European Journal of Case Reports in Internal Medicine

Chylous Ascites due to Mantle Cell Lymphoma

Ricardo Fernandes, Márcia Leite, Joana Cochicho, Rafaela Veríssimo, Agripino Oliveira

Internal Medicine Department, Centro Hospitalar de Vila Nova de Gaia-Espinho, Portugal

Doi: 10.12890/2018_000871- European Journal of Case Reports in Internal Medicine - © EFIM 2018

Received: 26/02/2017
Accepted: 05/03/2018
Published: 03/04/2018

How to cite this article: Fernandes R, Leite M, Cochicho J, Veríssimo R, Oliveira A. Chylous ascites due to mantle cell lymphoma. *EJCRIM* 2018;**5**: doi:10.12890/2018_000871.

Conflicts of Interests: The Authors declare that there are no competing interests.

ABSTRACT

Chylous ascites is rare and results in accumulation of lymph in the abdominal cavity, due to several mechanisms. The ascitic liquid is milky because of the high concentration of triglycerides (>200 mg/dl). The higher incidence compared to the past is explained by increased survival of patients with cancer and more aggressive surgery. We describe the case of an 87-year-old man admitted to the geriatric ward due to general oedema, chylous ascites and loss of weight, explained by mantle cell lymphoma.

LEARNING POINTS

- Chylous ascites is more frequent in trauma, but in some cases may be related to obstruction of the thoracic duct by retroperitoneal fibrosis, pancreatitis or neoplasms.
- It is a progressive, difficult to manage condition, with a spectrum of treatment ranging from a special diet to surgery.
- Its prognosis depends fundamentally on the predisposing condition.

KEYWORDS

Chylous ascites, mantle cell lymphoma, ascites

INTRODUCTION

Chylous ascites is a relatively rare form of ascites resulting in the accumulation of lymph in the abdominal cavity. It is characterized by a milky ascitic liquid with an elevated concentration of triglycerides (>200 mg/dl)[1]. Although there have been no recent large epidemiological studies, it is generally believed that its incidence has increased due to the longer survival of patients with cancer and more aggressive abdominal and cardiothoracic interventions.

The main pathophysiological mechanisms postulated for this condition are: (a) obstruction of the flow of lymph caused by external pressure leading to leakage from dilated subserosal lymphatic channels into the peritoneal cavity; (b) lymph exuding through the walls of dilated retroperitoneal vessels without valves, which leak fluid through a fistula into the peritoneal cavity, as in congenital lymphangiectasia; and (c) traumatic obstruction of the thoracic duct[2].

We describe a case of lymphoproliferative disease presenting as chylous ascites.

CASE REPORT

An 87-year-old man presented with abdominal distension, peripheral oedema and constitutional symptoms of fatigue, anorexia and involuntary weight loss (12.7%). His medical history was positive for hypertension and alcohol consumption of 36 g per day for the last 40 years. There was a positive family history of gastrointestinal cancer.

On physical examination, the patient had obvious ascites (grade III) and lower extremity oedema. Vital signs were normal. There was no

European Journal of Case Reports in Internal Medicine

jaundice or scleral icterus and also no signs of chronic liver disease, palmar erythema, enlarged parotids or gynecomastia. The abdomen was distended with bulging flanks, a fluid wave, shifting dullness and signs of hepatofugal collateral circulation. No abdominal organomegaly was detected. Cardiovascular and pulmonary examinations were normal. Pitting oedema was present in both legs up to the thighs. Two enlarged axillary lymph nodes were also detected. Initial investigation revealed a widened mediastinum on chest X-ray (*Fig. 1*).

Initial laboratory tests showed a normal haemogram and blood smear, erythrocyte sedimentation rate of 50 mm/hour (normal values: 0–20), C-reactive protein of 5.74 mg/dl, lactate dehydrogenase (LDH) of 370 IU/l (normal values: 135–225) and triglycerides of 88 mg/dl. Serum protein electrophoresis showed: albumin 3.02 (normal values: 3.9–5.1 mg/dl), alpha 1 0.23 g/dl, alpha 2 0.86 g/dl, beta (pt) 0.54 g/dl, and gamma 1.66 g/dl. Immunoglobulin G was elevated (1640.0 mg/dl), but immunoglobulins A and M were normal (*Table 1*).

