Successful conservative treatment of chylous ascites as rare intermediate complication after resection of an aboral esophageal carcinoma

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Background: Chylous ascites is an uncommon symptom that needs specific expertise with regard to the diagnostic and therapeutic management.

Methods: We are reporting on an uncommon and exceptional case of a 65-year-old man in whom postoperative chylous ascites was diagnosed using both daily ascites inspection and laboratory investigation. Treatment was initiated according to a previously established protocol, which summarized our own experiences and recommendations from the literature.

Results: The patient showed a prolonged postoperative course after partial esophagectomy of the aboral segment (2-cavity intervention) because of Barrett carcinoma (pT3N1M0G3R0), including lymphadenectomy and esophagogastric anastomosis, which was initially complicated by i) hemorrhage (d2), leading to relaparotomy, ii) prolonged weaning maneuver, and iii) suspicion of chylous ascites characterized by beige and milky effusion out of the abdominal drainage after appendicostoma placement for colonic decompression (d28). Diagnosis was confirmed by laboratory detection of chylomicrons and triglycerides of 4.8 mmol/L. After an observation period of two weeks, total parenteral nutrition (TPN) was initiated for 5 weeks. This resulted in the clearance of ascites (decrease of its amount partially after 5–10 d, completely after 20 d) and final removal of the abdominal drain. There were no further abnormal symptoms or signs.

Conclusions: The established therapeutic concept favoring conservative long-term TPN and avoiding reintervention, which was repeatedly used in rare cases of chylous ascites, is suitable to achieve spontaneous healing of lymphatic leakage by diminishing lymphatic flow due to decreased i) enteral fat absorption and ii) transport along the lymphatic vessels as shown in this exceptional case. To our best knowledge, this is only the sixth case with chylous ascites after esophagectomy reported in the literature.

Keywords: chylous ascites, total parenteral nutrition, esophageal carcinoma, postoperative complication

Introduction

Chylous ascites (Suarez Crespo et al 1995; Beal et al 1998; Seo et al 1999; Laterre et al 2000; Lord and Justin 2000; Rajasekar et al 2000; Aalami et al 2001; Kaas et al 2001; Leibovitch et al 2002; Huang et al 2004; Almakdisi et al 2005; Chan et al 2006; Jensen and Weiss 2006) (synonyma, chylascites [Hoffmann 1902; vam Turnhout et al 1997]; chylascos [Volnohradsky 1974; Beller 1988]; chyloperitoneum [Kostiainen et al 1983; Pomeranz et al 1984; Beller 1988; Földi and Földi 1991; Pabst et al 1993; Bauwens et al 1996; Tytgat et al 1996; Coluccio et al 1997; Muller and Lee 1998; Pui and Yueh 1998; Guillem et al 1999; Busch et al 2000; Kollmar et al 2000; Combe et al 2001]) is considered a rare and moderate to severe, spontaneously occuring postoperative complication. Chylous ascites can lead to a critical condition of the patient because of permanent loss of fluids, protein, fat, immunglobulins, and cells such as lymphocytes as well as possible dehydration, avitaminosis, immune suppression, cachexia, and even

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death (,Kostiainen et al 1983; Ruggiero and Caruso 1985; Savrin and High 1985; Villani and Japichino 1989; Földi and Földi 1991). It is defined as a filling of the abdominal cavity with chylum because of a leakage of the lymphatic vessels (Beal et al 1998), extending within the intra-or retroabdominal space due to – in the majority of cases – extensive intraoperative preparation (Savrin and High 1985; Kollmar et al 2000).

The aim was to report on our experiences in an uncommon and exceptional case, showing chylous ascites during its intermediate postoperative course after aboral esophagectomy and successful outcome after consequent first-line nonsurgical treatment during the complicated period of the patient's course at an intensive care unit.

