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Pathophysiology of Cyclic Hemorrhagic Ascites and Endometriosis

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ABSTRACT 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. Journal of Minimally Invasive Gynecology (2008) 15, 677–681 © 2008 AAGL. All rights reserved.

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Peritoneal fluid during the normal menstrual cycle was described to be predominantly formed as an ovarian exudate. Women without ovarian activity have less than 5 mL of peritoneal fluid whereas in cyclic women the volume of peritoneal fluid increases progressively up to a few hundred milliliters during ovulation [1]. The increased vascular permeability around the developing follicle was postulated as the underlying mechanism, explaining the high steroid hormone concentrations in peritoneal fluid mediated by local factors such as the extremely high estrogen concentrations, and other factors such as angiogenic factors, prostaglandins, histamine, and cytokines. The massive ascites in women with ovarian hyperstimulation syndrome have been explained by similar mechanisms [2]. The mesothelial cells of the peritoneum actively regulate the exchange between peritoneal fluid and the bloodstream, and the transport rate is much slower for larger molecules. This explains the fact that the concentrations of blood proteins are lower in peritoneal fluid. For example, albumin concentration being some 70% of the plasma concentration whereas the concentration of larger molecules such as gammaglobulins and fibrinogen is even lower [3,4]. This also is the reason that locally secreted large molecular–weight proteins, such as CA 125 and PP14, accumulate and their concentrations can be very high in peritoneal fluid [5].

Inflammation, either locally after a mechanical trauma such as surgery or more generalized during peritonitis, also increases the volume of peritoneal fluid by a mechanism of exudation. This fluid typically contains similar protein concentrations as in blood, with high fibrinogen content and fibrin deposition.

The Meigs syndrome is well known as the association of an ovarian fibroma, massive ascites, and hydrothorax [6]. The mechanism of the increased ovarian exudation is, to our knowledge, not yet identified. After the excision of the ovarian tumor or after adnexectomy, the ascites disappears. By analogy, a malignant ovarian tumor, or a metastasis in the ovary [7] together with ascites and hydrothorax is called a pseudo-Meigs syndrome. Other rare causes of pseudo-Meigs syndrome are the struma ovarii and rare cases of uterine fibroma [7–10].

Pelvic endometriosis is known to constitute a low-grade pelvic inflammation [11–13], and the peritoneal fluid volume is only slightly higher than in women without endometriosis

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[13,14]. 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 agonists, followed by intermittent administration of corticosteroids (Fiorenzo De Cicco) for unclear reasons. With this treatment she remained symptom free. Because of persisting primary infertility, a second-look laparoscopy was performed 1 year later showing few adhesions, no residual endometriosis, and no ascites. Two years later, the patient is without medical treatment except intermittent corticosteroids. She is symptom free without ascites, dysmenorrhea, or pelvic pain and with an excellent quality of life. Because the patient wants to continue this treatment we do not know whether stopping the treatment would cause a recurrence. In vitro fertilization will be considered in the near future.

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 [15,16]. We looked specifically for pathophysiology, volume of peritoneal fluid, presence of hematothorax, 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 $\pm$ 8.8 years

