

Special Article

Yellow fat disease in equids

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Introduction

In 1957, Hartley and Dodd identified a disease in foals in New Zealand, characterised by muscular dystrophy and inflammation of the adipose tissue. In The Netherlands, similar findings in the adipose tissue of 15 Shetland pony foals were described by Kroneman and Wensvoort (1968). This disease, called yellow fat disease or steatitis, is a generalised disorder of fat depots, characterised by extensive adipose cell degeneration and inflammation of adipose tissue. During these processes, a progressive peroxidation of unsaturated fatty acids may occur. Lipofuscin pigment, which is responsible for the typical yellow discolouration of the adipose tissue in diseased animals, is the final product of this peroxidation process (Danse and Steenbergen-Botterweg 1974).

Steatitis is observed in equids (Kroneman and Wensvoort 1968; Platt and Whitwell 1971; Glyn 1972; Peyton *et al.* 1981; Hamir 1982; Foreman *et al.* 1986; Taylor *et al.* 1988), pigs (van de Kerk and Danse 1973), cats, mink (Mason and Hartsough 1951) and many other animals. Two important aetiopathological factors, more or less dependent on each other, seem to be involved in yellow fat disease. First, there is the relatively high intake of polyunsaturated fatty acids, thereby increasing their concentration in adipose tissue and other organs of monogastric species (van de Kerk and Danse 1973). Second, oxidation of these acids may occur when the tissue concentration of biological antioxidants, such as vitamin E, is insufficient (Danse and Steenbergen-Botterweg 1974). Therefore, the disease is considered to be an expression of vitamin E deficiency. However, the aetiopathogenesis must be more complex than can be explained by vitamin E deficiency in animals that have a relatively high content of certain polyunsaturated fatty acids. In the horse, vitamin E deficiency is thought to be involved in a number of different clinical syndromes without changes in the adipose tissue, e.g. equine degenerative myelopathy (EDM) (Blythe *et al.* 1991), equine motor neuron disease (EMND) (Divers *et al.* 1997) and white muscle disease (Perkins *et al.* 1998). Moreover, generalised steatitis has been reported in a horse with normal serum vitamin E concentration (Foreman *et al.* 1986).

Depending on the localisation of the fat deposits involved, different clinical signs may be present. In cases in which the subcutaneous fat deposits are altered into hard and painful swellings, the clinical suspicion of steatitis soon arises. Histopathology of a biopsy of the nuchal ligament or from subcutaneous fat in the groin or axillary regions confirms the diagnosis of steatitis. However, in cases in which predominantly abdominal fat is involved, clinical signs may be nonspecific, hampering an early diagnosis.

In this report, the clinical and pathological findings of 14 equids with yellow fat disease are described, in order to clarify when this condition should be included in the differential diagnosis of equine abdominal disease.

Case selection

In the period 1996 to 2002, 14 equids were diagnosed as suffering from steatitis at the Department of Equine Sciences, Utrecht University. Age, breed, gender and clinical signs are listed in **Table 1**. Breeds included Shetland ponies (n = 6), Belgian draught horses (n = 2), Quarter Horse (n = 1), Arabian horse (n = 1), Haflinger pony (n = 1), Welsh pony (n = 1), Friesian foal (n = 1) and donkey foal (n = 1). Ages were 1.5–3.5 months (n = 6), 1–3 years (n = 6) and 8–21 years (n = 2). The cases were referred for one or more of the following clinical disorders: anorexia, colic, fever (that did not respond to antimicrobial therapy) and/or diarrhoea. The Haflinger pony (*Case 6*) and Friesian foal (*Case 14*) were referred for investigation of indurated subcutaneous swellings and a stiff gait. The Haflinger pony had been treated with vedaprofen, dexamethasone and vitamin E and selenium supplementation without success.

