

Diagnosis in Ovarian Masses! – A Big Dilemma!

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ABSTRACT : Endometriosis is a benign disease defined by the presence of endometrial glands and stroma outside uterus. Most commonly involving the ovaries (66%). We report a case of severe endometriosis which was misdiagnosed as ovarian malignancy in the light of Ca-125 being 914, LDH being 925 and USG, CT, MRI and FNAC all in favor of ovarian malignancy. Owing to her young age, infertility, history and examination our strong suspicion was towards endometriosis, which turned out right. Clinical suspicion in the face of contradictory laboratory and imaging findings is an oft encountered scenario. Hence, it may prove detrimental if clinical picture is ignored and treatment is based solely on investigations.

I. INTRODUCTION

Endometriosis is a benign disease defined by the presence of endometrial glands and stroma outside the uterus associated with dysmenorrhoea, chronic pelvic pain and infertility. Overall prevalence being 3-10%. Mean age being 25-35 years. More common in Asians>whites>blacks. Six to seven times higher in first degree relatives. More commonly occurs in the dependant part of the pelvis :-Ovaries(66% of women), broad ligament, peritoneal surface of cul-de-sac and uterosacral ligaments, rectovaginal septum, rectosigmoid colon, distant sites and laparotomy scars, etc.[1] Endometriotic cysts generally occur within the ovaries and they are the result of repeated cyclic hemorrhage within a deep implant. These cysts can completely replace the normal ovarian tissue. As the blood builds up over months and years, it turns brown and resembles chocolate hence also referred as *chocolate cyst*. [2]

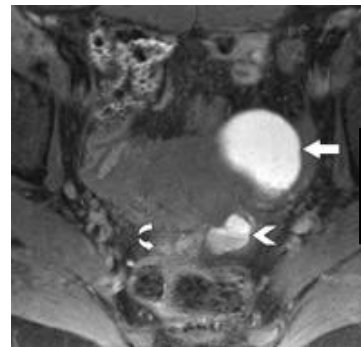
II. CASE HISTORY

A 29yr old nulligravida, married since 3yrs, known case of hypothyroidism on eltroxin 75mcg, came complaining of breathlessness, abdominal bloating, huge lump in abdomen, debilitating polymenorrhagia and severe dysmenorrhoea since 6 months along with infertility. loss of appetite was accompanied with weight loss and persistent low grade fever since one month. Hb of 3gm% coupled with rare A negative Blood Group added to the problem. She was averagely built, severely pale, pulse 100/min, hyperdynamic, BP 100/70 mm of Hg. Per abdomen, 18-20 wks fixed pelvic mass was felt, mildly tender, cystic to firm. Shifting dullness present suggested moderate ascites. Per vaginum, above mass was confirmed, arising from adnexa. All routine tests including thyroid profile were normal. 3 blood transfusions were given and Hb rose to 9gm%. ESR was 50mm at 1st hr. USG showed left ovarian complex cyst with solid component (9x6.7x8 cm), ET-5.2mm with PI 0.9 and RI 0.6 with good arterial and good diastolic flow. Moderate ascites present. CT scan showed left ovarian cystic lesion 10x10 cm with internal septum. No e/o solid component. MRI was suggestive of approximately 10x10 cm ovarian mass with no solid component, with internal echoes. Ca 125 was 914 (N- 0-35 U/ml) and LDH was 925 (N- 313-618 U/L) both alarmingly high. Radiologists suggested FNAC under USG guidance. It was done and was suggestive of ?TB, ?Malignancy, making the dilemma worse. Hence we arrived at a differential diagnosis suggestive of endometriosis; tuberculosis; ovarian malignancy. Owing to her young age, infertility, history and examination our strong suspicion was towards endometriosis. After explaining the risk of surgery to her we proceeded with Exploratory Laparotomy. Lt Oophorectomy, was done to remove a 10 x10 cm densely adherent chocolate cyst after separating adhesions. No blood was required intra operatively and no surrounding structure was damaged in this difficult surgery. Not only was our preoperative diagnosis of endometriosis correct, inspite of investigations suggesting malignancy but our conservative approach at Exploratory Laparotomy saved the patients fertility. Histopathological examination later on confirmed endometriosis. Patient is symptomatically relieved and now on Leuprolide 3.75mg (GnRH analogue) to prevent recurrence. After 3 doses she will be put on fertility enhancing therapy.



