Differential Diagnosis of Gynecologic Organ-related Diseases in Women Presenting with Ascites

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SUMMARY

Ascites is a pathologic accumulation of fluid within the peritoneal cavity, and usually develops as a result of liver disease, congestive heart failure or nephrotic syndrome. Ascites is also a common manifestation of some gynecologic diseases. It is important that health care workers consider gynecologic problems among the potential differential diagnoses in patients presenting with ascites. Various kinds of ovarian diseases, such as epithelial ovarian cancer, benign ovarian fibroma, stromal hyperplasia, ovarian hyperstimulation syndrome, primary peritoneal serous carcinoma, endometriosis and peritoneal tuberculosis, should be kept in mind when women are found to have ascites. [*Taiwan J Obstet Gynecol* 2008;47(4):384–390]

Key Words: ascites, endometriosis, epithelial ovarian cancer, ovarian hyperstimulation syndrome, peritonitis tuberculosis

Introduction

Ascites is a term that describes a pathologic accumulation of fluid within the peritoneal cavity. It can develop as a result of liver disease, congestive heart failure or nephrotic syndrome, and is not an uncommon complication accompanying some gynecologic diseases. It is, therefore, important that health care workers consider gynecologic problems among the potential differential diagnoses in patients with ascites. The following review explores the gynecologic diseases that can result in ascites.

Ovarian Malignancy

Epithelial ovarian cancer is possibly the most common type of cancer leading to malignant ascites [1–4]. In a 2-year, single institute, retrospective analysis, 37.7% of patients with ovarian cancer developed ascites, accounting for a quarter of the cases of malignant ascites [1]. Paracentesis is commonly used to evaluate ascites and to make a differential diagnosis. Around 70% of patients with ovarian cancer demonstrate cytologic evidence of ascites [5]. Nevertheless, in some cases where an ovarian malignancy is suspected, paracentesis may be withheld because of the risk of abdominal wall metastases developing at the paracentesis entry sites, especially since the occurrence of this phenomenon, together with the presence of ascites, is highly associated with advanced tumor stage [6,7]. The reported incidence of tumor implantations at trocar or puncture sites after invasive diagnostic procedures varies widely, ranging from 1.2–13.7% [6–9]. The mechanism behind puncture site metastasis is not well understood, but it is likely to be related to direct implantation following surgical trauma [10,11]. The concept of standard therapy for advanced ovarian cancer using primary debulking surgery followed by chemotherapy has recently been challenged by an alternative option using neo-adjuvant chemotherapy followed by interval debulking surgery [12–14]. This would increase the possibility for performing diagnostic paracentesis before getting a definite diagnosis. Although the presence of

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abdominal wall metastasis does not seem to be significantly correlated with survival [7], the potential danger of performing diagnostic paracentesis should always be kept in mind.

The presence of malignant ascites is usually indicative of a poor prognosis; however, malignant ascites of ovarian origin has a relatively higher survival rate than that of other origins [1–4]. The management of malignant ascites caused by operable ovarian cancer differs from that of other cancers. The mainstay of therapy for ascites accompanying ovarian cancer, unlike other cancers, is attempted removal of all detectable tumors by debulking surgery, followed by adjuvant chemotherapy. The ascites will subside if the ovarian cancer is responsive to chemotherapy. However, for those with malignant ascites caused by recurrent or refractory ovarian cancer, palliative treatment by serial paracentesis or drainage is a possible option to provide symptomatic relief [15–17]. Infectious morbidity is a major concern associated with invasive procedures. In addition, the removal of large volumes of ascitic fluid may result in protein loss and hypovolemia, with subsequent circulatory problems. Although diuretics and salt restriction have been empirically used in the treatment of ascites caused by different kinds of medical illnesses, the effectiveness of these approaches for malignant ascites has yet to be proven. Treatment of the causative malignant cells by intraperitoneal cytotoxic chemotherapy provides another possible approach for controlling malignant ascites. Various antineoplastic agents, including cisplatin, carboplatin, 5-fluorouracil and leucovorin, as well as metalloproteinase inhibitors, have been used to inhibit ascites formation. Intraperitoneal agents that block vascular endothelial growth factor have also been found to be a promising therapy for ascites related to carcinoma in both animal models and human clinical trials [18,19]. Some experimental strategies, such as the use of radiolabeled antibodies and immunotherapy, have been reported to be alternative options for ovarian cancer patients with malignant ascites [20].

