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Case Report

Massive ascites of unknown origin

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Abstract: Massive ascites of unknown origin is an uncommon condition, which represent a diagnostic challenge. Patients with delayed diagnosis and treatment may have a poor prognosis. A 22-year-old female was referred to this hospital due to a 4-year progressive abdominal distension with massive ascites of unknown origin. By thorough investigations, she was eventually diagnosed as chronic calcified constrictive pericarditis. She received pericardiectomy and had an uneventful postoperative course. With a few day paracentesis, ascites did not progress any more. She was doing well at 5-month follow-up and has returned to work. Extracardiac manifestations, such as massive ascites and liver cirrhosis, were rare in patients with constrictive pericarditis. Pericardiectomy can be a radical solution for the treatment of chronic constrictive pericarditis. In order to avoid delayed diagnosis and treatment, physicians have to bear in mind this rare manifestation of chronic calcified constrictive pericarditis.

Keywords: Ectopia cordis, paracentesis, pericardiectomy

Introduction

Ascites is a common clinical problem, which can be a result of liver cirrhosis, neoplasm, tuberculous peritonitis, pyogenic peritonitis, congestive heart failure, nephrosis and pancreatic disorders [1]. Malignancy accounted for 10% of all the ascites [2]. Of the malignant ascites, epithelial malignancies, in particular ovarian, endometrial, breast, colon, gastric, and pancreatic carcinomas, accounted for over 80%, while malignancies of unknown origin represented 20% [2]. Massive ascites of unknown origin is an uncommon condition with protean etiologies. In female patients, the most common causes of ascites of unknown origin were malignancies (40.3%), cirrhosis (16.7%), and tuberculous peritonitis (12.9%) [3]. Almost half of the malignant ascites of unknown origin were derived from the digestive or gynecologic systems [3]. Rarely is ascites of unknown origin a manifestation of constrictive pericarditis, which is often misdiagnosed at the first presentation of the patients, while with true reasons being overlooked for a long time. Patients with delayed diagnosis and treatment often have a poor prognosis. The differential diagnosis of ascites of unknown origin therefore always remains a diagnostic dilemma.

Tuberculosis used to be a common cause of constrictive pericarditis. However, the clinical spectrum of constrictive pericarditis has changed considerably with altered infectious origin. As tuberculosis has become less frequent, more common causes are previous heart operation, pericarditis, and radiation treatment [4]. Other causes may include infection (viral or tuberculous), connective-tissue disease, uremia, neoplasm, or idiopathic condition [5]. Patients with constrictive pericarditis frequently present with circulatory symptoms including dyspnea, orthopnea, or even heart failure [5], but rarely manifest extracardiac manifestations, such as massive ascites and liver cirrhosis [6]. The fibrotic or calcified pericardium is a common finding in such patients [7]. When the patient with constrictive pericarditis presents with extracardiac manifestations, they are prone to be misdiagnosed congestive heart failure, hepatic cirrhosis, or tuberculous peritonitis. This article is to describe a case of calcified constrictive pericarditis presenting with massive ascites of unknown origin misdiagnosed as hepatic cirrhosis.

Case report

A 22-year-old female was referred to the Department of Gastroenterology for a 4-year

Table 1. Blood tests

Parameter	Value	Normal range
Hemoglobin (g/L)	115	113-151
Platelet (× 10^9/L)	195	110-360
Prothrombin time (s)	13.30	9-13
Total protein (g/L)	76.6	60-80
Albumin (g/L)	38.7	35-55
D-dimer (ng/L)	0.31	<0.5
Glucose (mmol/L)	3.54	3.89-6.11
C-reaction protein (mg/L)	4.67	0-8
Erythrocyte sedimentation rate (mm/h)	28	0-20
Anti-tuberculosis antibody	negative	negative
Natriuretic peptide (pg/mL)	197	0-200
Troponin I (μg/L)	0.00	0-0.06
α-fetoprotein (ng/L)	2.5	0-9
Carcinoembryonic antigen (ng/L)	2.27	0-5
CA12-5 (ng/mL)	27.21	0-5
CA15-3 (ng/mL)	3.72	0-25
CA19-9 (ng/mL)	32.96	0-27

