Massive hemorrhagic ascites (4470 mL, range 1–10 L) in women with endometriosis is a rare condition occurring predominantly in black women. Of the 43 case reports published, 42 are compatible with the hypothesis that the hemorrhagic ascites is predominantly a consequence of excessive ovarian transudation similar to a Meigs syndrome. Indeed, bilateral ovariectomy cures the condition without recurrences, whereas after unilateral ovariectomy or cystectomy recurrence rate is more than 50%; during ovarian suppression by luteinizing hormone-releasing hormone agonist ascites disappears, but reappears after treatment. Superficial pelvic endometriosis also contributes to the ascites because after superficial endometriosis destruction the recurrence rate is only 4 in 14. Based on these data, it is suggested, to scrutinize the ovaries for tumors given the analogy with Meigs syndrome. In women desiring fertility, conservative treatment with destruction of endometriosis only can be attempted given the cure rate of some 20%. It is unknown what the effect of ovulation induction would be.
In addition, in women with severe bilateral cystic ovarian endometriosis and with extensive superficial endometriosis, the volume of peritoneal fluid is hardly increased. Occasionally, some women with endometriosis have an important hemorrhagic ascites as described in 43 case reports. The pathophysiology of this hemorrhagic ascites is unknown, but the widely held belief that the ascites is a consequence of the superficial endometriosis, similar to peritoneal metastases, remains speculative.

Two cases of massive hemorrhagic ascites together with endometriosis in women with mechanical fertility prompted us to review the literature in detail, to evaluate whether the pathophysiology could be similar to Meigs syndrome, and to decide whether ovulation induction for in vitro fertilization could reasonably be attempted.

Materials and Methods

Case Report 1

A 23-year-old nulligravida woman had severe dysmenorrhea and menstrual right shoulder pain. A hydrothorax was drained twice, confirming the diagnosis of endometriosis. During treatment with luteinizing hormone-releasing hormone agonists symptoms disappeared, but 3 months later symptoms recurred. A pleurectomy was performed with removal of many small endometriotic lesions on the pleura, a 2-cm nodule in the right diaphragm, a 4-cm nodule in the upper part, and a 3.5-cm nodule in the middle part of the right lung. Four months later, she was readmitted with severe pelvic pain and ascites. At laparoscopy, massive hemorrhagic ascites was found together with a frozen pelvis, bowel adhesions, and multiple spots of endometriosis on the peritoneum and the ovary. During treatment with luteinizing hormone-releasing hormone agonists the patient was free of symptoms but 1 year after stopping the treatment, she was readmitted with symptoms of subocclusion, ascites, and pain. A large sigmoid nodule was diagnosed on contrast enema. At laparoscopy, 1.5 L of hemorrhagic ascites was found together with severe adhesions and 2 big nodules of deep endometriosis. A low rectovaginal nodule of 5-cm diameter attached to the right spine was excised with a carbon-dioxide laser, together with ureterolysis over a double J because of hydronephrosis of the left ureter. For a sigmoid nodule of some 4-cm diameter with more than 50% occlusion of the bowel, a resection anastomosis was performed. A liver lesion was biopsied but revealed fibrosis only. Thorough inspection of the ovaries during surgery and by ultrasound failed to identify any tumor. After surgery she received 6 months of gonadotropin-releasing hormone therapy was started. With this therapy, patient is still symptom free after 3 years.

Case Report 2

A 26-year-old, Caucasian, nulliparous woman had an emergency laparoscopy and more than 1 L of hemorrhagic ascites was evacuated. One year later a second laparoscopy was performed for acute pain. Again, more than 1 L of hemorrhagic ascites was drained. Severe superficial endometriosis involving the bowel, peritoneum, and omentum was excised. Two years later an ultrasound-guided evacuation of 2 L of hemorrhagic ascites was performed for recurring pain. Two months later a third laparoscopy was performed because of severe pain, massive ascites, and increased concentrations of white blood cell count, increased concentration of C-reactive protein, and slight fever. Ascites was drained, and an adhesiolysis together with the excision of an endometriotic rectovaginal nodule was performed. Less than 1 year later the patient again had acute pain, important ascites, and signs of an inflammatory reaction. Another paracentesis was performed and 1.5 L of hemorrhagic fluid evacuated. Some 2 months later, the ascites had returned and pain was intolerable. Because at magnetic resonance imaging a 2-cm ovarian cyst was found, a laparotomy was performed. Massive adhesions were lysed, and an appendicectomy, an omentectomy, and a unilateral adnexectomy were performed. One year later symptoms and ascites had returned, and after another paracentesis to evacuate hemorrhagic fluid, gonadotropin-releasing hormone therapy was started. With this therapy, patient is still symptom free after 3 years.

Literature Review

All original case reports (n = 44) written since 1980 were reviewed in detail except 2 articles we could not retrieve. We looked specifically for pathophysiology, volume of peritoneal fluid, presence of hematotherax, CA 125 concentrations, age, parity, race, whether a tumor or mass was detected in the ovaries before or during surgery, and the outcome of ovarian suppression therapy, adnexectomy, and other therapies. For volume we recorded the original volumes reported, not the volume of recurrences.

