CASE REPORT

Atraumatic chylous ascites: an unusual presentation of bladder cancer

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SUMMARY

We describe a case of bladder cancer presenting with atraumatic chylous ascites, which remains an extremely rare presentation of this condition. A previously well, elderly ex-smoker with no prior history of abdominal surgery was referred for investigation of progressive dyspnoea, increasing peripheral oedema and new-onset ascites, on a background of long-standing alcohol consumption (four standard drinks daily). Liver biochemistry and coagulation profile were normal apart from marked hypoalbuminaemia. Doppler ultrasound of the liver demonstrated normal echotexture and patent vasculature. Abdominal paracentesis yielded 8 L of milk-coloured, triglyceride-rich fluid with abundant malignant cells. Urine cytology demonstrated malignant transitional cells, with radiological evidence of a large enhancing bladder mass, with evidence of adjacent lymphadenopathy and omental involvement. A diagnosis of metastatic stage IV transitional cell bladder cancer was made. The patient declined palliative chemotherapy and passed away 2 months after their initial presentation.

BACKGROUND

We describe a case of bladder cancer presenting with atraumatic chylous ascites, which remains an extremely rare presentation of this condition. Our review of the literature demonstrated only one published case of atraumatic chylous ascites associated with advanced transitional cell carcinoma.¹ In that case, the patient already had an established diagnosis of bladder cancer at the onset of ascites. Patients with urothelial carcinoma have been known to present with chylous ascites; however, this has largely occurred in the postnephrectomy setting, with one case series reporting an incidence of 5.1% following laparoscopic nephrectomy.²

CASE PRESENTATION

A previously well, elderly ex-smoker with no prior history of abdominal surgery was referred for investigation of progressive dyspnoea. Their medical history included hypertension, osteoarthritis, gout, atrial fibrillation and long-standing alcohol consumption (four standard drinks daily). The patient's preadmission medications included aspirin 100mg daily, paracetamol modified release 1330mg two times a day, frusemide 20mg daily, telmisartan 40mg daily, prazosin 2mg mane and 1mg nocte. Their hospital presentation was preceded by a 6-week history of increasing peripheral oedema, new-onset ascites,

anorexia and generalised lethargy. Of note, there was no history of infective symptoms such as fevers or night sweats. Pertinent findings on clinical examination included an elevated jugular venous pressure to the angle of the jaw, large volume ascites and bilateral lower limb pitting oedema extending up to the lower abdominal wall. The patient was afebrile with a respiratory rate of 20 breaths per minute, blood pressure of 144/77 mm Hg and also noted to be in atrial fibrillation with a ventricular rate of 108 beats per minute. Abdomen examination revealed no scars suggestive of previous surgery. The abdomen was soft to palpation but showed significant distension with evidence of shifting dullness suggestive of ascites. The patient was initially admitted under the gastroenterology team with suspicions of decompensated alcoholic liver disease.

INVESTIGATIONS

Initial blood evaluation (figure 1) demonstrated normal full blood count, electrolytes, renal function and coagulation profile. Liver biochemistry was unremarkable apart from marked hypoalbuminaemia and mildly elevated serum globulins. Initial chest X-ray demonstrated small bilateral pleural effusions, minor bibasal atelectasis, normal cardiomeastinal contour and no evidence of consolidation or pulmonary oedema. The albumin to creatine ratio was markedly elevated at 42.5 mg/mmoL in keeping with albuminuria.

	Serum	Units	
Hb	136	g/L	130–180
Plts	273	x10 ⁹ /L	150–400
Na	140	µmol/L	62–106
K	4.6		
Cr	82	µmol/L	62–106
Ur	5.7	mmol/L	3.0–9.2
Bili	6	µmol/L	<18
ALT	14	U/L	<41
GGT	37	U/L	<60
ALP	88	U/L	40–130
Alb	22	g/L	35–52
INR	1.1		

Figure 1 Admission blood results.

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	Ascitic fluid	Serum	Units	Reference
Albumin	17.4	22	g/L	35–52
Protein	39	64	g/L	64–83
Cholesterol	2.28	2.2	mmol/L	<5.5
Triglycerides	9.3	0.8	mmol/L	<2.0
Erythrocytes	620		$\times 10^6/L$	
Neutrophils	87		$\times 10^6/L$	
Culture	No bacteria			
Cytology	Malignant cells			

Figure 2 Characteristics of serum and ascitic fluid.