Pelvic or Peritoneal Tuberculosis

Tuberculosis is a mycobacterial infection that most commonly manifests as pulmonary disease, but may also affect extrapulmonary sites [21–23], such as the abdominopelvic region. It usually affects women of reproductive age [24]. The clinical presentation of pelvic tuberculosis is non-characteristic, and includes vague pelvic pain, abdominal distension, weight loss or fever. Infertility is a relatively frequent complaint in female patients with pelvic tuberculosis [25]. On diagnostic imaging, pelvic tuberculosis may appear as ascites, peritoneal nodules, omental or peritoneal thickening, or adnexal masses. The clinical manifestations and imaging features can mimic disseminated carcinomatosis [26–36]. However, there are some subtle ultrasonographic differences that can help to distinguish between pelvic tuberculosis and ovarian cancer: (1) tuberculous ovarian masses are usually almost unenlarged; (2) the septum inside the tuberculous ovarian mass is usually composed of multiple, delicate, incomplete septa, rather than of thick bands as found in ovarian malignancy; and (3) the blood vessels supplying the tuberculous implants are present in high impedance, which reflects vascular resistance, calculated after measuring peak systolic (S) and peak diastolic (D) velocities of blood flow by colored Doppler ultrasound using the formula: resistance index = (S – D)/S [37].

Ascites is a common presentation of pelvic tuberculosis. However, the diagnostic value of paracentesis in ascitic fluid analysis is still inconclusive. The sensitivity of direct preparations of ascitic fluid, for either acid-fast stain or tuberculosis culture, is relatively low [38]. Moreover, the long turnaround time required for culture of the microorganism makes this method impractical. Currently, ascitic fluid analysis by polymerase chain reaction for mycobacteria or for elevated adenine deaminase levels is considered to be a reliable diagnostic technique [39,40]. Blind percutaneous peritoneal biopsy was first advocated 40 years ago as the procedure of choice in cases of early tuberculous peritonitis [41], with varying reported rate of success [42]. Peritoneal biopsy was performed at the left lower quadrant of the abdomen, adjacent to the rectus muscle, using an Abrams needle or Cope needle, under local anesthesia. The limitations include complications such as bowel perforation and even mortality, and the technique is contraindicated in the absence of ascites. However, these limitations could be overcome by using ultrasound- or computed tomography-guided needle biopsy, which is deserving of further study [43]. Even though pelvic tuberculosis is usually secondary to previous tuberculosis infection, the tuberculin skin test and chest roentgenograms do not help to distinguish patients with pelvic tuberculosis [44,45]. Most patients in the literature were diagnosed by exploratory laparotomy and by confirmatory histopathology.

With significant improvements in laparoscopic techniques, laparoscopy has become an effective diagnostic tool to confirm uncertain abdominal conditions [46], and provides an alternative to traditional laparotomy [47] and a better choice than exploratory laparotomy [48]. The characteristic laparoscopic appearance includes free ascites with multiple yellowish-white

nodules of tubercles on the visceral and parietal peritoneum, peritoneal or visceral adhesion, and occasionally inflamed hemorrhagic areas on the peritoneum [42]. The results of a large case series that investigated laparoscopic evaluation (combined with peritoneal biopsy) of ascites of unknown origin found that a definite diagnosis was established in 80–97% of cases [42, 49–51]. Patients diagnosed with pelvic tuberculosis had a satisfactory response to anti-tuberculosis drugs and ascites generally resolved after anti-tuberculosis treatment [29,32].

Ovarian Hyperstimulation Syndrome (OHSS)

Ascites is a clinical manifestation of OHSS. OHSS is an iatrogenic complication of ovarian stimulation for assisted reproductive techniques [52,53]. The pathogenesis of OHSS may be associated with an exaggerated response to gonadotropin, resulting in increased vascular permeability via the activity of vascular endothelial growth factor, tumor necrosis factor, and interleukin. The syndrome is characterized by cystic enlargement of the ovaries and fluid leakage from the blood vessels into the interstitial space [52]. The clinical presentation of OHSS varies considerably, ranging from mild distension, discomfort and nausea, to dyspnea, oliguria, increased hematocrit, thromboembolism, or even abnormal creatinine clearance, liver dysfunction or respiratory distress. The diagnosis is usually established by a history of assisted reproductive technique, clinical appearance, and specific ultrasound findings. Examination of the abdomen should be deferred to prevent rupture of enlarged ovaries and subsequent hemorrhage. The most recent classification of OHSS was introduced in 1999 [54] and differs from previous systems in that a mild degree of OHSS is omitted from the classification, while severe OHSS is classified into three grades, depending on general clinical symptoms and laboratory parameters. The most effective treatment for OHSS is prevention [55]. For OHSS-associated ascites, ultrasound-guided paracentesis has been shown to result in the relief of abdominal distension, improvement of dyspnea, increased diuresis, and decreased hematocrit [56–59]. Concurrent intravenous rehydration and colloid solution supplement are needed to maintain euvolemia [60]. Only a few pharmacologic interventions are available to ameliorate extravascular leakage; diuretics play no role [61], but dopamine treatment and dopamine prodrugs have been used to reduce ascites [62,63]. A prospective, randomized, double-blind study in oocyte donors at risk of developing OHSS found that administration of cabergoline, a potent dopamine agonist, significantly reduced the occurrence of ascites [64].