Table 2. Ascites tests

Parameter	Value
Color	Yellow
Appearance	Turbid
Clot	Yes
Gravitide	1.020
Total protein (g/L)	47.3
Albumin (g/L)	23.9
Glucose (mmol/L)	4.85
Rivalta test	Positive
Adenosine deaminase (U/L)	10
Lactate dehydrogenase (U/L)	72
White blood cell count (× 10^6)	82
White blood cell morphology	More mononuclear cells than multinucleated cells cells
Red blood cell count (× 10^9)	4
Red blood cell morphology	Cell shrinkage
α-fetoprotein (ng/L)	2.1
Carcinoembryonic antigen (ng/L)	0.42
CA12-5 (ng/mL)	452.6
CA15-3 (ng/mL)	1.41
CA19-9 (ng/mL)	7.48
CA72-4 (ng/mL)	0.9

history of progressive abdominal distension. She had a medical history of pulmonary tuberculosis as being diagnosed in a provincial hospital in the previous year. She had been diagnosed as hepatic cirrhosis and received supportive treatments. She did not show considerable improvement, but contrarily progressed into a large umbilical hernia. On admission, physical examination revealed normal vital signs. There were no heart murmurs, muffled heart sounds, jugular venous distention, a paradoxical pulse or pedal edema. The navel was enlarged, with herniary contents extruding out while seating or standing, and the contents were reversible while lying down. She received a paracentesis with peritoneal drainage with more than 5,000 mL yellowish ascites drained within the first few days after admission. Blood tests showed an elevated erythrocyte sedimentation rate, slightly increased CA125 and CA199, and negative antibodies (antinuclear, anti-double strand DNA, Jo-1, Ro-52, Sc1-70, SS-A, SS-B, Sm, centromere protein B, ribosomal P-protein, nucleosome, nuclear ribonucleoprotein, and histone antibodies). The ascitic tests revealed a high ascetic total protein and a high serum ascites albumin gradient (SAAG) (**Tables 1** & **2**). Electrocardiogram displayed sinus rhythm with low voltage of the QRS waves in all leads. Echocardiography did not reveal transposition of the great vessels with normal atrioventricular junctions. Chest computed tomography illustrated outline of severely calcified pericardium and rightward-posteriorly ectopia cordis (**Figure 1**). Remarkably calcified pericardium could clearly be observed on the axial view (**Figure 2**). Abdominal computed tomography revealed massive ascites, severe abdominal wall edema and an umbilical hernia (**Figure 3**). Abdominal and gynecological investigations excluded the malignant possibilities. She was diagnosed as calcified constrictive pericarditis and was transferred to Department of Cardiothoracic Surgery for surgical treatment.

Rightward-posteriorly ectopia cordis was noted during the operation. The heart was intensely wrapped by the constrictive pericardium, with severe calcification covering the right ventricle and right atrium, and encircling the right atrioventricular groove (**Figure 4**). The diaphragmat-

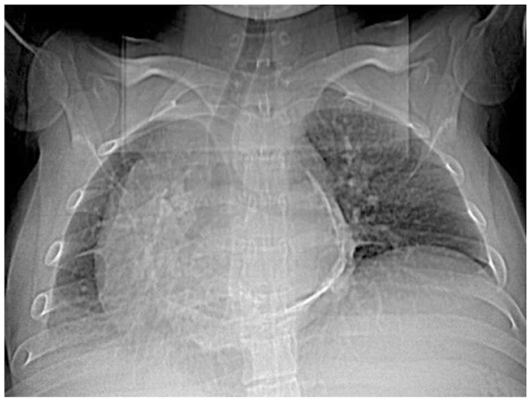

Figure 1. The first frame of chest computed tomography showing the outline of calcified constrictive pericarditis and rightward-posteriorly ectopia cordis.

