Chronic Weight loss and Acute-Onset “Choke” in Two Related Friesian Stallions

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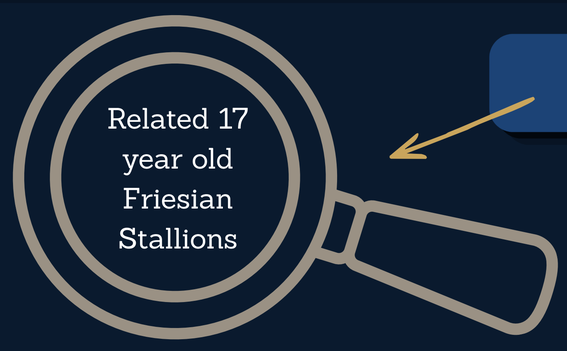
BACKGROUND

A 17-year-old Friesian stallion presented for investigation of an 11-month history of chronic weight loss and an acute “choke” episode. Given the recognised predisposition of Friesian horses to neuromuscular and connective tissue disorders, an underlying systemic cause of acquired megaoesophagus was considered.

A second 17-year-old Friesian stallion from the same property was evaluated concurrently for chronic weight loss and intermittent colic signs, raising suspicion of a shared or potentially heritable condition.



CASE 1

BCS 1.5/5

Hypermetric gait (Hindlimbs)

Marked muscle atrophy

“Choke” episode (12hour prior to arrival)

CASE 2

BCS 3.5/5

Normal gait

Mild Muscle Atrophy

No known “choke” episodes

DIFFERENTIAL DIAGNOSES

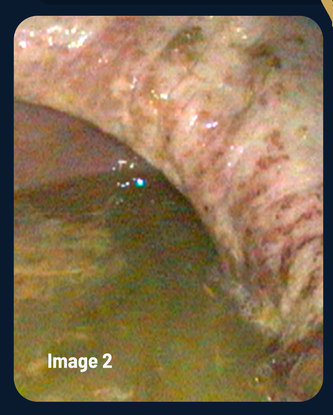
Mechanical obstruction (stricture, diverticulum, intraluminal mass)
Functional disease (megaoesophagus, neuromuscular dysphagia)
Hiatal hernia

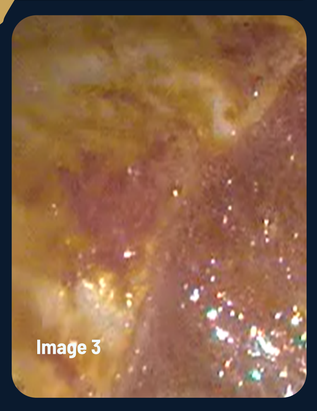
DIAGNOSTICS

Gastroscopy
- Diffuse oesophageal dilation with fluid pooling (Image 4)
- No obstruction or stricture
- Grade 4 squamous ulceration

Contrast oesophagram (unsedated)
- Persistent contrast retention at C6–C7 (Image 1)
- Functional oesophageal stasis with marked cervical dilation

Gastroscopy
- Grade 4 squamous ulceration (image 2 and 3)





TREATMENT

Dietary modification:
- Feed above shoulder height
- 2.5 kg Multavite® BID (gum nuts)
- Avoid hay and excessive feed soaking
- Omeprazole + sucralfate for 30 days

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- Progressive weight loss and aspiration pneumonia
- Gastroscopy (image 5) and contrast oesophagram confirmed megaoesophagus
- Treated with antibiotics, anti-inflammatories and dietary modification
- **Outcome**: Died from colic 2 months after diagnosis.

6-MONTH FOLLOW-UP

- Weight regained (BCS 4/5)
- No recurrent “choke” episodes
- Successfully managed with dietary modification

