OPEN

A case report of chylous ascites after cardiac surgery

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Abstract

Background: Chylous ascites (CA), an emulsion rich in triglycerides, is a rare type of ascites, especially after cardiac surgery. Also, there are few reports of the occurrence of CA after mitral valve replacement, aortic valve replacement, tricuspid valvuloplasty, and coronary artery bypass grafting.

Case presentation: We report the case of a 53-year-old man who underwent surgery for a diagnosis of mitral incompetence, aortic incompetence, tricuspid incompetence, and coronary atherosclerotic heart disease. Fifteen days after surgery, 3000 mL of milky-yellow ascites appeared in the abdominal cavity, diagnosed as CA by laboratory examination. The ascites gradually disappeared after being drained, and the patients improved. The curative was objective, but the exact cause is not clear.

Conclusion: Although CA is rare, it may occur during the surgical intervention in cardiac surgery.

Keywords: Cardiac intensive care unit, Cardiac valve replacement, Case report, Chylous ascites, Coronary artery bypass graft, Department of cardiac surgery

Introduction

Chylous ascites (CA) is a rare milky, triglyceride-rich ascites[1]; and it has been reported that the incidence rate of CA is about 1 in 20,000 people.[2] There is no obvious specificity in clinical manifestation and signs, mainly abdominal distension.[3] The main cause is the infiltration of thoracic or intestinal lymph into the abdominal cavity due to the destruction of the body's lymphatic system. The level of triglyceride is an important indicator for the diagnosis of CA. Currently, domestic and foreign consensus shows that a triglyceride level in ascites exceeding 200 mg/dL, and lymphangiography is the diagnostic standard for diagnosing CA.[4,5] Chylous ascites is uncommon after cardiac surgery as the thoracic duct is usually out of scope during routine median sternotomy and is less likely to disrupt the peritoneal lymphatic system. Therefore, we consider CA's possibility after cardiac surgery to be less likely, but it may also not be excluded as a complication after cardiac surgery.

Case presentation

A 53-year-old man was admitted to the hospital, with chest tightness for more than 2 years, which was aggravated after exercise for 1 week. Vital signs at admission: temperature, 36.5°C; pulse rate, 75 bpm; respiratory rate, 18 breaths/min; blood pressure, 120/75 mmHg. Autonomous body position, physical examination cooperation, clear respiratory sounds, and no rales in the bilateral lungs. Cardiac examination revealed: normal heart sounds and regular rhythm, diastolic sigh-like murmurs in the aortic valve auscultation area could be heard, and mitral valve auscultation showed systolic blow-like murmur, with no pericardial friction rub. Abdominal examination revealed a flat abdomen, with no tenderness or rebound pain. The liver and spleen were not palpable under the ribs. There was no edema in the bilateral lower extremities, muscle strength and muscle tension of the limbs were normal, and Barthel's sign was negative.

The patient underwent surgical treatment 11 years after myasthenia gravis and 6 years after regular tuberculosis treatment. The coronary stenting (anterior descending branch) was performed 5 years after coronary heart disease. After completing the relevant examination, the preoperative diagnosis: (1) mitral regurgitation (severe), aortic regurgitation (moderate), tricuspid regurgitation (mild-moderate), bilateral atrial enlargement, and pulmonary hypertension; (2) pericardial effusion; (3) coronary atherosclerosis after the percutaneous coronary intervention; (4) carotid atherosclerosis; (5) aortic sclerosis; (6) Emphysema; (7) pleural effusion; (8) after tuberculosis treatment; (9) after myasthenia gravis; and (10) brain ischemic changes. After excluding surgical contraindication, the patient underwent mitral valve replacement (ST Jude Master mechanical valve St Jude Medical Inc, St Paul, Minn), aortic valve replacement (ST Jude Master mechanical valve), tricuspid valvuloplasty (De Vega), and coronary artery bypass surgery (great saphenous vein-anterior descending branch) surgery under general anesthesia was performed. The patient was transferred to the cardiac intensive care unit for further postoperative treatment.