Case details

Clinical findings

Twelve cases were found to be more or less depressed and 9 of these were noticeably anorexic. Eleven cases had an abnormal faecal consistency, either diarrhoea or dry and firm

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TABLE 1: Subject details and clinical signs of 14 cases with generalised steatitis

Details/signs	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Breed	Shetland pony	Mini Shetland pony	Shetland pony	Mini Shetland pony	Quarter Horse	Haflinger pony	Belgian draught horse
Gender	♀	♂	♀	♀	♂	♀	♀
Age	3 months	3 months	8 years	3.5 months	1 year	3 years	2 years
Anorexia	+	ND	+	+	-	+	+
Depressed	+	+	+	+	+/-	+	+
Dull haircoat	+	ND	+	+	-	-	+/-
Nutritional status	Poor	Poor	Obese	Normal	Normal	Normal	Moderate
Pulse rate (beats/min)	100	80–100	120	96	80	48	82
Respiratory rate (breaths/min)	+	+	ND	+	-	-	+
Rectal temperature (°C)	39.0	40.2	39.8	39.7	38.9	39.0	38.8
Subcutaneous oedema	ND	ND	+	+	-	+	+
Induration of nuchal ligament/subcutis	+	-	-	-	-	+	+
Faecal consistency	Diarrhoea	Diarrhoea	Dry	-	Dry	Normal	Soft
Abdominal pain	-	-	-	+	+	-	-
Abdominal tenderness	-	ND	ND	ND	+	-	-
Rectal examination	NP	NP	NP	NP	Painful swellings mesenteric artery region	ND	Painful swellings splenic region
Abdominocentesis	ND	ND	ND	Increased, slightly orange WBC 0.2 x 10 ⁹ /l	Small amount, dark yellow	ND	ND
Details/signs	Case 8	Case 9	Case 10	Case 11	Case 12	Case 13	Case 14
Breed	Belgian draught horse	Arabian	Donkey	Shetland pony	Shetland pony	Welsh pony	Friesian horse
Gender	♀	♂	♀	♂	♀	♀	♂
Age	2 years	2 years	1.5 months	3 years	3.5 months	21 years	1 month
Anorexia	ND	+	+	-	+	+	-
Depressed	+	ND	+	+	+	+	+
Dull haircoat	ND	-	+	ND	+	ND	+
Nutritional status	Obese	Moderate	ND	Poor	Normal	Obese	Normal
Pulse rate (beats/min)	108	60	108	120	116	80	100
Respiratory rate (breaths/min)	24	12	68	36	60	48	48
Rectal temperature (°C)	38.6	38.3	38.8	38.8	38.4	39.3	38.9
Subcutaneous oedema	+	+	+	+	+	-	+
Induration of nuchal ligament/subcutis	ND	-	ND	+	ND	-	+
Faecal consistency	Diarrhoea	Dry	Dry	Diarrhoea	Dry	Dry	ND
Abdominal pain	+	+	+	+	+	-	-
Abdominal tenderness	ND	ND	+	ND	+	+	-
Rectal examination	NP	Stricture present	NP	NP	NP	Painful swellings, mesenteric artery region	NP
Abdominocentesis	ND	ND	Whiteish fluid WBC 1.8 x 10 ⁹ /l	ND	ND	Increased amount, haemorrhagic	ND

+ = present; - = absent; NP = not possible; ND = not determined.

faeces, sometimes covered with fibrinous material. Nutritional status varied between obese (*Cases 3, 8 and 13*), moderate to normal (*Cases 4–7, 9, 12 and 14*) and poor (*Cases 1, 2 and 11*). On general examination, consistent clinical findings were fever (mean \pm s.d. $39.0 \pm 0.5^\circ\text{C}$) a markedly increased heart rate (mean \pm s.d. 93 ± 22 beats/min) and, often, subcutaneous oedema ($n = 11$). Painful and indurated swelling of the subcutaneous tissues, predominantly in the nuchal ligament (**Fig 1**), groin and axillary regions, were noted in 5 cases. Seven cases showed signs of mild to moderate abdominal discomfort, and marked abdominal tenderness was present in 4 cases. Most subjects were too small for rectal examination; however rectal examination was

performed in *Cases 5, 7, 9, and 13* and painful swellings were palpated in the region of the cranial mesenteric artery or in the splenic region. In *Case 9*, a circular narrowing of the rectum was found. Faecal impaction of the descending (small) colon was identified cranial to this stricture (**Table 1**).