Case report

A 65-year-old man was referred to our surgical department because of esophageal cancer at the aboral third. His major complaints were regurgitation, dyspepsia, and a weight loss of aproximately 8 kg. His medical history was otherwise unremarkable. Physical examination turned out to be normal. Laboratory parameters were within the normal range. Esophagogastroscopy had revealed a 1-cm esophageal ulcer and a circularly growing tumor lesion. Biopsies were taken and Barrett carcinoma was diagnosed with histologic investigation. Computed tomography (CT) scan revealed a tumor lesion limited to the esophageal wall. Therefore, the patient underwent a surgical approach for tumor resection (R0) by a 2-cavity-intervention consisting of esophagectomy of the aboral segment, lymphadenectomy, mobilization of the stomach into the thoracic cavity and intrathoracic esophagogastric anastomosis. Histopathologic investigation revealed an esophageal adenocarcinoma, which had developed on Barrett-like mucosa, with the following tumor staging (according to TNM classification by UICC 1997) and grading: pT3N1M0G3. Postoperatively, the intubated patient was referred under mechanical ventilation to the surgical intensive care unit. The immediate postinterventional course was complicated by a sudden intraabdominal bleeding indicated by hemorrhagic effusion out of the abdominal drains and confirmed by abdominal ultrasound on the second postoperative day. Therefore, the patient underwent explorative relaparotomy showing slight, but diffuse venous bleeding after removal of a hematoma from the left subphrenic space. The patient's condition was stabilized by the cessation of bleeding and a perioperative substitution of fluids, blood transfusion, and additional administration of fresh frozen plasma and catecholamines. Postoperative artificial respiration was required for 6 days followed by a second period later of 16 d due to respiratory insufficiency caused by diffuse pneumonia, including necessary tracheotomy. Antibiotic treatment was initiated and adapted according to microbiologic investigations of tracheobronchial secretions. Postoperative course was further complicated by recurrent "subileus", leading to repeated colonoscopy for decompression of the ascending colon and finally the placement of an appendicostoma. After a final 4-weeks postoperative interval from the first surgical intervention, the residual effusion out of the remaining intraabdominally placed drain (left subphrenic space) indicated beige, milky, nonclear fluid with increasing amounts from 100 to maximally 300 mL/d, leading to the suspicion of chylous ascites (Figure 1a). This finding was supported by the detection of fluid within the abdominal cavity using abdominal ultrasound. The laboratory investigation detected chylomicrons and a triglyceride content of 4.8 mmol/L, which confirmed the diagnosis. Microbiological investigation of the ascites did not detect any microbial growth. Since there was no change of the effusion fluid characteristics and amounts over an observational period of two weeks (oral administration of tea only), total parenteral nutrition was initiated. While partial clearance of the fluid was already seen after 5 days, complete clearance in addition to decreased amounts of drain effusion was achieved after 20 days (Figure 1b). Total parenteral nutrition was continued up to 5 weeks to save the desirable therapeutic effect. Stepwise initiation of oral nutrition as well as physical training and mobilization did not reveal any complications or complaints afterwards. This enabled us to refer the patient to a nonintensive care medical floor, final removal of the abdominal drain, and discharge.

After slightly more than one year, the patient was lost for further follow-up investigation with, at that time point, no hints for recurrent tumor growth.

Discussion

In daily practice, chylous ascites is an infrequent and initially not threatening event, but can become a critical condition after persistence over a longer period of time because of i) permanent loss of essential compounds (fat, proteins), metabolites, and cells (eg, lymphocytes) or ii) potentiating existing irregularities in the case of an additional complication. It is generateds by i) chylous transudation due to impeding chylous flow or ii) lymphatic leakage caused mainly by four different conditions as shown in Table 1 (Ruggiero and Caruso 1985; Beller 1988; Dougenis et al 1992; Dugue et al 1998; Alexiou et al 1998; Merigliano et al 2000; Motoyama et al 2005; Nadesan et al 2005). Pathological chylum collection

