Case report:
Malnutrition and undernutrition as cause of mortality in farmed reindeer (*Rangifer tarandus tarandus* L.)

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Abstract: Chronic diarrhoea evolved during the third year of farming in a group of six reindeer farmed in central Sweden. The first death occurred in July, and despite offering supplemental feed, the deaths continued. Within 9 months five animals (83%) were dead. The necropsy findings indicated emaciation in all cases. The initially adequate clover vegetation in the paddock had been depleted over the years, leading to malnutrition and undernuttition of reindeer in the summer season.

Key words: nutrition, pathology, starvation.

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Case history

Farming reindeer or keeping reindeer in paddocks during varying periods of the year is becoming more common in Sweden not only within, but also outside traditional reindeer herding areas. A small scale reindeer farm was initiated in 1993 in central Sweden (60°N, 17°E), in a two hectare paddock with electric wire fence. In May the third year (1995) the herd numbered six animals; an old male (>5 years old), three adult females (all >10 years old), and two younger animals born in the paddock; a two-year-old female and a yearling male.

In 1993 the animals thrived on the abundant forage available in the paddock, mainly tall and dense white clover (*Trifolium repens* L.). In winter the reindeer were fed hay. The animals were dewormed yearly in early May with ivermectine, 200 µg/kg s.c. (Ivomec vet®, Veter AB, Södertälje, Sweden). In the autumn of 1994, it was observed that the reindeer were in poor body condition compared to previous autumns. Pelleted screening feed (Avrenspellets Standard, Odal Ek. för., Uppsala, Sweden) was then given continuously in addition to hay. Through the spring and the summer of 1995 an obvious change in the paddock vegetation was observed. Only patches of low clover with woody stalks remained. During this grazing season the reindeer also shared the paddock with three horses. The only calf born this year was delivered in early June. The reindeer did not appear to gain weight, some were observed with loose stools, and on 7 July the only female to have given birth this season, died. Suspecting a viral disease to be the cause of the poor general condition of the reindeer, specimens for virus isolation were taken at the necropsy as well as blood samples from the living animals. Apples, pellets, and beet pulp with molasses were then given continuously as feed supplementation. On 30

November the yearling male died. At necropsy, samples were taken for analysis of trace elements, for virus isolation, and for histopathology.

In the beginning of December all the remaining reindeer had loose stools and all except the old male developed a profuse, brown, watery diarrhoea. Supplementation was changed to pelleted reindeer fodder (Renfor bas, Fodercentralen AB, Umeå, Sweden), but only small amounts of this supplement were consumed. The reindeer were visibly emaciated and none of the animals had produced a normal winter fur coat. The remaining four animals were given supportive treatment. Cortisone temporarily increased appetite but had no lasting or significant effect. Different remedies intended to stimulate ruminal flora had no obvious clinical effect, while repeated i.m. injections of copper (Copamex Ltd, UK) combined with the administration of a rumen bolus containing copper and minerals appeared to aid in stemming the diarrhoea.

Nevertheless, on 22 January 1996 a second old female died. On 21 February the two-and-a-half year old female died and on 9 April the last old female died.

The surviving old male was tethered on a long leash and moved around in the forest where he readily consumed lichens and browsed herbaceous plants very selectively, nibbling preferred parts of plants and leaves. Within a few days the stools had a more normal appearance and he rapidly regained weight.

Methods

Necropsies were performed according to the Standard operating procedure (SOP) practised at the Department of Pathology, National Veterinary Institute in Uppsala, Sweden.

Tissues for light microscopy were fixed in 4% formaldehyde, trimmed, dehydrated, embedded in paraffin, cut to 5 µm thick sections and stained with hematoxylin/eosin. To visualise iron pigments, sections of spleen and liver were also stained with Prussian blue.

Random areas of formalin-fixed liver tissues were osmiocated, dehydrated and embedded in epoxy for electron microscopy. Sections were cut and stained for light microscopy. Selected areas were sectioned using a LKB-ultratome, picked up on copper grids, stained with uranylacetate and leadcitrate and examined in a Philips electron microscope 420 at 60 kW.

A micro-neutralisation test according to Frey & Liess (1971) was used for the detection of bovine virus diarrhoea (BVD) virus. Electron microscopy was conducted in search of herpes-, rota-, and coronavirus according to Doane & Anderson (1987).

Levels of copper in serum, and liver and kidney tissue samples were determined by direct current plasma atomic emission spectrometry (DCP-AES) according to Frank & Petersson (1984).

Crude protein in the white clover growing outside as well as inside the paddock was detected with the semi-automatic Kjeldahl procedure (Foss-Tecator, Höganäs, Sweden) according to standard Association of analytic chemistry (AOAC) procedures.