Haematology and serum biochemistry

Haematology and serum biochemistry of peripheral blood samples are presented in **Table 2**. A moderate to severe anaemia was present in 8 cases, leucocytosis in 7 cases and leucopenia in 3 cases. Metabolic acidosis was found in 7 of the 11 cases in which blood gases were measured.



Fig 1: A Shetland pony foal (Case 1) with painful swelling of the adipose tissue surrounding the nuchal ligament.

Hypoproteinaemia was present in 6 cases and hypoalbuminaemia in 5 of 9 cases in which total protein was determined and serum electrophoresis performed. Mean \pm s.d. serum enzyme activity of the 10 cases in which the enzyme activities of LDH and AST were determined was markedly increased at 3938 ± 2085 iu/l for LDH (reference range 150–420 iu/l) and 1218 ± 1290 iu/l for AST (reference range 10–275 iu/l), respectively. Moderately increased CK (549 ± 305 iu/l; reference range 5–160 iu/l) was found in 5 cases (Case 11, CK = 7736 iu/l). Decreased serum vitamin E concentrations were found in Cases 6, 7, 11 and 14; the serum vitamin E concentration of the dam of the Friesian foal (Case 14) was also abnormally low (1.2 μ g/ml). A decreased serum selenium concentration was found in Case 7 and a normal selenium concentration in Case 6. Vitamin E and selenium concentrations were not determined in any of the other patients.

Other examinations

Faecal examination was performed in Cases 5, 6, 7, 10, 11 and 12. Strongyle-type eggs were found in the faeces of Cases 5 and 6 and *Parascaris*-type eggs in Case 12. Urine analysis revealed proteinuria in Cases 5 and 11.

Radiographic examination of the thorax was performed in Case 7, after finding muffled lung sounds in the ventral thoracic region and increased lung sounds in the dorsal thorax on auscultation. Slight consolidation in the ventral portion of the lungs was seen on the radiograph. Ultrasonography revealed some fluid in the pleural cavity in which an echodense soft tissue mass was seen, together with some fibrin-like strands. The fluid obtained by thoracocentesis revealed no cytological abnormalities.

Diagnosis

In 5 cases (Cases 1, 6, 7, 11 and 14), a diagnosis of yellow fat disease was made *antemortem*, based on the presence of painful indurated subcutaneous swellings. In 2 of the cases, (Cases 6 and 11), the tentative diagnosis was confirmed by histopathology of a biopsy of the swellings. In Case 4, an



Fig 2: The same Shetland pony foal as in Figure 1 at necropsy. Note the yellow discoloration of the thickened nuchal ligament and surroundings.



Fig 3: Inflammation of adipose tissue in the ligamentum intercolicum.

exploratory laparotomy was performed to identify the cause of colic. Widespread discoloration and induration of the mesenteric fat were the only abnormalities found. In all other cases, the final diagnosis of steatitis was made at necropsy. Some of these patients received intensive treatment with analgesics, antibiotics and i.v. fluids for several days. Either no definite clinical diagnosis could be made or patients were suspected to suffer from other disease processes, such as ascarid obstruction (Case 12), severe arteritis of the mesenteric artery caused by strongyle larvae (Case 5) or pathology of the soft tissues in the abdomen of unknown origin (Cases 9 and 13). Most of the patients had to be subjected to euthanasia due to lack of response to treatment (Cases 1, 3–7, 10, 11 and 14). Cases 2 and 13 were already in a state of shock by the time of referral, while Cases 8 and 12 died shortly after arrival. Case 9 was subjected to euthanasia because of recurrent colic, caused by rectal stricture.