Meigs Syndrome, Pseudo-Meigs Syndrome, and Pseudo-pseudo Meigs Syndrome

Meigs syndrome is defined as a triad of benign ovarian tumors accompanied by ascites and pleural effusion [65]. The ovarian tumor may be a fibroma, thecoma or granulose tumor, of which ovarian fibromas are the most common cause of Meigs syndrome. Ovarian fibromas constitute less than 5% of all ovarian tumors, and only 1% of diagnosed cases have been associated with the clinical picture of Meigs syndrome [66,67]. Meigs syndrome is a rare clinical entity. Pseudo-Meigs syndrome is a variant with an identical clinical constellation, but without the original benign tumor cell types described by Meigs [68]. The benign tumors include those of the fallopian tubes and uterus, mature teratomas, struma ovarii, and ovarian leiomyomas. The distinction between Meigs syndrome and pseudo-Meigs syndrome is mostly academic, because the therapeutic strategy is the same in both cases [69]. The pathophysiology of ascites in Meigs syndrome or pseudo-Meigs syndrome remains enigmatic. Some have speculated that the accumulation of peritoneal fluid is caused by irritation of the peritoneal surfaces or by direct pressure on the surrounding lymphatics or vessels by the solid ovarian tumor [70–72]. Other proposed mechanisms suggest that the ascites is caused by fluid secretion from the ovarian tumor, or as a result of increased capillary permeability via release of mediators from the tumor [65]. The ascites usually disappears after resection of the tumor, and the prognosis is favorable.

In contrast to Meigs syndrome or pseudo-Meigs syndrome, pseudo-pseudo Meigs syndrome is a term that describes patients with ascites, pleural effusion, and enlarged ovaries secondary to systemic lupus erythematosus [73]. The ovaries in pseudo-pseudo Meigs syndrome may be enlarged, but without evidence of a tumor. Ascites in pseudo-pseudo Meigs syndrome is usually exudative, and may be associated with activated mesothelial cells in systemic lupus erythematosus [73,74].

Endometriosis

Endometriosis is a relatively common gynecologic disease, defined as the ectopic presence of endometrial epithelium and stroma outside the uterine cavity. Common clinical manifestations of endometriosis

include pelvic pain, pelvic tenderness, dysmenorrhea, dyspareunia, and a history of infertility. The fact that endometriosis can rarely be associated with ascites is not well known, even amongst practicing gynecologists. Fewer than 50 such cases have been reported since the first case was described in 1954 [75–91]. The vast majority of patients were nulliparous non-Caucasian women of reproductive age [76]. The ascites is mostly massive and may be concurrent with pleural effusion. Because endometriosis-associated ascites usually presents with a pelvic mass, poor appetite, weight loss and elevated tumor markers for ovarian neoplasms, it is commonly mistaken for ascites caused by ovarian neoplasms. Although cytologic examination via paracentesis has been used successfully to detect endometriosis in effusion [92], most patients were diagnosed by operative assessment and histologic confirmation. The pathogenesis of ascites associated with endometriosis is speculative. Ascites can form as a result of the rupture of endometriomas [89,93] or, rarely, from hepatic involvement by cystic endometriosis [86]. The hypothesis that endometrial cells shedding into the peritoneal cavity may irritate the peritoneum, thereby leading to ascites, is also widely accepted [94]. The definitive therapy for endometriosis-related ascites is, therefore, to eliminate ovarian function by oophorectomy. Medical treatment using either a progestational agent or gonadotropin-releasing hormone (GnRH) agonist is less reliable than surgical castration, and may lead to a failed response and a recurrence of ascites. However, GnRH agonists have been used successfully to manage various kinds of endometriosis and its related diseases [95–100].

Conclusion

This review indicates the need for clinicians to examine possible gynecologic sources in female patients presenting with ascites (Table). The initial evaluation of a female patient with ascites should rely on a detailed history and physical examination. Paracentesis provides a rapid and simple means of analyzing ascitic fluid, but its effectiveness in determining the underlying cause is limited in patients with ascites resulting from gynecologic disorders. The treatment of ascites depends on the cause of fluid retention. Most cases of gynecologic disease-associated ascites can be resolved following an accurate diagnosis and appropriate therapy.

Table. Characteristics of ascites resulting from various kinds of gynecologic diseases

Disease	Characteristics of ascites
Epithelial ovarian cancer	Exudate
Meigs syndrome	Most transudate, but few hemorrhagic exudate
Tuberculosis	Exudate
Ovarian hyperstimulation syndrome	Exudate
Endometriosis	Exudate

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