibility of an intra-abdominal malignancy via an abdominopelvic ultrasound, which did not reveal any pelvic masses. The decision to do laparoscopy was premised on the need to further evaluate the peritoneal cavity, drain the ascites and obtain tissue biopsy. Laparoscopy has the advantage of minimal tissue handling as well as providing a panoramic view of the peritoneal cavity.

Medical management was instituted in this case in view of the extensive endometrial deposits and the desire to conceive. This aims to create either a pseudo pregnancy or a pseudo menopausal state thereby halting the progression of the irritation from the endometriotic deposits. The pseudo menopausal state was favoured in this case and goserelin (zoladex) a GnRH analogue was the drug of choice. Goserelin is useful in pituitary downregulation in preparation for assisted conception and has been shown to be a quicker, more convenient and effective alternative to multiple doses of buserelin (suprefact)[14].

CONCLUSION

Endometriosis is prevalent amongst infertile women and may present in a bizarre manner. Endometriotic ascites is very rare and may pose a diagnostic challenge in resource poor settings where delayed presentation is quite common. Excluding intra-abdominal malignancies and instituting appropriate treatment in a timely fashion would halt the progression of the disease. Early recourse to assisted reproduction should be considered in patients with extensive disease and concomitant tubal disease.

REFERENCES

1. **Samora-Mata J,** Feste J.R. Endometriosis Ascites: A case report. JSLS (1999) 3:229-231
2. **Appleby R,** Saroya H, Postgate A, Meer Z. A young woman with abdominal distension. BMJ Case Reports. 2014; doi:10.1136/bcr-2014-203726
3. **Bignall J,** Arambage K, Vimplis S. Endometriosis: a rare and interesting cause of haemorrhagic ascites.

BMJ Case Reports. 2014; doi:10.1136/bcr-2013-010052
4. **Halme J.** Role of peritoneal inflammation in endometriosis-associated infertility. Ann N Y Acad Sci. 1991; 622:266-274
5. **Shinichi T,** Hiroaki K, Maki H, *et al.* A very rare case of endometriosis presenting with massive haemoperitoneum. JMIG 2015; 22(4):691-693
6. **Taub WH,** Rosado S, Kalaycioglu M, *et al.* Haemorrhagic ascites secondary to endometriosis. J Clin Gastroenterol 1989;11(4):458-460
7. **Philip A,** Fiadjoe P, Reddy K. Massive haemoperitoneum due to uterine artery erosion by endometriosis, a literature review. Eighteenth FIGO World Congress Book of Abstract. Monday November 6, 2006; 66.
8. **Mehedintu C,** Plotogea M.N, Ionescu S, Antonovici M. Endometriosis still a challenge. J Med Life. 2014 Sep 15;7(3):349-357
9. **Vercellini P,** Vigano P, Somigliana E, Fedele L. Endometriosis: pathogenesis and treatment. Nat Rev Endocrinol. 2014 May;10(5):261-275.
10. **Matthew LM,** Hugh ST. Endometriosis and Infertility: A review of the pathogenesis and treatment of endometriosis-associated infertility. Obstet Gynecol Clin North Am. 2012 Dec; 39(4): 535–549.
11. **Verkauf BS.** Incidence, symptoms, and signs of endometriosis in fertile and infertile women. The Journal of the Florida Medical Association. 1987;74(9):671-675
12. Endometriosis and infertility: a committee opinion. Fertility and sterility. 2012;98(3):591–598
13. **Holoch KJ,** Lessey BA. Endometriosis and infertility. Clinical obstetrics and gynaecology. 2010; 53(2):429-438
14. **Oyesanya OA,** Teo SK, Quah E, *et al.* Pituitary down-regulation prior to in-vitro fertilization and embryo transfer: a comparison between a single dose of Zoladex depot and multiple daily doses of Suprefact. Hum Reprod. 1995 May;10(5):1042-1044.