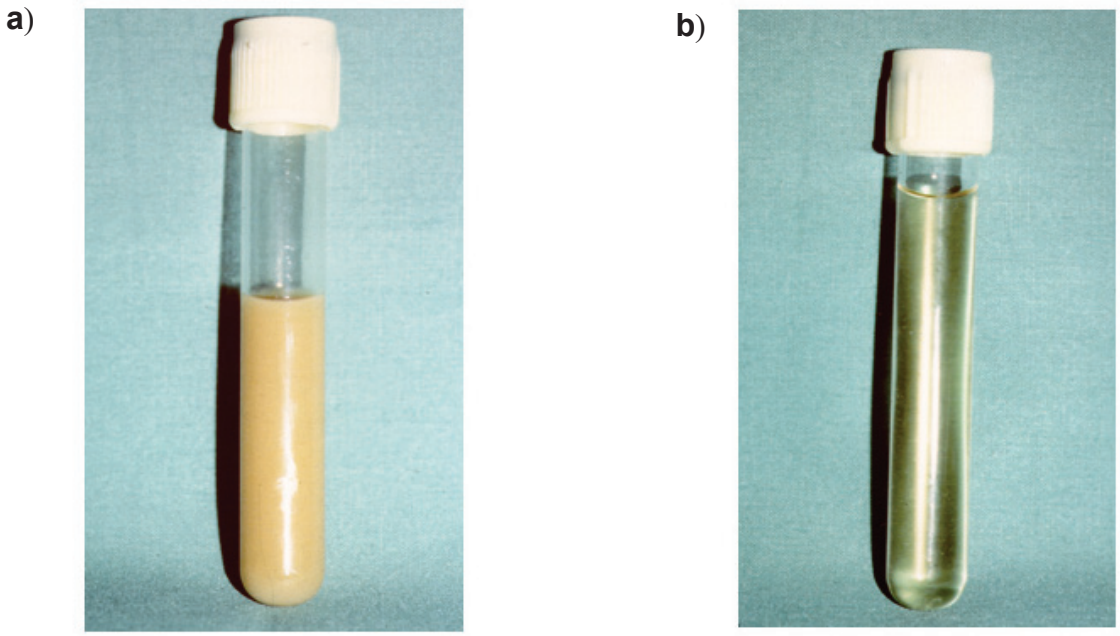

Figure 1 Chylous effusion fluid: **a**) Pre-treatment: Beige, milky, nonclear fluid, indicating chylum; **b**) Post-treatment: Cleared fluid, indicating nonfatty chylus (after 20 days of parenteral nutrition).

can also occur within other cavities such as the thorax (Cevese et al 1975; Gertsch and Mosimann 1983; Zerkowski et al 1983; Ruggiero and Caruso 1985; Fleisher et al 1987; Naber and Isringhaus 1988; Combe et al 1992; Duogenis et al 1992; Tytgat et al 1996; Alexiou et al 1998; Dugue et al 1998; Pui and Yueh 1998; Guillem et al 1999; Merigliano et al 2000; Binkert et al 2005; Motoyama et al 2005; Nadesan et al 2005), pericard (Hoffmann 1902; Kostiainen et al 1983), uterus (Hoffmann 1902; Földi and Földi 1991), or even in urine (Pabst et al 1993). Pathophysiologically, leakages are sustained by high lymphatic flow, in particular, within the abdominal cavity because of the need of fat absorption in the small bowel and its transport along the lymphatic vessels. Besides the visual aspects of chylum, appearing nonclear, beige, milky, sterile, viscous, and not smelling (Pabst et al 1993), elevated triglyceride concentration (Pomeranz et al 1984; Ruggiero and Caruso 1985; Naber and Isringhaus 1988), and laboratory detection of chylomicrons give appropriate evidence for chylum-containing fluids as shown in the presented case. Morphologically, it is presumed that the leakage was located at the single mesenteric trunk near the confluence with the hepatic branches, the descending thoracic branch, and the two lumbar trunks within the cysterna chyli. The most likely cause of chylous ascites in our case may have been a subsequent condition after surgical intervention and extensive preparations even after a postoperative symptom-free interval of approximately five weeks before the occurrence. Preexisting dilatations of the lymphatic vessels can not be fully excluded, but were not described intraoperatively. Also, impeding the lymphatic flow along the thoracic duct during the postoperative course does not appear to be very likely.

In the past, therapeutic recommendations from the literature had been partly confusing as well as contradictory, and had comprised a spectrum, spanning from observation (Bradham et al 1970; Zerkowski et al 1983; Beller 1988; Williams et al 1991) to immediate surgical intervention (Meinke et al 1979). Our previously published protocol (Halloul et al 1995) consists of the following options in subsequent order, giving reasonable step-by-step conceptual advice, which has been successfully used in single cases ever since (Table 2):

1. The aim of the observation period is to wait and see whether chylous ascites is a temporary symptom or to wait for spontaneous occlusion of the lymphatic leakage (Bradham et al 1970; Zerkowski et al 1983; Beller 1988; Williams et al 1991) due to opening of collaterals (Földi and Földi 1991); however, this is unlikely. If it is necessary, therapeutic paracentesis can be performed periodically to diminish the amount of ascites within the abdominal cavity.
2. Fat-poor diet, but still allowing fat-containing middle-chain fatty acids (C8–C12) (Suarez Crespo et al 1995) exclusively, circumvents fat reabsorption through the intestinal chylus barrier. Thus, fat content of the chylum and subsequently its flow are decreased, allowing the leakage of the lymphatic vessel to heal.