The patient underwent uncomplicated diagnostic abdominal paracentesis, yielding 8 L of milk-coloured fluid. The fluid was sent off for microscopy, culture, cytology and biochemistry (figure 2). Initial microscopy revealed 620 erythrocytes, 20 unidentified cells and 250 leucocytes composed of 87 neutrophils and 163 mononuclear cells with no bacteria seen on Gram stain. Serum albumin was 2.2 g/dL, while ascitic albumin was 1.7 g/dL, demonstrating a low serum albumin to ascitic gradient (SAAG) of 0.5 g/dL, which did not suggest a portal hypertensive aetiology. Serum protein was 6.4 g/dL, while ascitic protein was 3.9 g/dL, demonstrating a high serum protein to ascitic gradient of 2.5 g/dL. Serum cholesterol and triglycerides were 2.2 mmol/L and 0.8 mmol/L, respectively, with corresponding ascitic values of 2.28 mmol/L and 9.3 mmol/L, suggestive of chylous ascites.

Ascitic fluid cytology revealed abundant malignant cells presenting in papillary like structures, with marked hyperchromatic pleomorphic nuclei with moderately abundant cytoplasm (figure 3). Immunohistochemical stains demonstrated strong positive staining for CK7, patchy staining for CK20, equivocal patchy staining for uroplakin and positive staining for GATA-3. Subsequent urine cytology revealed transitional, inflammatory and malignant cells. This profile was thought to be most in keeping with metastatic urothelial carcinoma. Alpha fetoprotein was normal, although CA19.9 was elevated at 166 kU/L.

Doppler liver ultrasound postabdominal paracentesis revealed mild ascites without features of cirrhosis, portal hypertension or portal vein thrombosis. However, note was made of moderate left-sided hydronephrosis secondary to an irregular, hypoechoic, exophytic mass in the bladder measuring 10.8 $\times$ 4.6 $\times$ 4.1 cm. This was thought to represent a bladder malignancy. The patient proceeded to a staging CT scan of the chest, abdomen and pelvis, which demonstrated a large enhancing mass within the base and left lateral wall of the bladder causing obstruction of the

Figure 3 Loosely cohesive groups of malignant epithelioid cells are seen in an inflammatory background (ascitic fluid, Papanicolaou stain, $\times 400$).

left distal ureter with mild-moderate proximal dilatation of the left collecting system. Several adjacent small soft tissue nodules were thought to represent local tumour invasion, while omental caking, peritoneal nodularity and aortocaval lymphadenopathy were suggestive of metastatic disease. Notably, there were no definitive osseous or pulmonary metastasis detected, and the thoracic duct was not well seen.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of chylous ascites

- Neoplastic: lymphoma, lymphangiomyomatosis, carcinoid, others
- Congenital: primary lymphatic hypoplasia, yellow nail syndrome, primary lymphatic hyperplasia, Klippel-Trenaunay syndrome
- Postoperative: abdominal aneurysm repair, retroperitoneal lymph node dissection, catheter placement for peritoneal dialysis, inferior vena cava resection
- Infectious: tuberculosis, filariasis, *Mycobacterium avium*
- Trauma: blunt abdominal trauma
- Inflammatory: radiation, pancreatitis, constrictive pericarditis, sarcoidosis, coeliac sprue, Whipple's disease, retroperitoneal fibrosis

TREATMENT

Following a good response to intravenous diuretic therapy with documented weight loss, the patient was transitioned onto regular ongoing oral diuretic therapy. The patient was also commenced on a regular beta-blocker in the context of atrial fibrillation with rapid ventricular rate. Due to the cytology and radiological findings, the case was discussed with the urology team. However, as the patient had metastatic disease, surgical intervention was not deemed appropriate. A cystoscopy to establish a formal tissue diagnosis was not deemed necessary as the urine cytology was consistent with transitional cell carcinoma. The oncology team reviewed the patient and subsequently suggested an outpatient review for consideration of palliative chemotherapy as the patient's Eastern Cooperative Oncology Group status was 2.

