

# Case Report

## Congenital lymphangiectasia and chyloperitoneum in a foal

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### Introduction

Chyloperitoneum is an uncommon cause of colic and abdominal effusion in neonatal foals. Congenital lymphangiectasia results in the engorgement of intestinal lymphatic vessels, and subsequent rupture results in the leakage of triglyceride- and cholesterol-rich chyle into the abdominal cavity (Melzer and Sellon 2002). Clinical signs of chyloperitoneum include abdominal discomfort, protein-losing enteropathy and electrolyte disturbances.

**This report describes the clinical findings, case progression, and post mortem findings associated with chyloperitoneum in a neonatal colt secondary to congenital lymphangiectasia.**

### Case details and history

An 18 h old Quarter Horse colt was referred for evaluation of colic of 11 h duration. The foal's birth was unattended, but no complications were reported.

### Clinical and clinicopathological findings

On admission, the colt was bright, alert and responsive. Temperature, pulse and respiratory rate were within normal limits. A mild bilateral carpal flexural deformity was present. Gastrointestinal motility was decreased in all quadrants, and there was a small amount of diarrhoea on the foal's perineum and tail. A 3 cm umbilical hernia was present.

The foal's IgG level was greater than 8 g/l. A complete blood count revealed leucopenia ( $2.1 \times 10^9$  cells/l), hyperfibrinogenaemia (6.0 g/l), PCV of 44% and hypoproteinaemia (44 g/l). Serum chemistry values were within normal limits with the exception of hypoalbuminaemia (18.4 g/l). Results of a venous blood gas analysis were within normal limits.

Ultrasonography of the colt's abdomen was performed using a ventral approach (**Fig 1**). There was a moderate amount of anechoic fluid within the peritoneal cavity with gastrointestinal viscera floating in the fluid. The visible small intestinal sections were markedly thickened and oedematous, but there was no luminal distention.

### Treatment and outcome

Based upon the ultrasonographic findings, abdominocentesis was attempted. Although not shown in **Figure 1**, the urinary bladder and urachus appeared intact on ultrasonographic examination. The initial abdominocentesis attempt was unsuccessful due to omental interference and mild omental prolapse from the centesis site with removal of the teat cannula. Abdominal exploration was declined. An indwelling i.v. catheter was placed. Intravenous crystalloids, hyperimmune plasma (1 l), ceftiofur sodium (2.2 mg/kg bwt i.v. q. 12 h), amikacin (20 mg/kg bwt i.v. q. 24 h) and ranitidine (6.6 mg/kg bwt *per os* q. 8 h) were administered overnight. The colt did



**Fig 1:** Ventral abdominal ultrasonography of an 18 h old Quarter Horse colt referred for evaluation of colic of 11 h duration. Multiple loops of thickened small intestine and associated mesentery are visible surrounded by anechoic peritoneal fluid.

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not exhibit signs of colic during that time, and nursed and urinated normally. Mild diarrhoea was observed.

The following morning, the foal's albumin level had decreased further, despite plasma transfusion, to 17.2 g/l. Ultrasonography revealed increased abdominal effusion and mural thickening of the affected small intestine (**Fig 2**). Abdominocentesis at this time yielded a copious amount of orange-yellow, markedly opaque fluid. The fluid did not separate with centrifugation. Chyloperitoneum was suspected, and the fluid submitted for determination of triglyceride and cholesterol levels. Systemic therapy was continued and a second unit of hyperimmune plasma administered.

The foal began to demonstrate intermittent signs of colic and depression, and the albumin level continued to decrease to 13.3 g/l after administration of the second unit of plasma. The triglyceride and cholesterol levels of the abdominal fluid were 4.04 g/l (4.6 mmol/l) and 0.91 g/l (2.4 mmol/l), respectively, confirming the diagnosis of chyloperitoneum. The foal was subjected to euthanasia and necropsy performed.



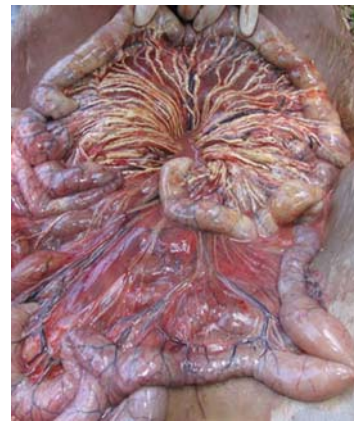
**Fig 2:** Ventral abdominal ultrasonography performed on Day 2 of hospitalisation. Note the increased mural thickness of the affected intestine (red arrows) and increased echogenicity of the effusion. The mesentery appears more prominent and thickened compared to the previous examination.