Results

Investigations on animals

The necropsies revealed almost identical changes in all five animals. The skin was dry and wrinkled, the eyes were sunken and the carcass dry and pale, indicating dehydration and anaemia. The animals had not shed the summer pelt properly in winter, or grown any winter pelage. In all animals the antlers appeared shorter (in one case these were only 10 cm long) and more slender than in previous years. The plantar sides of the hind legs and hocks were covered with thick layers of faecal material. The musculature was severely atrophic and all animals revealed a serous atrophy of bone-marrow and pericardial fat indicating emaciation. The adrenal glands generally showed a severe thickening of the cortex, indicating a long-term stress condition. Approximate ageing of the adult animals according to teeth-wear was ten yrs or older. None of the adults had completely worn down teeth, or jaw joint disease that could indicate problems ruminating. The last female that died also had multiple old rib fractures and brittle bones indicative of osteoporosis. Microscopic examination of bone showed abnormally thin medullar bone trabeculae.

Mucosal erosions were found in varying numbers in the mouth and oesophagus of all dead animals (Fig. 1). Microscopically the lesions, focal areas of unspecified cell degeneration, differed markedly from lesions produced by pestivirus (Morton *et al.*, 1990) and herpesviruses (Rockborn *et al.*, 1990). The appearance was instead similar to lesions associated with vitamin deficiencies, malnutrition, and undernutrition (Barker, 1992).

A common necropsy finding in all animals was

Fig. 1. Ulcerations (➨) in the epithelium, here in the hard palate, were seen in the digestive tract of the emaciated reindeer. Initial suspicion of viral ethiology was not confirmed. Microscopical examination showed degenerative lesions, not inflammatory reactions.

ruminal indigestion with foul-smelling romen contents. A notable finding was a pronounced oedematous thickening of the abomasal mucosa which was three to four times thicker than normal and intensely red in colour (Fig. 2). Microscopically the changes in the abomasum were due to congestion and oedema. The intestines were in contrast generally thinwalled and flaccid, macroscopically a catarrhal enteritis. Microscopically there were no signs of infectious bacterial diseases. Virus was not isolated from sera or tissues and was not found at electron microscope examination of the digestive tract mucosa. Small numbers of gastrointestinal nematodes were found in the digestive tracts examined for parasites.

The liver appeared dark, almost black in colour and small in size, the capsules slightly wrinkled. The spleen was small and thin. In light microscopy moderate amounts of intracytoplasmic small yellowish pigment granules were observed in most of the hepatocytes of all animals. Electron microscopy confirmed that these were lipofuscin granules (Fig. 3). Microscopically the liver and spleen showed heavy, or excessive, deposits of hemosiderin (Fig. 4).

The chemical analysis of serum, and liver and kidney tissues (Table 1) demonstrated low levels of copper.

The necropsy findings in all five animals was emaciation, ruminal indigestion, catarrhal enteritis, and in addition, osteoporosis in the last case. The cause of death was concluded to be emaciation, with the other findings considered to be secondary to the state of malnutrition.

Investigations on soil and forage

The Geological Survey of Sweden incidentally had a monitoring site close to the paddock. Soil samples from the area showed that the levels of both copper and molybdenum were within the normal range (Aastrup, pers. comm., 1998).

Chemical analysis of the forage showed a higher protein content (19.5%) in clover growing inside the paddock compared with clover growing on the outside of the enclosure (10.8%).

Discussion

In most deer species the winter reserves are mainly stored as protein in type 2B muscle fibres (Kiessling *et al.,* 1986; Renecker & Hudson, 1986). For rein-