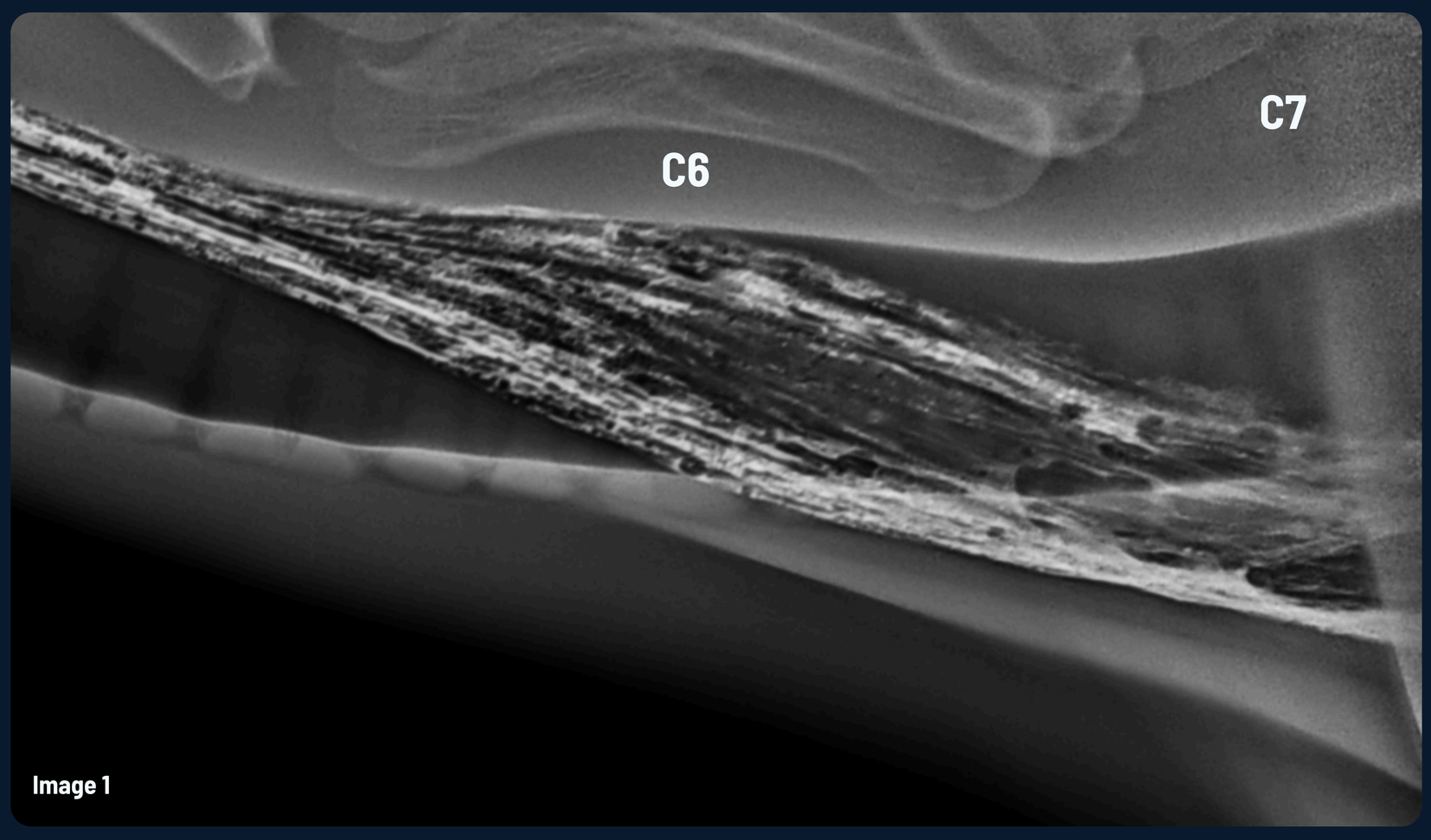

Image 1

DISCUSSION

These cases support a diagnosis of diffuse megaoesophagus secondary to a functional oesophageal motility disorder in two related Friesian stallions. Friesians are predisposed to neuromuscular and connective tissue disorders affecting the oesophagus, and the occurrence in related male horses further supports a breed-associated, potentially heritable condition.[1,3,5] Komine et al. (2014) proposed a possible X-linked inheritance pattern.[2]

The cases likely represent different stages of disease. Case 1 demonstrated advanced megaoesophagus with “choke” and marked oesophageal dilation, whereas Case 2 exhibited chronic weight loss consistent with early oesophageal dysmotility despite the absence of gross dilation.

Both horses were 17 years old, suggesting clinically significant disease may also present later in life than previously reported.[1,3] Ploeg et al. (2015) similarly reported that severe oesophageal dysfunction may occur without gross dilation.[3]

A recent videofluoroscopic study demonstrated transient thoracic inlet contrast retention in normal horses following xylazine and anticholinergic administration.[6] In contrast, persistent cervical oesophageal dilation centred at C6 was identified in the unsedated horse, supporting true cervical megaoesophagus rather than physiological or sedation-associated stasis.

CLINICAL RELEVANCE

Chronic weight loss, recurrent “choke”, or subtle neuromuscular abnormalities in Friesian horses should prompt early oesophageal evaluation for megaoesophagus.
Contrast oesophagram was diagnostic in both cases, demonstrating persistent cervical oesophageal dilation centred at C6.
Clinically significant disease may occur in older horses and may precede marked oesophageal dilation. Given the suspected heritable basis, evaluation of related horses should be considered.

Prognosis is variable. One horse was successfully managed with dietary modification, whereas long-term outcome in the second horse could not be assessed following death from colic two months after diagnosis.

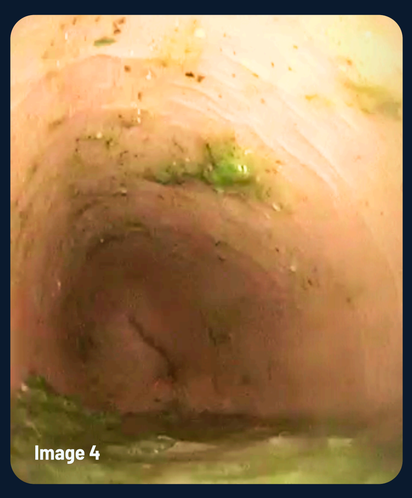

Image 4

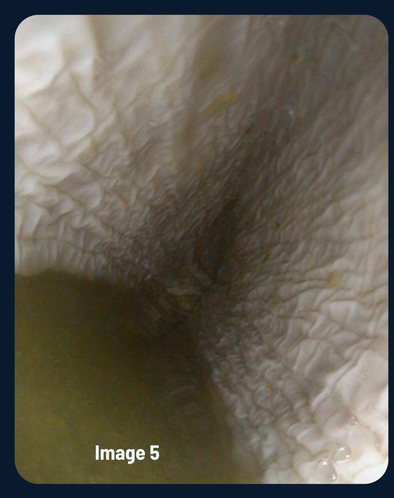

Image 5

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