Hypotension and airway hemorrhage occurred the day after surgery. The patient's condition improved after symptomatic support treatment, such as an intra-aortic balloon pump (IABP) to maintain blood pressure, vasoactive drugs to preserve circulation, bronchoscopy to reduce airway bleeding, and fluid rehydration to control internal environment stability and anti-bacterial administration. On the second day, the bedside echocardiography showed that the patient's cardiac prosthetic valve was good, the left ventricular systolic function data was within the normal low range, and the right ventricular systolic function was slightly low. The IABP was removed on the fourth day after surgery. However, poor preoperative

The datasets generated during and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Table 1

Nutritional Composition of 500 mL Enteral Nutritional Emulsion (TPF)

Items	Numerical Value		
Energy (KJ) (kcal)	2100 (500 kcal)	Cu (μg)	900
		Mn (μg)	1650
Protein (g)	20.0	F (mg)	0.5
Nitrogen (g)	3.15	Mo (μg)	50
NPC: N	133:1	Se (μg)	28.5
Energy%	16	Cr (μg)	33.4
Carbohydrate (g)	61.5	I (μg)	65
Sugar (g)	5.0	Vitamin	
Polysaccharide (g)	55.5	A (IU)	1365.3
Lactose (g)	<0.125	Carotenoid mixture (mg)	1.0
Energy%	49	D (μg)	3.5
Fat (g)	19.45	E (mg)	9.35
Saturated (g)	1.45	K (μg)	26.5
Polyunsaturated (g)	6.15	B1 (mg)	0.75
ω6/ω3	5:1	B2 (mg)	0.8
Energy%	35	Nicotinic acid (mg)	4.35
Dietary fiber (g)	7.5	Pantothenic acid (mg)	2.65
Water (g)	425	B6 (mg)	0.85
Mineral		Folic acid (μg)	133.5
Na (mg)	500	B12 (μg)	1.05
K (mg)	750	Biotin (μg)	20
Cl (mg)	625	Vitamin C (mg)	50
Ca (mg)	400	Choline (mg)	185
P (mg)	360	PH	6.4–6.8
Mg (mg)	115	Osmotic pressure (mOsm/L)	250
Fe (mg)	8	Renal solute load (mOsm/L)	300
Zn (mg)	6		

pulmonary function made it difficult to remove the endotracheal tube, and the patient started enteral nutrition (Enteral Nutritional Emulsion [TPF] 1000–1500 mL, composed of water, maltodextrin, casein, vegetable oil, dietary fiber, minerals, vitamins, and trace elements, etc., the specific nutritional composition is shown in Table 1) as supportive therapy on the third day after surgery.

On the 14th day after surgery, the patient developed abdominal distension, and the bedside ultrasonography revealed a large amount of pleural and abdominal fluid. Hence, under bedside color Doppler ultrasound guidance, puncture and drainage of the thoracic and abdominal cavities were performed. Clear yellow fluid drained from the thoracic cavity, and light yellow turbidite was drained from the abdominal cavity (Fig. 1). Fifteen days after surgery, routine and biochemical examination of the pleural fluid was performed, and the results are presented in Table 2. The results of routine ascites and biochemical examination are shown in Table 3. Ascites biochemistry suggested high triglycerides, and we considered CA. Therefore, we improved the ascites lipid test, and the results are listed in Table 4. The result showed no obvious abnormalities in the lipids of in blood sample. Meanwhile, we performed an enhanced computed tomography (CT) scan of the chest and abdomen, which suggested no significant abnormalities, including the location of the abdominal drainage tube (located extra-intestinal).

Treatment and patient outcome

We performed the consultation and asked the gastroenterology, gastrointestinal surgery, and endocrinology departments to specify the diagnosis and treatment plans. Due to conditions, our hospital could not conduct lymphangiography, so we adjusted the nutrition program from external nutrition to parenteral nutritional support + medium-chain triglyceride diet + parenteral nutrition (10% glucose injection 100 mL + 50% glucose injection 300 mL + a variety of oil fat emulsion injection 200 mL + 20 compound amino acid injection 400 mL + water-soluble vitamin for injection 10 mL + fat-soluble vitamin injection 10 mL + a variety of trace element injection 10 mL) support. Over the next 10 days, the patient's abdominal drainage fluid gradually became clear in color, and the ascites decreased. During the routine and biochemical examination, the ascites was diagnosed as exudate, and CA did not recur during the follow-up treatment (1 month).

