EJGO European Journal of Gynaecological Oncology

Case Reports

Gross painless transudative ascites in a patient with ovarian cancer

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Summary

Transudative ascites are a rare entity in cancer which may sometimes make their diagnosis difficult. Here, the authors report an unusual case of transudative ascites in a 50-year-old woman with ovarian cancer. The patient first presented with progressive painless gross transudative ascites for the past five months with no associated nephrotic syndrome or liver cirrhosis, and chylous ascites developed on day 14 of the admission. The ascites were transudate with serum-ascites albumin gradient (SAAG) above 11 g/L. Repeated screening of cancer cells from ascites revealed adenocarcinoma originated from ovary.

Key words: Ascites; Transudate; Chylous; Ovarian cancer.

Introduction

Gross ascites are a frequent presentation of cancer; however, ascites are always described as an exudative with a serum-ascites albumin gradient (SAAG) below 11 g/L, unless associated with nephrotic syndrome [1].

The authors report an unusual case of transudative ascites in a 50-year-old woman with cancer. Though multiple abnormal foci were present within abdominal and pelvic cavity, the ascites were a transudate with SAAG above 11 g/L with no associated nephrotic syndrome or liver cirrhosis.

Case Report

In November 2014, a 50-year-old female was referred to the present hospital with progressive abdominal distention and shortness of breath for the past five months. She had been diagnosed with gross ascites with no clear reason identified at the local hospital, and then sent to the present hospital for further evaluation. She had no history of hepatic cirrhosis, kidney disease, heart diseases, trauma, surgery nor did she take any medication.

On admission, the blood pressure was 140/80 mmHg, and her temperature was 36.3°C. She had no apparent symptoms suggestive of portal hypertension, urinary infection, heart disease or abdominal infection. Physical examination showed malnutrition with weight loss from 60 kg to 45 kg during the last five months. Cardiovascular and respiratory examinations were unremarkable. On abdominal examination, there was gross abdominal distention with positive shifting dullness and no tenderness. No leg edema, skin rash, or arthralgia were observed. Laboratory findings were as follows (with normal ranges shown in parentheses): white blood cell (WBC) count 4.49×10^9/L, red blood cell (RBC) count 3.98×10^{12}/L, hemoglobin 119 g/L, hematocrit 35.6%, platelet count 252×10^9/L, erythrocyte sedimentation rate (ESR) 10 mm/h, C-reactive protein (CRP) 5.8 mg/L (0–8 mg/L), and blood lactic dehydrogenase (LDH) 162 IU/L (109–245 IU/L). Liver function tests showed total protein 56.6 g/L, albumin 32.7 g/L(34–48 g/L), aspartate aminotransferase of 16 U/L(8-40 U/L), alanine aminotransferase of 9 U/L (5–35 U/L), and γ-glutamyl transferase of 12 U/L (7–32 U/L). Kidney function tests showed blood urea nitrogen (BUN) 5.6 mmol/L (2.9–8.2 mmol/L), creatinine (Cr) 45.9 μmol/L (53–97 μmol/L). The blood glucose was 5.31 mmol/L (3.9–6.1 mmol/L), and her cholesterol was 5.4 mmol/L (0–5.69 mmol/L), which were within normal range. Thyroid function tests and coagulation studies were normal. Tumor markers such as carcinoembryonic antigen (CEA), carbohydrate antigen (CA) 19-9, and alpha fetoprotein (AFP) were within the normal range except for elevated CA12-5 of 327.8 U/ml (0–35 U/ml).

Laboratory tests for autoimmune diseases were also performed. The serum immunoglobulins showed a normal IgG (10.70 g/L, normal range 7.51–15.6 g/L), a normal IgM (1.10 g/L, normal range 0.4–2.74 g/L), and a normal IgA (1.26 g/L, normal range 0.82–4.53 g/L). Further serological studies were negative for anti-Sm, anti-ds-DNA, anti-nuclear antibody, and anti-histones. Rheumatoid factor level was within the normal range (< 20I U/ml). The serum complement levels of C3 and C4 were normal.