Table 1 Etiology of chylous ascites derived from Beller (1988) and Ruggiero and Caruso (1985)

Main causes	Subdivided causes
– Traumatic	Nonsurgical (Volnohradsky 1974):
(Beller 1988; Volnohradsky 1974; Beal et al 1998; Lord and Justin 2000)	*Rupture of:
	– Thoracic duct (Coluccio et al 1997)
	– Cysterna chyli (Beller 1988)
	– Mesenteric cysts (Pfeiffer et al 1981; Beller 1988)
	– Lymphocele (Seo et al 1999)
	Iatrogenic (Beller 1988):
	– Peritoneal dialysis (Pomeranz et al 1984; Beller 1988; Heyl and Veen 1989)
	– Post-radiation (Beller 1988)
	Surgical (Boyd et al 1989):
	– Post-cholecystectomy (Jensen and Weiss 2006)
	– Post-anterior resection for rectal cancer (Leibovitch et al 2002; Chan et al 2006)
	– Post-lymphadenectomy (Beller 1988; Naber and Isringhaus 1988)
	– Post-vagotomy (Beller 1988)
	– Post-aortic surgery (Bradham et al 1970; Meinke et al 1979; Lausten and Engell 1984; Jensen et al 1986; Sarazin and Sauter 1986; Fleisher et al 1987; Schein et al 1987; Teboul et al 1987; Williamson and Provan 1987; Boyd et al 1989; Heyl and Veen 1989; Villani and Japichino 1989; Ablan et al 1990; Sanger et al 1991; Williams et al 1991; Halloul et al 1995; Muller and Lees 1998; Busch et al 2000)
	– Post-colon/liver surgery (Ablan et al 1990)
	– Post-esophagectomy (Tytgat et al 1996; Cope 1998; Guillem et al 1999)
	– Post-gastrectomy (Rajasekar et al 2000)
	– Post-duodenopancreatectomy (Kollmar et al 2000)
	– Post-placement of mesocaval shunt (Ablan et al 1990)
– Nontraumatic	Tumor lesions/metastases (Volnohradsky 1974; Ruggiero and Caruso 1985; Beller 1988; Almakdisi et al 2005)
(Ruggiero and Caruso 1985)	Mesenterial lymphadenitis (Volnohradsky 1974; Beller 1988)
	Peritonitis (Volonohradsky 1974)
	Pancreatitis (Beller 1988)
	Aortic aneurysm (Ruggiero and Caruso 1985)

Hodgkin's disease (van Tunrhout et al 1997)
Postinflammatory generation of cords (Hoffmann 1902)
Thrombotic occlusion of the left venous arch (Hoffmann 1902)
Liver cirrhosis (Volnohradsky 1974; Ruggiero and Caruso 1985; Földi and Földi 1991)
Tuberculosis (Volnohradsky 1974; Ruggiero and Caruso 1985; Beller 1988)
Bilharciosis (Volnohradsky 1974)
Filariasis (Volnohradsky 1974; Beller 1988)
Stones (Volnohradsky 1974)
Torsion of the mesenteric radix (Beller 1988)
"Incarcerated" hernia (Beller 1988)
Ductus atresia/aplasia
– Congenital
Lymphangiectasia (Ruggiero and Caruso 1985)
(Hoffmann 1902; Ruggiero and Caruso 1985; Beller 1988; Földi and Földi 1991; D'Agostino et al 1998)
Lymphangiomyomatosis (Kanbe et al 1987; Guillem et al 1999)
Malrotation (Hoffmann 1902)
– Ideopathic
(Kostiainen et al 1983; Beller 1988)

3. Total parenteral nutrition blocks any intestinal reabsorption completely, all showing more potential for leakage healing than a fat-poor diet.
4. Surgical intervention is considered the *ultima ratio* if the previous options fail. This approach is comprised of reexploration and ligation of the leakage, which can include i) use of glue, but showing relatively poor outcome (Naber and Isringhaus 1988), and ii) insertion of a peritoneovenous shunt (Hoffmann 1902; Kanbe et al 1987; Schumpelick and Riesner 1993; Gupta et al 2004).