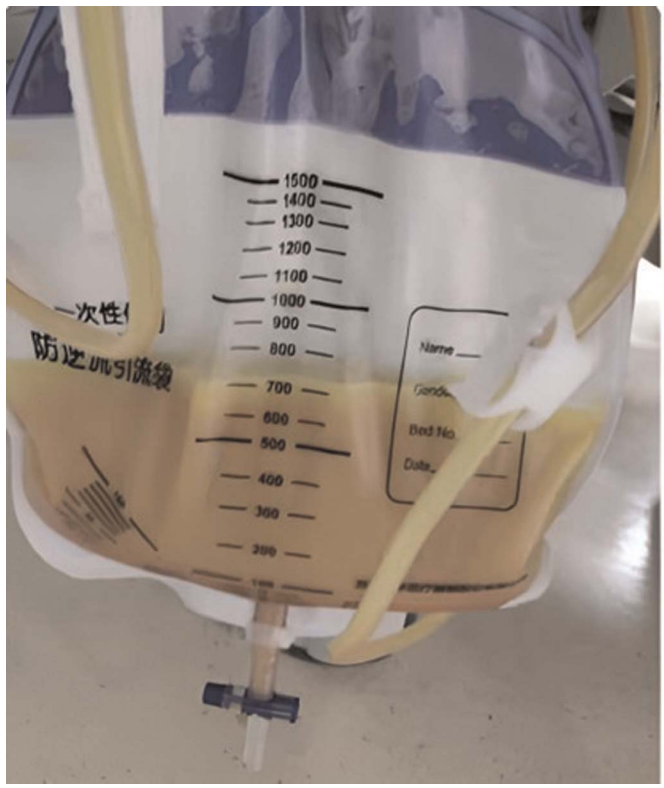

Figure 1. Drainage bag after ascites puncture.

Table 2

Routine and Biochemical Summary of Pleural Effusion

Items	Numerical Value	Unit
Color	Yellow	
Transparency	Limpid	
Red blood cell	2+	
Karyocyte	293	10^6/L
Neutrophilic granulocyte percentage	16	
Lymphocyte percentage	47	
Monocyte percentage	37	
Basophilic granulocyte percentage	0	
Eosinophilic granulocyte percentage	0	
Total protein	24.5	g/L
Chlorine	103.1	mmol/L
Triglyceride	1.08	mmol/L
Lactic dehydrogenase	255	U/L
Glucose	5.29	mmol/L
Amylase	27	U/L

Discussion

CA is caused by the destruction of the lymphatic system in the body. The root cause is the interruption of lymph flow. The lymphatic circulation is the interconnecting network between the lymphatic and the venous systems in which the large macromolecular fats and proteins absorbed through the intestine are absorbed and merged into the intestinal trunk through the lymphatic vessels, and then into the left internal jugular vein and subclavian vein through the chylous cistern and thoracic duct (Fig. 2). Lymphatic vessels drive lymphatic circulation by the active (contraction and relaxation) and passive external pressure (skeletal muscle, central venous pressure, respiratory movement, and pulsing of adjacent arteries).[6]

The most common symptoms of CA are abdominal distention, pain, diarrhea, dysphagia, and progressive peripheral edema. Other symptoms may include nausea, vomiting, malnutrition, fever, night sweats, shortness of breath, weight gain, and increased abdominal circumference because of increased abdominal pressure.[7] Studies show that there are many causes of CA, including thoracic duct block or destruction (trauma, surgery, infection, acute pancreatitis, cardiovascular disease, and liver cirrhosis), invasion and destruction of normal lymph flow (lymphoma, sarcomas, leukemia, solid organ malignancies) and lymphangitic abnormalities (lymphangiectasis in adults or children, Klippel-Trenaunay-Weber syndrome, and Yellow-nail syndrome). In developed countries, the basic causes of CA are abdominal malignancy and cirrhosis, where infectious diseases, especially tuberculosis, are common in developing countries. In adults, abdominal malignancy, liver cirrhosis, lymphatic interruption, and infection after abdominal surgery are the main causes, while congenital lymphatic system abnormalities and trauma are considered the most common factors.[8]