Initial peritoneocentesis revealed clear ascites with leucocytes 213×10^6/L and with negative microbiologic studies including bacteria, fungus, and Mycobacterium tuberculosis. Ascitic fluid cytology included 90% lymphocytes without malignant cells. The fluid biochemistry showed reduced albumin (7 g/L) and reduced protein (16 g/L) with SAAG of 25 g/L (> 11 g/L), glucose of 6.3 mmol/L, and LDH of 89 U/L (normal range 109–245 IU/L). All these laboratory findings indicated transudative ascites.

The serum ascites albumin gradient suggested portal hypertension, but she did not have signs of chronic liver disease after physical examination and laboratory and radiological studies.

Revised manuscript accepted for publication October 8, 2015

Abdominal ultrasound showed patent hepatic veins with no evidence of portal hypertension or cirrhosis. Upper endoscopy did not reveal esophageal varices or signs of portal hypertension. Cardiac ultrasound examination was normal.

Chest computed tomography (CT) showed no pleural effusion. The MRI of the pelvic with contrast revealed multiple abnormal foci within abdominal and pelvic cavity, including peritoneal, colon, bladder, bilateral and adnexa.

Repeat peritoneocentesis was again compatible with a transudative effusion without any malignant cells. On the day 14 from admission, the ascites became a milky white transudate with leukocyte count of 101×10^6/L (90% lymphocytes). Measurement of triglycerides was performed and suggested chylous ascites. Cytologic examination of 1,000 ml of chylous fluid revealed adenocarcinoma cells originated from ovary with Ki67 of 50%. The patient refused to accept any further examinations and medical treatment and then returned home.

Discussion

Gross ascites are a frequent presentation of many diseases. The mechanisms involved could be an increased filtration rate to the peritoneal cavity or an impaired drainage of the peritoneal cavity [2].

The classification of ascites as 'exudative' and 'transudative' is always the first step to diagnosis. Parameters for classification include appearance, transparency, proportion, solidification, ascitic fluid total protein, LDH, cell count, cell classification, bacteriological examination, and cytology. Common causes of transudative ascites include congestive heart failure, liver cirrhosis, nephrotic syndrome, and hypoproteinemia, while the common causes of exudative ascites include infections (such as tuberculosis and bacterial infection), tumor (such as lymphoma, lung cancer, mesothelioma), rheumatoid arthritis, systemic lupus erythematosus (SLE), and some trauma. It has now been accepted that the SAAG is a better distinguishing marker than ascitic fluid total protein (AFTP) in differential diagnosis of ascites [3, 4].

In the present case, ascites was with low protein concentration and high SAAG above 11 g/L, indicating transudative ascites. However, the usual causes of transudative ascites, including liver cirrhosis, nephrotic syndrome, cardiac failure, constrictive pericarditis, and Budd-Chiari syndrome, were all excluded in the patient. It was unlikely that the mild hypoalbuminemia alone would have explained the massive ascites.

The present authors reviewed the literature through PubMed using search terms “ascitic fluid” [mesh] OR “ascites” AND transudat*. Some unusual reasons of transudative ascites had been identified, including SLE [5, 6], ovarian hyperstimulation syndrome [7], liver hydatid disease [8], Azelnidipine-induced chyloperitoneum [9], sarcoidosis [10], hypothyroidism [11], and Schistosomiasis [12]. However, all the aforementioned conditions were also excluded. Though MRI of the pelvic revealed multiple abnormal foci within abdominal and pelvic cavity, repeated peritoneocentesis did not reveal malignant cells until the transudative effusion became chylous. How the transudative effusion becomes chylous will need to be further discussed and with some specific investigation, such as lymphangiography, which was not performed in the present patient. However, the authors could give some possible explanations according to known mechanisms.

The mechanisms of chylous ascites refer to disruption of the lymphatic system, due to either traumatic or obstructive causes [13]. In the present patient, no traumatic history existed, so the authors assume that cancer cells obstructed and destroyed the abdominal lymph flow, thus producing a transudation through the lymphatic walls and then causing abdominal distention. With progression of the disease, lymph vessels were destroyed, and chylous ascites developed. From the case report, the authors could draw the conclusion that a cancer patient may present first and mainly with transudative ascites because of the obstruction of the lymph flow even when multiple focis exist within abdominal and pelvic cavity.

In summary, the current report presents a patient with an unusual cause of transudative ascites. In the context that transudative ascites in cancer is uncommon, its recognition could probably prompt clinicians to consider this specific etiology to make a quicker and more accurate diagnosis of ascites.

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