Abdominal paracentesis revealed a milky peritoneal fluid with albumin of 1.1 g/dl and a serum-ascites albumin gradient of 2.1 g/dl. The ascitic fluid contained 1461 leucocytes/µl (79% mononuclear cells), triglycerides 2298 mg/dl, glucose 85 mg/dl, adenosine deaminase (ADA) 34 IU/l and LDH 174 IU/l. Culture was negative as was cytological examination for malignant cells. Further testing revealed an elevated B2-microglobulin of 11.7 mg/l (normal values 0.8–2.2). Viral serologies (HIV, HCV, HBV) were also negative. In the urinalysis no pathological alteration was found, except for low grade proteinuria. Serum electroimmunofixation was positive for IgM lambda.

Subsequent investigation with computerized tomography (CT) revealed multiple thoracic and abdominal lymphadenopathies forming large conglomerates, the largest abdominal mass having a diameter of 20 cm (*Fig. 2*).

The patient began oral furosemide 40 mg and spironolactone 100 mg with ascites volume reduction and better symptom control.

Excisional biopsy of the axillary adenopathy revealed mantle cell lymphoma with a diffuse architectural pattern, classic type with a proliferative index of 20% (*Fig. 3*). Bone marrow and gastrointestinal involvement was not investigated since treatment would not be effective. Stage III/IV mantle cell non-Hodgkin lymphoma was diagnosed and after discussing the goals of treatment, palliative chemotherapy according the PEPC protocol (prednisolone 20 mg once a day (OD), cyclophosphamide 50 mg OD, etoposide 50 mg OD and procarbazine 50 mg OD). The patient died from pneumonia 1 month after beginning treatment.

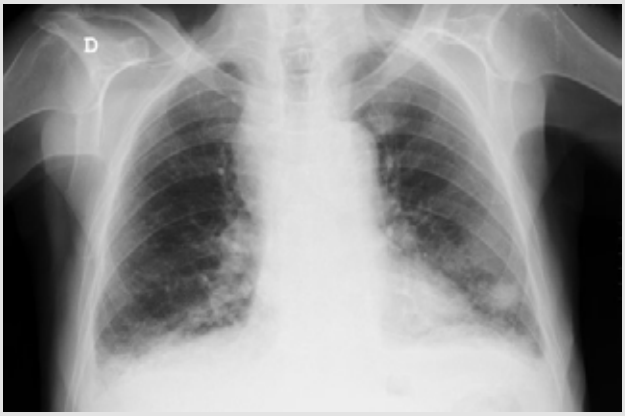

Figure 1. Enlargement of the mediastinum.

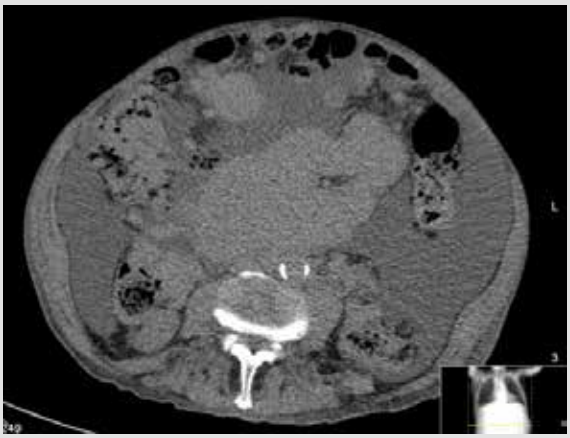

Figure 2. Abdominal CT scan showing the abdominal conglomerate

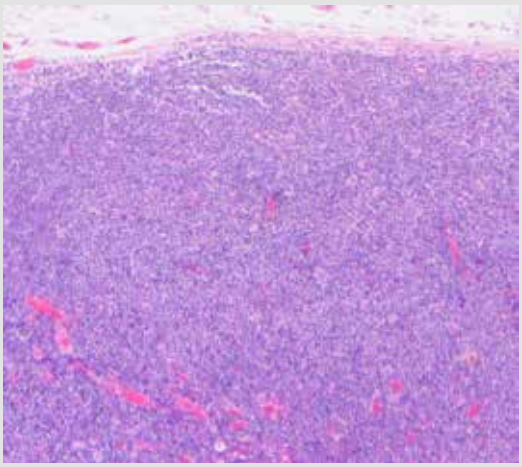

Figure 3. Histology of the axillary lymph node showing a diffuse architectural pattern and varying cytology with a proliferative index of 20%