### Post mortem findings

Necropsy examination revealed approximately 80% of the proximal and mid-jejunum was markedly congested, oedematous, and dark red in colour. No anatomic abnormalities (volvulus, entrapment etc.) were identified. Approximately 2 l of opaque fluid, identical in appearance to that obtained on abdominocentesis, were present in the abdominal cavity. The mesenteric lymphatics of the affected intestine were severely congested and tortuous, with numerous focal rupture sites (**Figs 3** and **4**). Histopathology of the affected intestine revealed acute, diffuse, severe lymphangitis with intestinal oedema and lymphangiectasia of the mesentery.

### Discussion

Ingested triglycerides are absorbed and bound to chylomicra in the small intestine. The chylomicra enter the central lacteal



**Fig 3:** Necropsy photograph. The affected, proximal small intestine is at the top of the image and the unaffected, distal intestine is at the bottom of the image. The foal's head is directed toward the top of the image.



**Fig 4:** Necropsy photograph. Note the severely congested and tortuous mesenteric lymphatics with numerous focal rupture sites leaking chylous fluid (black arrows). The foal's head is directed toward the top of the image.

of the intestinal villi and then pass through the afferent lymphatics to the mesenteric lymph node. From the lymph node, they pass through the efferent lymphatics to the *cisterna chyli*, then through the aortic hiatus within the thoracic duct. The contents of the thoracic duct enter the bloodstream via the subclavian vein and cranial vena cava (Melzer and Sellon 2002).

The term lymphangiectasia refers to dilation of the lymph vessels within the intestine (Melzer and Sellon 2002). If the lacteals in the intestinal villi become engorged with lymph, there can be a resultant loss of lymph into the intestinal lumen as they rupture (Melzer and Sellon 2002). If the mesenteric lymphatics or *cisterna chyli* rupture, chyle is lost into the abdominal cavity. Rupture of the thoracic duct results in chylothorax.

Disruption of lymphatic flow results in loss of proteins, fats, fat-soluble vitamins, sodium, potassium and chloride into the body cavity or into the lumen of the gastrointestinal tract; therefore, these substrates are not available for maintenance of normal metabolism and homeostasis (Melzer and Sellon 2002). Intestinal lymphangiectasia is one cause of protein-losing enteropathy in dogs, and the severity of hypoalbuminaemia may be indicative of the severity of the disease (Melzer and Sellon 2002). In addition, chyle is irritating to the pleura and peritoneum and contains increased amounts of fibrinogen; this favours adhesion formation (Meadows and MacWilliams 1994).

Causes of lymphatic obstruction or disruption include trauma, increased intrathoracic pressure, congenital abnormalities (aplasia), neoplasia, fungal disease, adhesions, cirrhosis, lung lobe torsion, increased central venous pressure, heart disease and iatrogenic damage during surgery (Melzer and Sellon 2002; Mair 2002). Chyloperitoneum in foals has been reported secondary to intra-abdominal abscessation, ileus and congenital abnormalities (Hanselaer 1983; Edwards *et al.* 1994; Campbell-Beggs *et al.* 1995; Mair 2002).

Chylous fluid resembles markedly purulent exudates, but does not separate with centrifugation. The addition of ether results in clearing of chylous effusions (Meadows and

MacWilliams 1994). Triglyceride levels exceeding 1.1 g/l (1.2 mmol/l) and a abdominal fluid:serum triglyceride ratio greater than 12:1 are confirmatory of chylous effusion (Fossum *et al.* 1986). Additionally, a cholesterol to triglyceride ratio of less than 1:1 is indicative of chylous effusion (Meadows and MacWilliams 1994). The triglyceride:cholesterol ratio of the fluid obtained in this case was 0.22:1.

Treatment of chylous effusions involves correction of the primary problem and metabolic and dietary support (Melzer and Sellon 2002). Drainage of chylous fluid is vital, but palliative. Reducing fat intake is also important in reducing the volume of chyle produced (Melzer and Sellon 2002). Several foals with chyloperitoneum recovered successfully following surgical resection of the affected segments of intestine (Edwards *et al.* 1994). However, in the case reported here, the length of affected intestine exceeded the maximum amount that could be safely resected.

## References

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