Statistics

Statistics were performed with the SAS system (SAS Institute, Inc., Cary, NC), using Spearman correlation.

Results

The age of the women reported in the literature ranged from 20 to 50 years with a mean age of 31.9 ± 8.8 years.
Table 1

<table>
<thead>
<tr>
<th>N</th>
<th>Study</th>
<th>Year</th>
<th>Age (yrs)</th>
<th>Race</th>
<th>Parity</th>
<th>Volume (mL)</th>
<th>Color</th>
<th>CA125</th>
<th>Pleural fluid</th>
<th>Ovarian cyst</th>
<th>Surgical treatment</th>
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<td>2500</td>
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<td>Yes</td>
<td>Biopsies</td>
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<td>1800</td>
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</table>

Race distribution was 21 black, 5 Asian, 1 Hispanic, 3 white, and 13 not reported (p = .001 for black).

Ussia et al. Hemorrhagic Ascites in Endometriosis 679
An endometrioma was described in 25 women with 2 rupturing. All other ovaries were reported as normal at inspection. In none of the reports nor in our 2 cases was an ovarian tumor or mass identified by preoperative computer aided tomography scan (n = 17) magnetic resonance imaging (n = 1), ultrasound (n = 16), during surgery or by pathology after ovariectomy. Surgical treatment consisted of bilateral salpingooophorectomy (with or without hysterectomy) in 14 women followed by ovarian suppression in 2. Unilateral oophorectomy was performed in 6 followed by medical treatment in 3; cystectomy in 5 with medical treatment in all 5; and destruction of peritoneal endometriosis and adhesiolysis in 10 followed by ovarian suppression in 8. In all 14 women treated by bilateral salpingo-oophorectomy, ascites disappeared without recurrence. In all 26 patients receiving ovarian suppression, the ascites disappeared during treatment of up to 5 years. After unilateral oopherectomy, ascites redeveloped in 2 of 6. After excision of ovarian endometriosis only, the recurrence rate was 2 of 3 and after destruction of superficial endometriosis, was 4 of 14.

Discussion

Hemorrhagic ascites together with endometriosis belongs to the rare but seemingly well-known pathologies, with massive ascites, either dark brown or hemorrhagic, but without clots. The pathophysiology repetitively was suggested to be caused by rupture of an endometrioma or by exudation from widespread pelvic endometriosis. The available evidence suggests, however, that both suggestions are either erroneous or insufficient. An endometrioma was found in only 65%. Rupture of an endometrioma is a well-known pathology, with acute pain, slight fever, and less than 500 mL of fluid at laparoscopy/laparotomy [17,18]. Very extensive pelvic superficial endometriosis can be associated with a slight increase in peritoneal fluid but is not associated with massive ascites. In the case reports, ascites recurred in 4 of 14 after destruction of the endometriotic implants. This is difficult to interpret because it can be viewed as supporting the hypothesis that destruction of superficial implants is not effective. The recurrence in only 4 of 14, however, can also be viewed as supporting the hypothesis of peritoneal leakage as seen in cancer metastasis. Unfortunately, in the case reports, the extent and activity of the pelvic endometriosis was insufficiently documented to relate this to effectiveness of treatment.

The effectiveness of ovarian suppression therapy and bilateral ovariectomy is consistent with the hypothesis that the ovary is the origin of the massive ascites (i.e., similar to Meigs syndrome). Although we understand the increased vascular permeability caused by, for example, excessive estrogens during follicular proliferation and ovarian hyperstimulation syndrome, we today do not have data identifying the factors leading to the increased leakage of fluid in Meigs syndrome. We can only speculate that if this increased fluid leaking from the ovary is associated with active endometriotic lesions or an open cystic ovarian endometriosis, some blood staining will occur, resulting by accumulation of red blood cells in dark brown ascites fluid, with some red blood cells in all cases when reported. The 50% recurrence rate after unilateral ovariectomy or cystectomy also is compatible with the concept that the ovary is the source of the fluid.

The pathology clearly is acquired and not congenital. Symptoms start many years after menarche, and are unrelated to a pregnancy. No explanation exists as to why the prevalence is higher in black women than in white, as observed before, nor for the observation that the age of the women in the case reports increases over time.

In conclusion, the pathophysiology of the hemorrhagic ascites is suggested to be similar to Meigs syndrome (i.e., a local intraovarian factor). Whether this is related to the endometriosis is unknown, although the deep brown color of the ascites suggests a causal relationship. Unfortunately we do not yet have any conclusive evidence for this, as we do not know the pathophysiology of Meigs syndrome. Superficial pelvic endometriosis is suggested to be a cofactor, contributing to the ascites and to the dark brown color. Ovulation induction or in vitro fertilization was not reported yet.

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References