Ovarian mass

Figure 1



Ovarian malignancy

Figure 2



Chocolate cyst

Figure 3

III. DISCUSSION

The primary mechanism of pathogenesis of endometriosis being Sampson's theory of retrograde menstruation and implantation, others being coelomic metaplasia, vascular or lymphatic dissemination or direct transplantation of endometrial tissue. Altered immune function both cellular and humoral predisposes to endometriosis. It may also involve auto-immune mechanisms. Molecular mechanism includes high local estrogen and prostaglandin production and resistance to the action of progesterone, which induces a chronic inflammatory response in a feed-forward, self-perpetuating cycle [1]. Focal leaks with inflammation, fibrosis and adhesion formation are characteristics of endometriosis, whereas acute cyst rupture is a relatively uncommon complication [2]. Cases of acute cyst rupture are rare, but they may be associated with severe peritonitis and systemic disturbance, followed by adhesion formation [3,4].

A theory on the formation of ascites in endometriosis was postulated by Bernstein et al. [5], who suggested that the blood and endometrial cells shed into the peritoneal cavity may irritate and stimulate the peritoneum, thereby resulting in ascites. Others have reported that rupture of endometriotic cysts with subsequent peritoneal irritation and the production of reactive exudates may provide an explanation [6]. Ruptured endometriotic cysts sometimes present a diagnostic problem and surgical challenge because patients with a ruptured cyst present with symptoms of an acute abdomen associated with severe abdominal pain and unstable vital signs [8]. Ruptured ovarian endometriotic cysts can sometimes mimic ovarian malignancy because of the extremely elevated serum CA 125 concentration [3]. Infertility is 6 to 8 times more likely to have endometriosis than fertile women [7]. Despite extensive research, no agreement has been reached and several mechanisms have been proposed to explain the association between endometriosis and infertility. These mechanisms include distorted pelvic anatomy, endocrine and ovulatory abnormalities, altered peritoneal function, and altered hormonal and cell-mediated functions in the endometrium. Based on common observations in laparoscopy, pelvic anatomy distortion, the so-called "pelvic factor",

can more readily explain infertility in patients with severe forms of endometriosis. Major pelvic adhesions or peritubal adhesions that disturb the tubo-ovarian liaison and tube patency can impair oocyte release from the ovary, inhibit ovum pickup, or impede ovum transport [8]. Women with endometriosis may have endocrine and ovulatory disorders, including luteinized unruptured follicle syndrome, impaired folliculogenesis, luteal phase defect, and premature or multiple luteinizing hormone (LH) surges [9]. Endometriosis is associated with high chances of recurrence[1].

IV. CONCLUSION

Clinical suspicion in the face of contradictory lab and imaging findings is an oft encountered scenario. There is a tendency to ignore clinical picture and rely solely on investigations. Treatment then proceeds along wrong lines. All masses with raised CA 125 are not malignancy. Evidence may point in a certain direction. However, before embarking on any surgical procedure, these investigations should be seen in light of the whole clinical picture. This will avoid errors in diagnosis and unpleasant surprises on opening up the patient. Further, many patients are closed without completing surgery as arrangements for prolonged and complicated surgery have not been made in advance. This may expose the physician to unnecessary litigation. Again whenever, there is long standing infertility, endometriosis must top the chart of possible causes.

REFERENCES

- [1] Clinical Gynecologic Endocrinology and Infertility by Leon Speroff.
- [2] Woodward PJ, Sohaey R, Mezzetti TP Jr. Endometriosis: radiologic-pathologic correlation. *Radiographics* 2001; 21: 193- 216.
- [3] Johansson J, Santala M, Kauppila A. Explosive rise of serum CA 125 following the rupture of ovarian endometrioma. *Hum Reprod* 1998; 13: 3503-3504
- [4] Evangelinakis N, Grammatikakis I, Salamalekis G, Tziortzioti V, Samaras C, Chrelias C, et al. Prevalence of acute hemoperitoneum in patients with endometriotic ovarian cysts: a 7-year retrospective study. *Clin Exp Obstet Gynecol* 2009; 36: 254-255
- [5] Bernstein JP, Perlow V, Brenner JJ. Massive ascites due to endometriosis. *Am J Dig Dis* 1961;6:1-7
- [6] el-Newihi HM, Antaki JP, Rajan S, Reynolds TB. Large bloody ascites in association with pelvic endometriosis: case report and literature review. *Am J Gastroenterol* 1995;90:632-634 Ozkan S, Murk W, Arici A.
- [7] Verkauf BS. The incidence, symptoms, and signs of endometriosis in fertile and infertile women. *J Fla Med Assoc.* 1987; 74(9):671-5.
- [8] Schenken RS, Asch RH, Williams RF, Hodgen GD. Etiology of infertility in monkeys with endometriosis: luteinized unruptured follicles, luteal phase defects, pelvic adhesions and spontaneous abortions. *Fertil Steril.* 1984; 41:122-30.
- [9] Practice Committee of the American Society for Reproductive Medicine (ASRM). Endometriosis and Infertility. *Fertil Steril.* 2006; 14:S156-60.