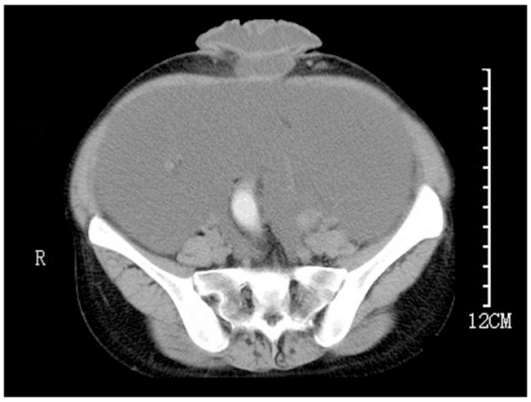

Figure 3. An axial view of abdominal computed tomography showing massive ascites, severe abdominal wall edema and an umbilical hernia.

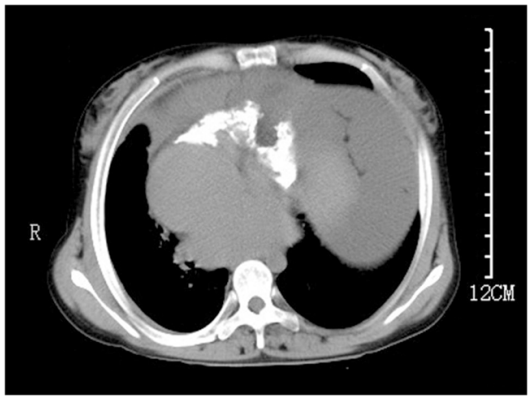

Figure 2. An axial view of chest computed tomography showing the severely calcified pericardium over the right heart.

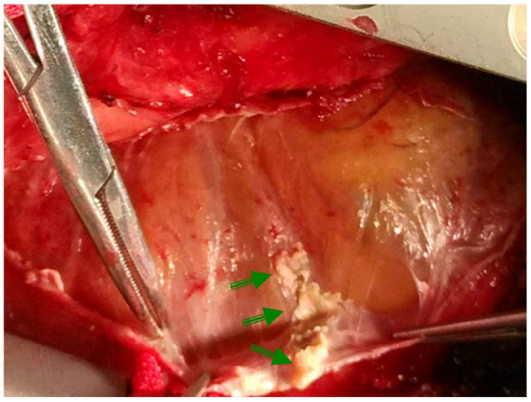

Figure 4. Pericardiectomy was performed for severe calcification covering the right ventricle, right atrium and encircling the right atrioventricular groove (arrows).

ic pericardium was extensively calcified and severely constrained the inferior vena cava. Pericardiectomy was performed by peeling off the fibrotic pericardium from the left ventricle, and the calcified pericardium from the right ventricle and right atrioventricular groove. The calcified diaphragmatic pericardium and the calcified pericardial and fibrous tissues encircling the inferior vena cava were completely released. The calcified pericardium was also released from the left atrium to the level of the right pulmonary veins. There was no constrains to the superior vena cava.

The postoperative course was uneventful. Ascites did not increase after a few days of abdominal drainage. She had an uneventful recovery and was discharged home. The umbilical hernia operation was planned at a later stage. At 5-month follow-up, she was doing well and an abdominal ultrasound showed mild ascites.

Discussion

The term “ascites of unknown origin” was firstly expressed in the literature by Ward [8] in 1982. However, the concept of ascites of unknown origin has not been properly defined up to date. Han et al. [1] defined ascites of unknown etiology as the etiology of ascites that cannot be determined after conventional laboratory examinations (including cell count, albumin level, total protein level, Gram stain, culture and cytology) and further imaging investigations (including ultrasound and computed tomography scan). However, some more details have to be updated for a complete definition, which are, ascites persistent for at least more

than 4 weeks [9]; no significant improvement with multiple therapeutic paracenteses, escalating doses of diuretics [10], and anti-inflammatory drugs [11]; and no causative agent could be identified after extensive laboratory investigations [12]. According to the course of disease, ascites of unknown origin can be divided into acute (new-onset), subacute, and chronic, with a duration of onset time of <10 days, 10 days-3 months, and >3 months, respectively. According to the eventual discrimination of the etiologies, it can be divided into true ascites of unknown origin (with eventual recognition of the etiologies) and false ascites of unknown origin (with eventually unrecognized etiologies). For the interpretation of true ascites of unknown origin, a peritoneal reaction to repetitive inflammation, which is a process that may develop into ascites in extreme cases [13].