Table 1
Literature review of hemorrhagic ascites in endometriosis

N	Study	Year	Age (yrs)	Race	Parity	Volume (mL)	Color	CA125	Pleural fluid	Ovarian cyst	Surgical treatment	Medical treatment
1	Chervenak et al [19]	1981	29		0	1500	DB		No	Yes	BSO	No
2	Chervenak et al [19]	1981	26	B	0	4000	DB		No	No	Partial USO	Danazol
3	Gaulier et al [20]	1983	22	B	0		DB		Yes	Yes	Cystectomy	Danazol
4	Jenks et al [21]	1984	33	B	0	5000	DB		No	No	TAH, BSO	No
5	Halme et al [22]	1985	23	B	0	7500	H		No	No	Adhesiolysis	Danazol
6	Iwasaka et al [23]	1985	35	A	0	2500	DB	17	No	Yes	TAH, BSO	No
7	Iwasaka et al [23]	1985	25	A	0	150	DB		No	Yes	USO	Danazol
8	Naraynsingh et al [24]	1985	24	B	0	6000	H		No	No	Biopsies	Depo-Provera
9	Chichareon and Wattanakitkrailert [25]	1988	31		0	1800	H		No	Yes	TAH, USO	Depo-Provera
10	Feigin et al [26]	1988	28	W	0	1600	DB		No	No	Biopsies	Danazol
11	Olubuyide et al [27]	1988	19	B	0	4600	H		Yes	Yes	Biopsies	Norethisterone
12	Taub et al [28]	1989	32	B	1	3400	H		Yes	Yes	BSO	Depo-Provera
13	Yu and Grimes [29]	1991	26	A	0	3000	H		Yes	Yes	Adhesiolysis, USO, omentectomy	GnRH agonist
14	Williams and Wagaman [30]	1991	27	B	0	7500				No	TAH, BSO	No
15	London and Parmley [31]	1993	29	B	0	6000	DB		No	No	TAH, BSO, omentectomy	No
16	Jose et al [32]	1994	30	A	0	5000	DB		No	Yes	Biopsies	Danazol
17	Schlueter and McClennan [33]	1994	20	B	0	5000	H		Yes	No	Biopsies	GnRH agonist
18	El Newihi et al [34]	1995	32	B	0	4000	DB	118	Yes	Yes	Biopsies, TAH, BSO	GnRH agonist
19	Myers et al [35]	1995	65			3000		440	Yes	No	TAH, BSO	No
20	Myers et al [35]	1995	47		0	499		33	Yes	Yes	TAH, BSO, omentectomy	No
21	Shek et al [36]	1995	21		0	999		15	Yes	No	No	GnRH agonist
22	Spitzer and Benjamin [37]	1995	25	B	0	6000	DB	237	No	Yes	Cystectomy	Norethisterone
23	Mejia et al [38]	1997	44		0	10000	H	7	No	Yes	Biopsies, TAH, BSO	No
24	Sailesy	1996	25		0	1300	H		No	No	Excision	Oral contraceptive
25	Muneyyirci et al [39]	1997	26	B		2000		15	Yes	Yes		GnRH agonist, danazol
26	Muneyyirci et al [39]	1998	31	B	0	10000	DB		Yes	Yes	Biopsies, BSO	Depo-Provera
27	Muneyyirci et al [39]	1998	32	B	0	5700	DB		No	Yes	Wedge resection	GnRH agonist
28	Muneyyirci et al [39]	1998	35	B	1	3000	H	266	No	Yes	Cystectomy	GnRH agonist, norethindrone
29	Bhojawala et al [40]	2000	34		0	9000	DB		Yes	Yes	TAH, USO	No
30	El-Khalil	1999	36			3500	H		No	Yes	Biopsies	Oral contraceptive
31	Samora-mata and Feste [41]	1999	43	Hispanic	3	2000	DB		Yes	Yes	USO, TAH, USO	No
32	Dias et al [42]	2000	41	B	0	10000	H		No	Yes	USO	GnRH agonist
33	Cheong and Lim [43]	2003	41	A	1	5600	DB	15	Yes	No	Biopsies	
34	Donnez and Jadoul [44]	2005	35		0	5000	H		No	Yes	Biopsies	GnRH agonist, tibolone
35	Fortier et al [45]	2005	33	B	0	4000		257	Yes	Yes	Cystectomy	GnRH agonist
36	Goumenou et al [46]	2006	46		0	4000	H	3504	Yes		TAH, BSO	Chemotherapy
37	Mwenechanya and Beck [47]	2007	31		0	1000		20	Yes	Yes	Biopsies	No
38	Alabi et al [48]	2007	30	B	0	5000	H	56	No	No	Adhesiolysis	No
39	Ekoukou et al [49]	2007	28	B	0	10000			No	No	Biopsies	GnRH agonist
40	Ferrero and Remorgida [50]	2007	36			4800			No	No	Excision	Norethindrone
41	Palayekar et al [51]	2007		B	1	5000		34	No	Yes	TAH, BSO	No

A = Asian; B = black; BSO = bilateral salpingo-oophorectomy; DB = dark brown; GnRH = gonadotropin-releasing hormone; H = hemorrhagic; TAH = total abdominal hysterectomy; USO = unilateral salpingo-oophorectomy; W = white.

(Table 1). Surprisingly, during this 27-year period, the age of the women published increased significantly ($p = .001$). The volume of the ascites was high at 4470 ± 2625 L. CA 125, if reported, was elevated. Color of the fluid was described as dark brown in 17 and as hemorrhagic in 15 (9 missing). Peritoneal fluid was liquid without clots in all cases.

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

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 salpingo-oophorectomy (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 oophorectomy, 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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