Post mortem examination

Macroscopic findings

Macroscopic examination was performed in all cases. Most showed extensive hardened, multinodular, focally haemorrhagic fat tissues with yellow-brown discoloration and an opaque

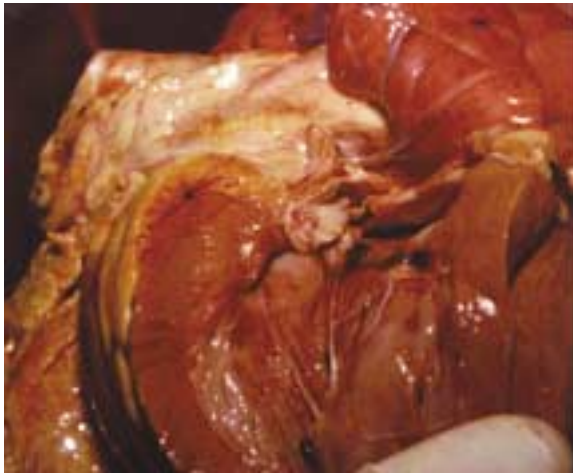


Fig 4: Adipose tissue in the coronary sulci of a horse with generalised steatitis. The hardened pericardial and mediastinal fat showed a yellow-brown discolouration with multinodular haemorrhagic foci.

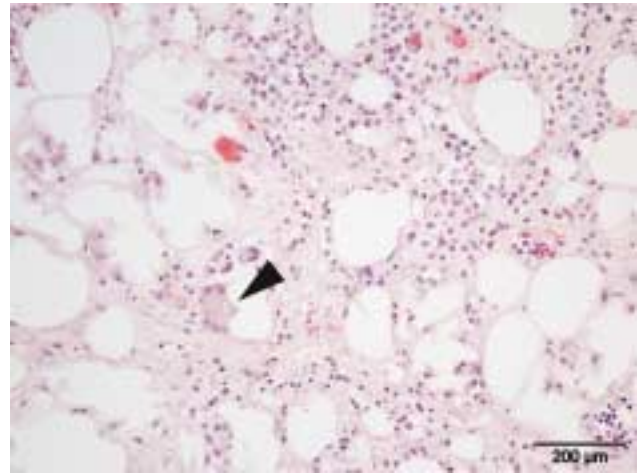


Fig 6: Steatitis, degeneration and necrosis of adipose tissue with a mixed inflammatory infiltrate with abundant numbers of macrophages and a Langhans-type syncytial giant cell (arrowhead). H&E staining.



Fig 5: Close-up of the ventral abdominal wall with peritoneum, showing steatitis. Note the irregular appearance of the fat tissue under the peritoneum.

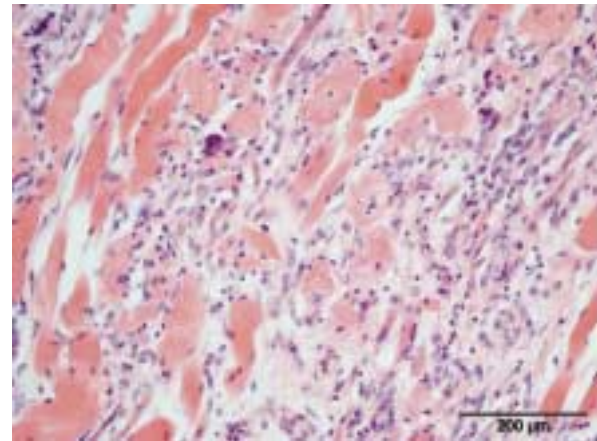


Fig 7: Myositis, degeneration and fragmentation of muscle fibres with lymphohistiocytic infiltrations and moderate fibrosis. H&E staining.

appearance of the cut surface. This pansteatitis was noted in Cases 1, 2, 4, 5, 7, 8 and 10–14. In Case 3, altered adipose tissue was found solely in the mesenteric fat, whereas in Case 6 the subcutaneous tissues were primarily involved. In Case 9, local fat necrosis in the rectal submucosa caused a stenotic stricture. The most affected areas involved varying amounts of fat in the nuchal ligament and surroundings (Fig 2), intra-abdominal regions (Fig 3), perirenal and retroperitoneal regions, mediastinum, coronary sulci (Fig 4) and subcutaneous tissues. Other frequent findings were the presence of subcutaneous oedema of the ventral abdominal wall (Fig 5) and, less frequently, of the lower parts of the hindlimbs, hydrothorax and hydroabdomen. In Cases 1, 8, 10 and 14, the skeletal muscles showed generalised discolouration and a striated appearance. The myocardium showed similar, although less severe, macroscopic abnormalities in Cases 2, 9, 10 and 13). Additional pathological findings were cyathostomiasis (Cases 7 and 8), chronic enteritis (Cases 12 and 13) and a necropurulent

bronchopneumonia (Case 14). Bacterial examination of the liquid intestinal contents of the cadavers of Cases 2, 7, 10, 11 and 14 showed no overgrowth of enteric pathogens.