Distributions of etiologies of ascites of unknown origin have been presented in larger patient settings. Chu et al. [14] made a diagnostic evaluation of ascites of unknown origin in 129 patients, and discovered the etiologies were carcinomatosis peritonei (60.5%), tuberculous peritonitis (20.2%), cirrhosis 5.4%, and no gross abnormality (14.0%). Han et al. [1] described the etiologies of 176 patients who underwent laparoscopy for ascites of unknown origin during a 20-year span, with carcinomatosis peritonei in 56.2%, tuberculous peritonitis in 17.6%, cirrhosis in 10.8%, and miscellaneous 15.4%, with similar distributions comparing the first with the second 10-year patients. Laparoscopy with biopsy could clarify the causes of unexplained ascites in the majority of cases, but failed to reveal any gross abnormality in only 15%. Gotthardt et al. [9] presented the underlying etiologies of persistent ascites after liver transplantation were bacterial or fungal peritonitis (69.7%), renal dysfunction (6%), obstruction of the portal vein (3%), obstruction of the liver vein outflow (12%), and unclear etiology (9%).

Ascites of unknown origin may be derived from rare diseases, which include idiopathic colonic phlebitis [15], toxocara canis infection [16], chronic granulomatous disease (CGD), a rare inherited immunodeficiency syndrome that results from abnormal nicotinamide adenine dinucleotide phosphate (NADPH) oxidase function [17], POEMS syndrome [18], familial Mediterranean fever [13], benign cystic mesothelioma (BCM) [19], celiac disease [20], ruptured remnant of urachal diverticulum [21], peritoneal sarcomatosis from desmoplastic small round cell tumor (DSRCT) [22], unknown origin irrespective of extensive investigations [23, 24].

Several physiological changes and benign lesions may mimic ascites and cause misdiagnoses. These conditions may involve bladder distention or diverticulum, hydronephrosis, non-pancreatic pseudocyst, large uterine or ovarian tumors, and giant mesenteric, omental and echinococcal cysts can mimic ascites [25], and giant intra-abdominal cysts and pseudocysts [26]. Therefore, distinguishing disgnoses with these conditions are indispensible.

Cytologically malignant peritoneal fluid can be a reliable way for clinical evaluation of the origin of malignant ascites [27]. Clinical observations revealed that ascetic lactate dehydrogenase, ascitic CA12-5, ascitic CA19-9, serum CA12-5, and serum CA19-9 of the patients with malignant ascites were 3, 4, 4, 10, and 100 times the levels of the benign, respectively. The sensitivity of serum CA12-5 was significantly higher than that of ascitic fluid CA12-5, while the sensitivities of ascitic fluid CA19-9 and carcinoembryonic antigen were significantly higher than that of serum CA19-9 and carcinoembryonic antigen. The detection rates of ultrasound scan and computed tomography scan were 94.4% and 95.6% respectively, but without specificity. The positive rate of ascitic fluid cytology was 39.6% [3]. However, cytomorphological examination alone may provide only limited sensitivity for the detection of metastatic malignant cells [28]. Cytology has good specificity but low sensitivity for the diagnosis of malignant ascites [29]. A high SAAG (>1.1 g/dL) indicates a portal hypertensive ascites; whereas a low SAAG (<1.1 g/dL) indicates a non-portal hypertensive nature [2]. In a group of patients with ascites of unknown origin, the SAAG was <1.1 in 73.17% patients, indicating a non-portal hypertensive nature of ascites in majority of the patients [29]. Jiang et al. [30] described with updated SAAG values of patients of ascites of differential etiologies, 2.12 ± 0.44 g/dL in cirrhotic ascites, 0.85 ± 0.37 g/dL in malignant ascites, and 5.48 ± 2.64 g/dL in tuberculous ascites. The median value of SAAG was 0.7 (0.5-1.2) g/dL in patients with ascites of unknown origin, 0.80 (0.6-1.8) g/dL in patients