Browse et al.[9] proposed 3 mechanisms for the destruction of the CA lymphatic system. The first is congenital abnormalities (such as congenital lymphangiectasia) that cause lymphatic leakage. The second is fibrosis of the chronic lymphatic system (like a malignant tumor or tuberculosis) leading to the obstruction of lymph flow from the gut to the chylous sac into the abdominal cavity. Another mechanism is acquired lymphatic damage (for example, surgery, trauma, and liver cirrhosis), which is caused by lymphatic canal leakage or lymphatic pressure caused by increased.

The treatment of CA is multifaceted, and current treatments mainly include the following aspects: nondrug therapy, drug therapy, and surgical therapy. Nonpharmacological treatments maintain an optimal nutritional balance to reduce lymphatic production and flow. The key first step of treatment is to optimize the patient's nutritional status, consisting of low salt, high-protein, low-fat diet with medium-chain triglycerides. If the above measures are ineffective, enteral nutrition should be stopped, and total parenteral nutrition should be provided.[3,8,10] Drug therapy includes orlistat, somatostatin, and their synthetic analogs, octreotide and etilefrine. Orlistat is a gastric and pancreatic lipase inhibitor that has been shown to reduce the triglyceride concentration in ascites and could be used as an adjunct.[11] The exact mechanism is not fully understood for somatostatin and its synthetic octreotide analogs.

However, studies have found that they can be applied to a specific receptor in intestinal wall lymph drainage. They inhibit glucagon and other intestinal peptide-mediated proteins by reducing portal pressure and intestinal peristalsis and decreasing intestinal fat absorption.[12] Etiforine is an adrenergic agonist with sympathetic-like effects, mainly through the contraction of lymphatic smooth muscle.[13] Surgical treatment consists of laparotomy, laparoscopic suturing or ligating damaged lymphatic vessels, intrahepatic portal shunt via jugular vein, splenorenal shunt, and lymphatic blocking embolization.[14–16] Although there are many surgical options for celiac ascites, conservative management remains the cornerstone of treatment until the disease progresses.

In the pale yellow turbid-like ascites, triglyceride was 6.48 mmol/L (574.13 mg/dL), which was consistent with the diagnostic evidence of CA. The patient also presented with abdominal pain and

Table 3

Routine and Biochemical Summary of Ascites

Items	Numerical Value	Unit
Color	Yellow	
Transparency	Turbidity	
Red blood cell	2+	
Karyocyte	288	10^6/L
Neutrophilic granulocyte percentage	26	
Lymphocyte percentage	33	
Monocyte percentage	41	
Eosinophilic granulocyte percentage	<1	
Basophilic granulocyte percentage	<1	
Total protein	32.9	g/L
Chlorine	102.6	mmol/L
Triglyceride	6.48	mmol/L
Lactic dehydrogenase	233	U/L
Glucose	6.19	mmol/L
Amylase	41	U/L

Table 4

Three Blood Lipids of Ascites

Items	Numerical Value	Unit
Cholesterol	1.77	mmol/L
Triglyceride	9.31	mmol/L
High-density lipoprotein	0.04	mmol/L