Microscopic findings

Histopathology performed in 12 of the 14 cases revealed severe degeneration and necrosis of fat cells. These necrotic cells and tissues were surrounded by macrophages and histiocytic syncytial giant cells often laden with light brown globular lipopigments, accompanied by infiltrations of various amounts of lymphocytes, plasmacytes and neutrophilic and eosinophilic granulocytes (Fig 6). In more advanced stages, in between and surrounding necrotic dystrophically calcified fat tissues, there were broad hypercellular bands of fibrohistiocytic proliferations. The affected muscular tissues also showed degenerative changes and necrosis ranging from minor hydropic degeneration to fragmentation and dystrophic

TABLE 2: Haematology and serum biochemistry of 14 cases with generalised steatitis

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Normal value
Haemoglobin	7.4	5.9	10.4	5.2	7.2	6.7	8.7	7.5–9.0 mmol/l
PCV	0.32	0.24	0.48	0.23	0.39	0.31	0.37	0.36–0.42 l/l
Leucocytes	10.9	14.1	13.1	2.9	3.0	14.4	25.5	7.0–10.0 x 10 ⁹ cells/l
pH	7.25	7.32	7.23	7.37	7.41	ND	ND	7.35–7.45
PCO ₂	7.2	4.65	3.79	6.1	5.5	ND	ND	4.7–6.0 kPa
Base excess	-4.9	-7.2	-14.7	0.5	1.5	ND	ND	-3.0–3.0 mmol/l
Act. bicarb	22.8	17.6	11.4	25.6	25.8	ND	ND	mmol/l
Urea	20.8	40.2	40.8	8.2	7.5	5.8	7.5	mmol/l
Creatinine	146	354	868*	91	135	ND	ND	μmol/l
Total bilirubin	ND	ND	17.6	18.0	ND	ND	ND	0–40 μmol/l
Not conjugated	ND	ND	13.6	11.6	ND	ND	ND	μmol/l
Conjugated	ND	ND	4.0	6.4	ND	ND	ND	μmol/l
AP	ND	ND	1273	255	292	ND	297	140–300 iu/l
LDH	3530	ND	ND	6717*	ND	1552	2638 [†]	150–420 iu/l
CK	582	ND	ND	970 [†]	ND	675	190	5–160 iu/l
AST	2577	ND	ND	556 [†]	ND	315	263	10–275 iu/l
GSHpx	ND	ND	ND	ND	ND	146	ND	
γGT	ND	24	ND	4* [†]	ND	ND	7* [†]	5–20 iu/l
Total protein	ND	ND	ND	50	64	70	47	60–90 g/l
Albumin (%)	ND	ND	ND	44.4	42.7	43.0	34.3	35–55%
α-globulins (%)	ND	ND	ND	24.0	22.6	12.7	12.4	15–20%
β-globulins (%)	ND	ND	ND	22.0	20.2	20.1	24.0	<22%
γ-globulins (%)	ND	ND	ND	9.6	14.5	24.2	29.4	<25%
Vitamin E	ND	ND	ND	ND	ND	0.2	0.1	4.2–7.0 μg/ml
Selenium	ND	ND	ND	ND	ND	118.3	84.8	100–300 μg/l