with tuberculous ascites and was 1.5 (0.77-1.7) g/dL in patients with cirrhotic ascites [29]. An SAAG >1.1 g/dL can also be seen in cardiogenic ascites including constrictive pericarditis, right-sided heart failure, and severe tricuspid regurgitation, etc. [31]. The ascites examination of the present patient with an SAAG of 1.48 g/dL supported the diagnosis of constrictive pericarditis. 2-deoxy-2-[fluorine-18] fluoro-D-glucose integrated with computed tomography (18F-FDG PET/CT) assisted in detecting the original cause of ascites. The differential diagnostic ability of 18F-FDG PET/CT was superior to that of computed tomography alone, tumor markers, and cytology. The maximal standardized uptake values in patients with malignant primary and metastatic lesions was significantly higher than that in healthy volunteers and in patients with benign ascites [32]. Diagnosis of ascites of unknown origin due to POEMS syndrome should be made by bone marrow biopsy [18].

Pathological examination of the omental biopsies can be helpful in determining the origin of the lesions [33]. By providing clear visualization and direct biopsy of abdominal organs, laparoscopy is a preferred technique that facilitates diagnosis, preoperative assessment, and therapy of ascites [34]. It has been utilized to confirm the diagnosis in ascites of unknown origin in terms of cirrhotic or non-cirrhotic as carcinoma peritonei, tuberculous peritonitis, or nephrogenic origin. Moreover, it is also used in peritoneal lavage cytology, preoperative assessment and staging of gastric, pancreatic and liver neoplasms, and judgment of curability of the abdominal carcinomas. Therapeutic roles were also discussed as in hemorrhagic pancreatitis, chylous ascites and catheter deployment for dialysis, and neoadjuvant chemotherapy [35]. A systematic approach to treatment using subcutaneous octreotide and a fat-free diet, resulting in complete resolution of the condition [36]. For this condition, there was no surgical mortality, and the morbidity was as low as 1% [14].

Constrictive pericarditis is a rare cause of massive ascites. Clinical manifestations of constrictive pericarditis are usually insidious. Over half of the patients with constrictive pericarditis did not show cardiopulmonary symptoms such as dyspnea and orthopnea, and therefore patients often present to non-cardiac physicians at their initial onset of symptoms. Ascites can be a manifestation of effusive-constrictive pericarditis [37]. Patients with untreated constrictive pericarditis sooner or later have ascites, edema, and pleural effusions [38]. van Deuren et al. [39] summarized the extracardiac manifestations of severe patients with constrictive pericarditis: ascites (100%), hepatomegaly (100%), edema (85.7%), narrow pulse pressure (≤35 mmHg) (71.4%), electrocardiogram abnormalities (100%) and pericardial calcifications on the chest X-ray (71.4%). Low serum protein may be resulted from slightly raised liver enzymes and a protein-losing enteropathy. Ascites secondary to constrictive pericarditis typically occurs before the edema of the lower limbs, and thus referred to as “ascites praecox” [40]. Long history of stable, insidiously progressive ascites strongly exclude malignant or infectious etiologies [41]. No exertional dyspnea prior to ascites may suggest a non-cardiac origin of increased right heart pressures [41]. Augmented lymph production and high impedance to lymph drainage caused by central venous hypertension were considered etiologies of ascites of constrictive pericarditis [42].

The diagnosis should be evident from right heart catheterization and computed tomography of the thickness or calcification of the pericardium [43]. Pericardial calcification or thickening may be present on X-ray, computed tomography and magnetic resonance imaging [44]. An SAAG ≥1.1 g/dL and an ascites fluid total protein >2.5 g/dL are typical of constrictive pericardial disease, whereas liver cirrhosis displays a higher serum-ascites albumin gradient >1.1 g/dL but an ascites fluid total protein <2.5 g/dL [45]. A comprehensive literature search of “serum ascites albumin gradient” and “pericarditis” yielded five reports with seven patients [10, 37, 45-47], totally eight patients including the present one (**Table 3**). Of the patient setting, four were males and four were females. Their ages were 53.8 ± 18.4 (range, 22-77; median, 55) years (n = 8). The duration of ascites was 27.1 ± 23.3 (range, 0.3-60; median, 24) months (n = 6). Their ascetic total protein was 4.3 ± 0.6 (range, 3.2-5.2; median, 4.2) g/dL (n = 7), and their SAAG was 1.58 ± 0.08 (range, 1.48-1.7; median, 1.6) g/dL. No correlation was found between ascites total protein and SAAG ($Y = -0.1020X + 2.0418$, $r = -0.5078$, $p = 0.3824$). Imaging showed that