European Journal
of Case Reports in
Internal Medicine

Analytic parameters	Admission	Day 2	Day 3	Day 9	Normal values
Haemoglobin (g/dl)	12.32	11.3	11.3	11.5	13–18
Erythrocytes (x10^{12})	4.05	3.67	3.67	3.78	4.4–5.9
Haematocrit (%)	37.1	33.1	33.1	34.8	40–52
Leucocytes (x10^{3})	8.55	6.09	5.60	6.82	3.8–10.6
Neutrophils (%)	78.5	73.6	71.1	71.3	1.3–8.8
Lymphocytes (x10^{3}/%)	1.13/13.2	0.95/15.6	0.84/15.0	1.25/18.3	1.0–4.8
Platelets (x10^{3}/µl)	296	280	296	360	150–440
Sedimentation velocity (mm/s)	–	50	–	–	0–15
Glucose (mg/dl)	86	76	–	–	60–100
Creatinine (mg/dl)	1.05	1.33	1.49	1.53	0.67–1.17
Urea (mg/dl)	49	51	49	95	17–50
Sodium (mmol/l)	135	134	134	131	136–145
Potassium (mmol/l)	5.14	5.54	5.19	4.94	3.5–5.0
Chloride (mmol/l)	49	51	91	87.0	17–50
Calcium (mg/dl)	–	8.2	–	–	8.4–9.7
Uric acid (mg/dl)	–	–	8.40	–	0.2–5.7
Total bilirrubin (mg/dl)	0.46	0.41	–	0.34	0.1–1.1
Lactacte dehydrogenase (U/l)	370	300	258	273	135–225
Aspartate aminotransferase (AST – U/l)	33	27	26	36	4–33
Alanine aminotransferase (ALT – U/l)	11	11	12	21	4–50
Gamma glutamyl transferase (GGT - U/l)	25	19	19	26	5–61
Alkaline phosphatase (U/l)	93	87	83	91	40–129
Total proteins (g/dl)	–	6.3	6.5	–	6.4–8.3
Albumin (g/dl)	3.3	–	3.1	–	3.4–4.8
Triglycerides (mg/dl)	98	88	100	–	<200
C reactive protein (CRP – mg/dl)	5.13	5.74	4.01	3.69	0–0.5
B 12 vitamin (pg/ml)	–	–	304.7	–	197–771
Folic acid (ng/ml)	–	–	1.9	–	4.6–18.7
Antinuclear antibodies (ANA)	–	<1/160	–	–	<1/160
B2-microglobulin (mg/l)	-	11.7	–	–	0.8–2.2
Immunoglobulin G	1640		–	2030	680–1450
Immunoglobulin A	–	389	–	427	83–407
Immunoglobulin M	–	115	–	131	34–214
Kappa chain	–	–	–	48	200–440
Lambda chain	–	–	–	344	110–240
Kappa/lambda relationship	–	–	–	1.4	1.35–2.70

Table 1. Analytic diagnostic workup and evolution

DOI: 10.12890/2018_000871

European Journal
of Case Reports in
Internal Medicine

DISCUSSION

The standard treatment for chylous ascites has not yet been established. However, the best results have been obtained by treating the underlying cause (in this specific case a lymphoma). For patients in whom the cause is not found or who do not respond to treatment of the underlying cause, nutritional therapy with a high-protein and low-fat diet with medium-chain triglycerides is recommended. Other approaches include frequent paracentesis, surgery (using sclerosing agents and suture ligation of the thoracic duct) or peritoneovenous shunting[3]. Our patient had concomitant portal hypertension, the underlying mechanism being pre-hepatic due to extrinsic compression of portal circulation. Although he responded favourably to diuretics, he died due to a nosocomial pneumonia. This case highlights the fact that although most cases of chylous ascites are related to trauma, some may be due to other causes such as lymphoproliferative diseases, pancreatitis[2] or retroperitoneal fibrosis[4].

REFERENCES

1. Almakdisi T, Massoud S, Makdisi G. Lymphomas and chylous ascites: review of the literature. *Oncologist* 2005;**10**:632–635.
2. Baban CK, Murphy M, O'Sulleabháin C, O'Hanlon D. Chylous ascites as a consequence of idiopathic pancreatitis. *BMJ Case Rep* 2014; doi:10.1136/bcr-2013-200132.
3. Al-Busafi SA, Ghali P, Deschênes M, Wong P. Chylous ascites: evaluation and management. *ISRN Hepatol* 2014;2014:240473.
4. Ur Rahman A, Ramaraj R, Humphreys E, Slowinska E. Retroperitoneal fibrosis as a cause of chylous ascites. *ACG Case Rep J* 2017;**4**:e115.