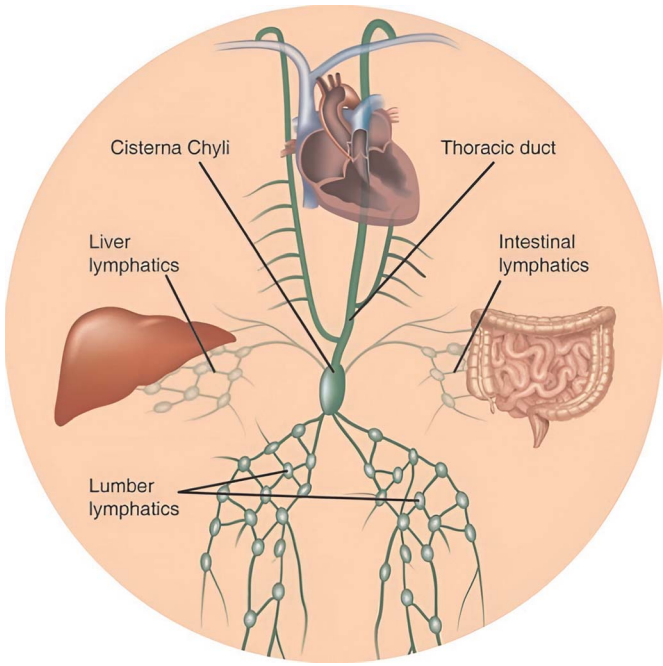

Figure 2. Distribution of lymphatic vessels in the body.

abdominal distension. However, finding a clear etiology during therapy is not easy. The chest CT with contrast enhancement did not find tumor markers, abdominal ultrasound, or other infectious diseases. The patient had a history of tuberculosis treatment, but there was no evidence of tuberculosis in the chest CT or the tuberculosis test. Hence, we considered the possibility unlikely. Because of temporary constraints, we could not perform lymphography check.

We speculated that the following reasons might be responsible for this CA. First, the catheter traction caused by the operation of the heart and large vessels during cardiac surgery resulted in thrombosis at the junction of the left jugular and subclavian veins, obstructing lymphatic reflux. Second, lymphatic vessel damage caused during surgery may have resulted in changes in the position of the thoracic duct in the patient who had previously undergone thoracic thymoma surgery. In addition, according to the recollection of the operator, the thoracic adhesion during intraoperative exposure was heavy, and the separation of the surgical field was long and difficult, which resulted in injury to the thoracic duct.

Third, after surgery, the patient suffered from high fever, and the alveolar lavage fluid was cultured many times to produce *Klebsiella pneumoniae, Burkholderia cepacia,* and *Candida tropical,* is which we considered the cause of the infection. Fourth, the patient had tricuspid incompetence and pulmonary hypertension before surgery and the postoperative bedside echocardiography indicated that the left ventricular systolic function was normal, right ventricular systolic function was low with a diastolic limitation which had resulted in excessive right heart preload pressure and affected lymphatic reflux. Furthermore, after tricuspid valve formation, the patient had valve regurgitation during postoperative recovery, resulting in excessive right heart preload.

Fifth, pericardia constriction was caused by pericardia adhesion. We performed a bedside echocardiography on the eighth postoperative day, which showed mild to moderate tricuspid valve regurgitation, pericardial adhesion, large left atrium, and limited left and right ventricular diastolic function, resulting from increased lymphatic pressure. Finally, the patient required prolonged mechanical ventilation. With postoperative bleeding of the bronchial and poor pulmonary function, a tracheotomy was performed on the 12th day after surgery, and he was started on oxygen cure of wetting treatment on the 27th day. Prolonged mechanical positive pressure ventilation has the opposite effect on hemodynamics, hinders venous return, leads to change in vena cava pressure, and affects lymphatic return.

Conclusion

After the drainage and the diet improvement, the patient's celiac ascites disappeared. We believe that the cardiac surgery was the cause of these factors, but we could not explain the exact mechanism.

Conflict of interest statement

The authors declare no conflict of interest.

Author contributions

Yuan S, Xie P, and Li P wrote the main article. Liu X prepared the figures. All authors have read and approved the final article.

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Ethical approval of studies and informed consent

This study was approved by the University of Hong Kong-Shenzhen Hospital Ethics Committee (no. [2022] 094, April 25, 2022) and written informed consent was obtained from the patient. This article has the consent of the patient for the use of his data and for the publication of the data that appear in the article. All methods were carried out following the relevant guidelines and conformed to the declaration of Helsinki.

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