Table 3. Serum ascites albumin gradient in patients with chronic constrictive pericarditis

Year	Author	Gender	Age (year)	Duration of ascites (m)	Total ascites protein (g/dL)	SAAG (g/dL)	Pathology of pericardium	Pericardiectomy	Prognosis
2002	Kerzner et al. [46]	f	58	?	5.2	1.5	Marked pericardial thickening (MRI)	Yes	No further ascites
2012	Howard et al. [45]	m	77	24	3.9	high	Extensive pericardial calcification (computed tomography)	No	Died 18 months of unrelated causes
2012	Howard et al. [45]	m	72	?	3.2	high	Unremarkable (computed tomography)	Yes	Died intraoperatively of severe bleeding of pericardiectomy site
2012	Barosa et al. [37]	m	34	24	--	>1.1	Thickened (MRI)	Yes	
2013	Doustkami et al. [47]	m	52	0.3	4.1	1.6	Thickened and calcified (computed tomography and echocardiography)	Yes	Improved
2013	George et al. [10]	f	63	6	4.2	1.6	Marked thickening of the parietal pericardium (MRI, pathology)	Yes	Improved
2013	George et al. [10]	f	52	60	4.6	1.7	Normal, thin parietal pericardium (pathology)	Yes	Improved
2014	present	f	22	48	4.73	1.48	Thickened and calcified (computed tomography)	Yes	Improved with mild ascites

the pericardium was thickening in 3 (37.5%), calcified in 1 (12.5%), both thickening and calcified in 2 (25%), and with normal or unremarkable changes in 2 (25%) patients, respectively ($\chi^2 = 1.333$, $p = 0.72123$; Fisher's exact test). Seven (87.5%) patients received a pericardiectomy, and 1 (12.5%) patient showed good response to diuretic treatment and thus a pericardiectomy was not performed ($\chi^2 = 9$, $p = 0.0027$; Fisher's exact test). Six (75%) patients had significant clinical improvement after pericardiectomy, and 2 (25%) patients died ($\chi^2 = 4$, $p = 0.0455$; Fisher's exact test). The patient had diuretic treatment died of unrelated causes 18 months later, and another patient died intraoperatively of severe bleeding of pericardiectomy site.

In overall, most patients with constrictive pericarditis had improved symptoms after pericardiectomy [48]. Functional improvement was observed in 88% patients with pericardiectomy at the 1-year follow-up [49]. McCaughan et al. [50] reported that 28% of patients presented low output syndrome following pericardiectomy, independently of the extension of pericardial resection, but related to ventricular dysfunction associated with cardiac dilation and myocardial atrophy. Chowdhury et al. [51] reported total pericardiectomy was performed in 338 (85.6%) patients, and partial pericardiectomy in 57 (14.4%) patients. Operative and late mortality rates were 7.6% and 4.9% for total and partial pericardiectomy, respectively. The risk of death was 4.5 times higher in patients undergoing partial pericardiectomy [51]. Advanced age, atrial fibrillation, concomitant tricuspid insufficiency, inotropic support and low cardiac output were significant predictors of mortality. Actuarial survival at 5 years was 75.9% ± 9.14% [49].

This patient was unique with massive ascites of unknown origin as a principle manifestation of chronic calcified constrictive pericarditis in an ectopic heart. The patient was lack of significant clinical features of constrictive pericarditis except for a low voltage of the QRS waves on the electrocardiogram, thereby making the diagnosis difficult. Although calcified pericardium was not as broad as the patient with eggshell calcification reported by Son et al. [7], the heart was severely constrained by the fibrocalcified pericardium as an underlying etiology of massive ascites production and successive umbilical hernia.

In conclusion, massive ascites of unknown origin as a principle manifestation of constrictive pericarditis is rare. Such a condition often leads to a delayed diagnosis and further treatment. Pericardiectomy can be a radical solution for the treatment of calcified constrictive pericarditis.

Disclosure of conflict of interest

None.

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