OUTCOME AND FOLLOW-UP

Four weeks following their initial presentation, the patient required a brief admission for therapeutic abdominal paracentesis as his ascites had reaccumulated. The patient was subsequently referred to oncology outpatients where the role of palliative chemotherapy was discussed. Following careful consideration and discussion with family members, the patient declined chemotherapy, and passed away within 2 months of their initial hospital presentation.

DISCUSSION

The presence of milky, triglyceride-rich peritoneal fluid represents a rare form of ascites often known as chylous ascites and is due to the presence of thoracic or intestinal lymph in the abdominal cavity.³ The reported incidence of chylous ascites was approximately 1 in 20000 admissions at a large hospital over 20 years, although the incidence is believed to be increasing in the context of more aggressive cardiothoracic and abdominal intervention and improved rates of survival among oncology patients.⁴

Chylous ascites arises following disturbance of the lymphatic system. This can arise from direct trauma, or lymphatic obstruction secondary to acquired or congenital aetiologies. Chylous

ascites can also be divided into atraumatic and traumatic aetiologies, and a systematic review of 190 patients across 131 studies demonstrated that the most common adult causes of atraumatic ascites included lymphatic anomalies (32%), malignancy (17%), cirrhosis (11%) and mycobacterial infection (15%).^{5–6} Intra-abdominal malignancy, lymphatic abnormalities and cirrhosis account for over two-thirds of all cases in the developed world, with infectious aetiologies accounting for the majority of cases across developing countries.³

Lymphomas account for the majority of malignancy-associated chylous ascites with breast, oesophageal, colon, renal, testicular, pancreatic, prostatic and gynaecological malignancies representing the most common solid neoplastic aetiologies.^{5–9} Malignancy-associated chylous ascites develops from progressive obstruction of lymph flow owing to malignant lymphatic invasion; peritoneal lymph accumulation occurs secondary to leakage through dilated subserosal lymphatics.¹⁰

A small subset (<1%) of cirrhotic patients develop chylous ascites. It is thought to develop secondary to the rupture of dilated serosal lymphatic channels. Although new-onset chylous ascites may be the presenting feature of cirrhosis, it can manifest at any stage of disease, particularly as a consequence of shunt surgery, locally advanced hepatocellular carcinoma, sclerotherapy-induced thoracic duct injury or other causes of portal hypertension such as portal vein thrombosis.^{11–14}

The vast majority of patients with chylous ascites present with progressive painless abdominal distention, with associated non-specific complaints such as abdominal pain, altered bowel habit, fevers or night sweats, weight loss or gain and breathlessness owing to increased abdominal girth.^{7–8,15}

Diagnostic evaluation should include careful history and examination, paying particular attention to any history of recent surgery, clinical history suggestive of underlying malignancy or stigmata of chronic liver disease on physical examination.

Learning points

- Diagnostic abdominal paracentesis remains an important investigation to ascertain the aetiology of new-onset ascites. Ascitic serum albumin to ascitic gradient (SAAG), biochemistry, culture and cytology remain important first-line investigations.
- Chylous ascites typically has a milky appearance, with findings of a markedly elevated ascitic-to-serum triglyceride count suggestive of the diagnosis.
- In the absence of underlying liver disease, atraumatic chylous ascites commonly stems from a malignant aetiology within the elderly population. Associated findings on ascitic fluid analysis include low SAAG, high ascitic to serum protein gradient and malignant cytology.

Chylous ascites is diagnosed on the basis of abdominal paracentesis with characteristic findings of elevated ascitic triglyceride levels typically above 200 mg/dL. Ascitic fluid should also be analysed for cell count, culture, Gram stain, total protein concentration, albumin, glucose, lactate dehydrogenase, amylase and cytology.¹⁶ SAAG should also be calculated to help ascertain the aetiology of the ascites.

In the setting of symptomatic or moderate to severe volume ascites, therapeutic abdominal paracentesis remains an effective means of relieving breathlessness and abdominal discomfort. Reaccumulation of ascites may demand repeated paracentesis, especially in the context of incurable disease.

Contributors AS, RA and AL reviewed the literature and prepared the manuscript. AS and DJ have reviewed and revised the manuscript critically. All authors prepared the final version of the manuscript and approved the final draft prior to submission.

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