In our case, chylous ascites was diagnosed in an appropriate manner by the detection of chylomicrons and an elevated content of triglycerides – though lymphoscintigraphy is also suggested (Pui and Yueh 1998; Seo et al 1999), but not always suitable (Cope 1998) – during the patient's reconvalescence period after long-term artificial respiration. It further impaired the continuing nonstable condition of the patient due to repeated temporary subileus.

Obviously, there must have been a leakage of the intraperitoneal branches of the abdominal part of the lymphatic trunk because of the intraperitoneally located tip of the abdominal drain, which drained the chylous fluid (resembling periodical therapeutic paracentesis listed in the treatment protocol above). The short-term observation period (including laboratory and microbiological analysis of chylum) after finding chylum-like effusion did not result in cessation due to spontaneous occlusion of the lymphatic leakage. Therefore, total parenteral nutrition was initiated (option 3 of the therapeutic concept), leaving out fat-free diet period (option 2) since the patient had not reached full enteral nutrition status during the postoperative course at that time point. Already five days after initiation of total parenteral nutrition, distinct clearance of the chylum was observed, with complete clearance by day 20. For safety reasons, a full 5-week period was accomplished, showing no relapse during follow-up. The parenteral nutrition period was associated with monitoring and substituting electrolytes, minerals, and vitamins in addition to proteins, fat and carbohydrates. Maintenance of psychological motivation in the otherwise further stabilizing condition of the patient and long-term cost-intensive nursing period were two of the major problems.

To our best knowledge, our report is the sixth of such complications following radical esophagectomy. Rajasekar and colleagues (2000) have reported on a similar case and successful therapeutic approach after gastrectomy while Guillem's group (Guillem et al 1999) has described three patients and Bauwens and colleagues (1996); Tytgat and colleagues (1996); and Cope (1998) described one subject each, respectively, with "chyloperitoneum" post-esophagectomy. Interestingly,

Table 2 Stepwise treatment concept for chylous ascites according to Halloul and colleagues (1995)

Subsequent treatment steps	Period
1. Observation and/or periodical therapeutic paracentesis	2 weeks
2. Fat-poor diet (including middle chain fatty acids [Suarez Crespo et al 1995])	3–4 weeks
3. Total parenteral nutrition	5–6 weeks
4. Surgical intervention:	Later period
a) Ligation of the fistula	
c/o covering the leakage	
b) Fibrin glue (Zerkowski et al 1983)	
c) Peritoneovenous shunt according to the procedure by LeVeen (Schumpelick and Riesner 1993)	

Bauwens and colleagues (1996) did not achieve a decrease of the chylous volume by conservative treatment and, therefore, favored surgical ligation. Guillem and colleagues (1999) treated their patients with etilefrine infusion.

In conclusion, the patient tolerated the therapeutic concept well, which is favored by healing of the lymphatic leakage through a noninterventional treatment option despite the intermediate duration of total parenteral nutrition but with a lower morbidity and mortality risk as well as a better prognosis for successful outcome than with a surgical approach as reported by others (Busch et al 2000; Kollmar et al 2000; Rajasekar et al 2000). As alternative treatment options, percutaneous transabdominal catheterization of the cysterna chyli has been suggested to identify and possibly embolize chylous fistula (Cope 1998; Binkert et al 2005). In addition, intravenous or subcutaneous application of the somatostatin analog (Octreotide) can be considered a promising further (conservative) therapeutic option. In particular, the combination with middle chain triglycerides or total parenteral nutrition can lead to therapeutic success within short-term intervals. The basic mechanisms seem to be reduced gastrointestinal motility, a reduction of blood and lymphatic flow within the intestinal region, as well as a reduced chylum production (Jensen and Weiss 2006). Based on that, several authors recommend somatostatin alone or in combination already at early time points of the patient's course of chylascites (Leibovitch et al 2002; Jensen and Weiss 2006).

Disclosure

The authors report no conflicts of interest in this work.

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