Parameter	Case 8	Case 9	Case 10	Case 11	Case 12	Case 13	Case 14	Normal value
Haemoglobin	5.5	9.9	7.9	3.2	4.6	ND	7.3	7.5–9.0 mmol/l
PCV	0.24	0.49	0.39	0.15	0.19	0.41	0.30	0.36–0.42 l/l
Leucocytes	17.8	7.2	17.1	15.6	6.9	8.8	2.3	7.0–10.0 x 10 ⁹ cells/l
pH	7.34	7.36	7.3	7.37	7.3	7.22	ND	7.35–7.45
PCO ₂	4.16	5.7	5.6	5.4	5.0	3.2	ND	4.7–6.0 kPa
Base excess	-7.7	-1.1	-8	-2.2	-7.0	-14.9	ND	-3.0–3.0 mmol/l
Act. bicarb	ND	23.9	18.3	22.4	18.2	ND	ND	mmol/l
Urea	ND	5.6	22.9	15.3	12.6	ND	23.0	mmol/l
Creatinine	ND	109	137	106	123	ND	ND	μmol/l
Total bilirubin	ND	115.5	8.8	25.6	29.7	ND	ND	0–40 μmol/l
Not conjugated	ND	110.3	2.2	14.5	17.0	ND	ND	μmol/l
Conjugated	ND	5.2	6.6	11.1	12.7	ND	ND	μmol/l
AP	271	317	145	234	124	ND	257	140–300 iu/l
LDH	4406	2065	7380	6008	2759	ND	2321	150–420 iu/l
CK	ND	ND	ND	7736	ND	ND	330	5–160 iu/l
AST	819	1429	4304	905	242	ND	773	10–275 iu/l
GSHpx	ND	ND	ND	ND	ND	ND	ND	
γGT	4	19	ND	6	13	ND	ND	5–20 iu/l
Total protein	43	52	60	75	65	ND	57	60–90 g/l
Albumin (%)	ND	54	38.2	24.8	26.3	ND	50.0	35–55%
α-globulins (%)	ND	18	35.7	12	18.7	ND	27.9	15–20%
β-globulins (%)	ND	18	18.6	24.1	28.7	ND	15.9	<22%
γ-globulins (%)	ND	10	7.5	39.1	26.3	ND	6.2	<25%
Vitamin E	ND	ND	ND	0.8	ND	ND	0.3	4.2–7.0 μg/ml
Selenium	ND	ND	ND	ND	ND	ND	ND	100–300 μg/l

*Determined after i.v. fluid administration; [†]determined in blood samples taken on the second day of hospitalisation. Act. bicarb = actual bicarbonate concentration; γG = gamma glutamyltransferase; GSHpx = glutathion peroxidase.

mineralisation of muscle fibres with surrounding infiltrates of macrophages, syncytial giant cells, round cells and granulocytes with fibrosis (Fig 7). The myocardial changes were characterised by focal loss of cross-striations and some calcification of fibrils and diffuse presence of hypochromatic-swollen nuclei. The pericardial adipose deposits showed extensive necrotic changes as described above.

Discussion

Generalised steatitis (GS) has been described mostly in foals (Dodd *et al.* 1960; Kroneman and Wensvoort 1968; Platt and Whitwell 1971; Glyn 1972; Peyton *et al.* 1981; Hamir 1982; Foreman *et al.* 1986). Only a few cases have been reported in mature ponies and horses (Wensvoort 1974a,b; Taylor *et al.*

1988). In a controlled histopathological study performed on 173 horses and ponies by Wensvoort (1974a,b), changes in the adipose tissue were found in 44 animals including 6 near-term fetuses, 22 foals and 15 animals age 1–4 years. Of the 14 cases reported here, 6 were foals and 6 were young horses age 1–3 years. This seems to support the possibility that GS is a congenital disease in which foals are born already affected or predisposed to the condition. Oxidation of adipose tissue (with low levels of antioxidants) might occur when these foals encounter infectious agents such as viruses and parasites, leading to inflammation of body tissues, including the adipose tissue.

Normally, around parturition an increase in plasma vitamin E in mares is noted, giving rise to its accumulation in colostrum (Schweigert and Gottwald 1999). However, Maenpaa *et al.* (1988) found that the serum profiles of vitamins A, E and D in mares are subject to seasonal influences, with values being lowest from February to May and highest from June to August. They also found that serum levels of tocopherol in foals are significantly lower during the first 4 months *post partum* compared to their dams (Maenpaa *et al.* 1988). Low serum glutathione peroxidase activity (Hamir 1982; Perkins *et al.* 1998) or low serum concentrations of selenium and tocopherol (Higuchi *et al.* 1989) have been reported in dams giving birth to foals affected by white muscle disease. The dam of the Friesian foal here reported with GS (*Case 14*) also showed an abnormally low serum vitamin E concentration. To the authors' knowledge, vitamin E concentrations of dams giving birth to foals with GS have not been reported so far. In 4 of our cases, serum vitamin E concentrations were determined and found to be abnormally low. The question remains as to whether this was part of the cause or due to the excessive use of antioxidants in the inflamed adipose tissue. Thirteen of the 14 cases had been at pasture during the 2–3 months prior to developing GS. None of the cases had been given any supplemental feed. These facts could support the theory that there is a relative lack of antioxidants due to receiving a ration which has a low to normal vitamin E content and is high in unsaturated fatty acids.

The relatively high incidence in Shetland ponies and other cold-blooded types of horses is remarkable. Of the 14 cases reported with GS, 6 were Shetland ponies, 4 were cold-blooded types of horse and 1 was a donkey. In the study of Wensvoort (1974a,b), 75% were Shetland ponies. This raises the question as to whether there are possible hereditary differences in, for instance, transplacental passage and liver storage of selenium, colostrum vitamin E or in the composition or turnover of adipose tissue. Could it be that ponies and cold-blooded types of horse are predisposed to developing GS, or is it that this type of pony/horse is relatively over-represented in suboptimally fed equids?

Making a diagnosis of yellow fat disease can be simple in cases with an obvious painful thickening of the nuchal ligament and subcutaneous fat of the groin and axillary region. However, as in *Cases 3, 4 and 5*, signs of abdominal disease caused by inflammation of mesenteric and/or subperitoneal fat may be more obvious than the involvement of the

subcutaneous tissues. Some consistent clinical findings can be of help in putting steatitis in the right place on the clinician's list of differential diagnoses. The patients involved are often foals or young mature horses, and are mostly ponies or cold-blooded types. The clinical history often mentions anorexia and an abnormal faecal consistency, either diarrhoea or excessively firm faeces with a covering membrane of fibrinous material, suggesting delayed transit. The one horse (*Case 5*) in which the abdominal adipose tissue was not markedly involved at the time of referral had maintained a good appetite and faeces of normal consistency. Clinical examination often yields an increased rectal temperature, a markedly elevated heart rate and subcutaneous oedema. The marked tachycardia might be due to myocardial necrosis in some of these patients (Hong *et al.* 1996; Shite *et al.* 2001), and/or because of inflammation of fat surrounding the coronary arteries. Normal functioning of the intestines is probably impaired by extensive inflammation of the mesenteric fat. This might explain why the contents of the large colon are often liquid and faecal examination reveals no enteric pathogens. This and the fact that the animal is anorexic and in a catabolic state might be responsible for low serum albumin concentrations, resulting in subcutaneous oedema and ascites. Abdominocentesis might show either an increased amount of clear fluid, as in ascites, or just a small amount of turbid fluid, possibly with some red blood cells. Radiopaque densities on thoracic radiographs, which appeared to be calcification of adipose tissue, were reported by Kroneman and Wensvoort (1968). In *Case 7*, it remains uncertain whether the thoracic findings can be attributed to the process of generalised steatitis. The most helpful serum biochemical changes proved to be highly elevated LDH and AST activity, together with a moderate to highly increased CK activity.

Based on the histopathological changes seen in the adipose tissue, Wensvoort (1974a,b) classified cases of steatitis into 3 different stages of disease. It was concluded that less advanced stages of steatitis may be present in horses and ponies who show no clinical signs or do not die from GS. These stages may be characterised not only by less extensive spread of the affected adipose tissue, but also by a much simpler histopathological structure. There is, however, only little evidence that diseased animals may recover (Glyn 1972; Peyton *et al.* 1981). All 14 cases reported here had to be subjected to euthanasia or died because of GS. It is therefore concluded that the prognosis in equids showing obvious signs of GS is poor. Making an early diagnosis of yellow fat disease is important to prevent lengthy and costly treatments (including laparotomy) and avoid unnecessary suffering. Also, other members of the herd need to be screened for lack of antioxidants and should subsequently receive appropriate supplementation.

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