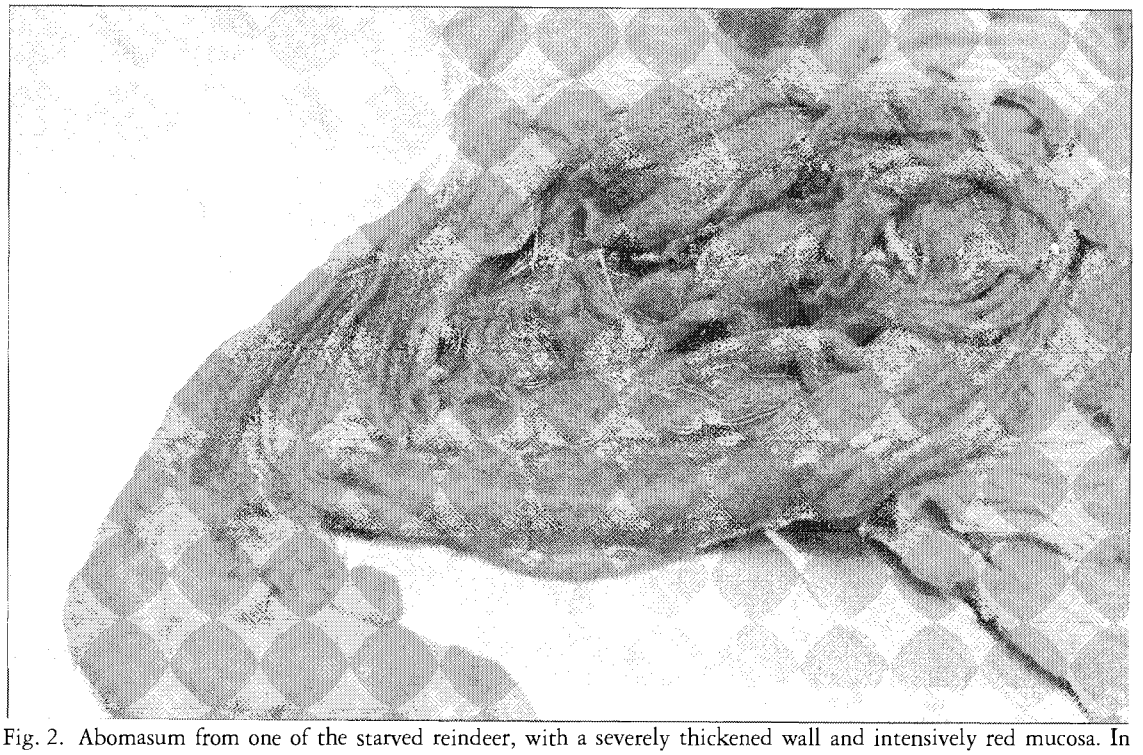

Fig. 2. Abomasum from one of the starved reindeer, with a severely thickened wall and intensively red mucosa. In gross examination consistant with gastritis, but microscopic examination showed no inflammation, only congestion and oedema.

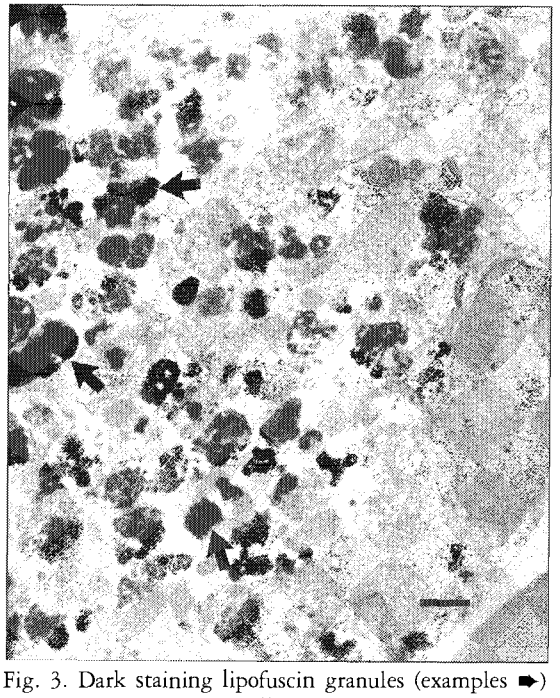

Fig. 3. Dark staining lipofuscin granules (examples ➨) seen in the liver cells of the emaciated reindeer. Bar is in the order of 181 nm.

deer, summer nutritionally represents an anabolic period while winter is a catabolic period. During summer, reindeer very carefully select the plants they consume, browsing plants rich in nutrients and protein, and consuming four to eight times the amount of dry matter consumed during the same time span in winter. If the nutritional value of the fodder consumption during summer is reduced to only twice that of winter, the reindeer will starve to death as they do not build up enough reserves for the coming winter (White, 1983; Reimers, 1983).

Starvation, or a lack of appropriate protein-rich forage, produces a secondary copper deficiency and a microcytic anaemia (Valli, 1993). The deposits of hemosiderin in liver and spleen could be explained by the fact that ceruloplasmin, a copper-containing protein important to iron metabolism, rapidly diminishes in starving animals, and results in both low copper levels and excessive deposition of iron in the spleen (Valli, 1993). The presence of lipofucsin granules in hepatocytes is a typical finding in starved animals (Gahdially, 1988).

The forage initially available in the paddock was enough for the maintenance of the reindeer. After

Table 1. Reindeer kept in a paddock; sex, age, date of death, serum copper levels, organ copper levels, and final diagnosis. Reference values from Department of Chemistry, National Veterinary Institute in Uppsala, Sweden, and Puls (1994).

Animal	Age yr.	Diseased date	Serum-Cu (mg/kg) 996-01-17	Kidney-Cu (mg/kg wet .w.)	Liver-Cu (mg/kg wet.w.)	Final diagnosis
Female	>10	1995-07-07	-	-	-	Emaciation
Male	1½	1995-11-30	-	18.0	6.8	Emaciation
Female	>10	1996-01-22	8	13.3	4.4	Emaciation
Female	2½	1996-02-21	8	14.0	23.0	Emaciation
Female	>15	1996-04-09	10	14.0	5.4	Emaciation, osteoporosis
Ref. values	-	-	11-28	5-50	15-85	-

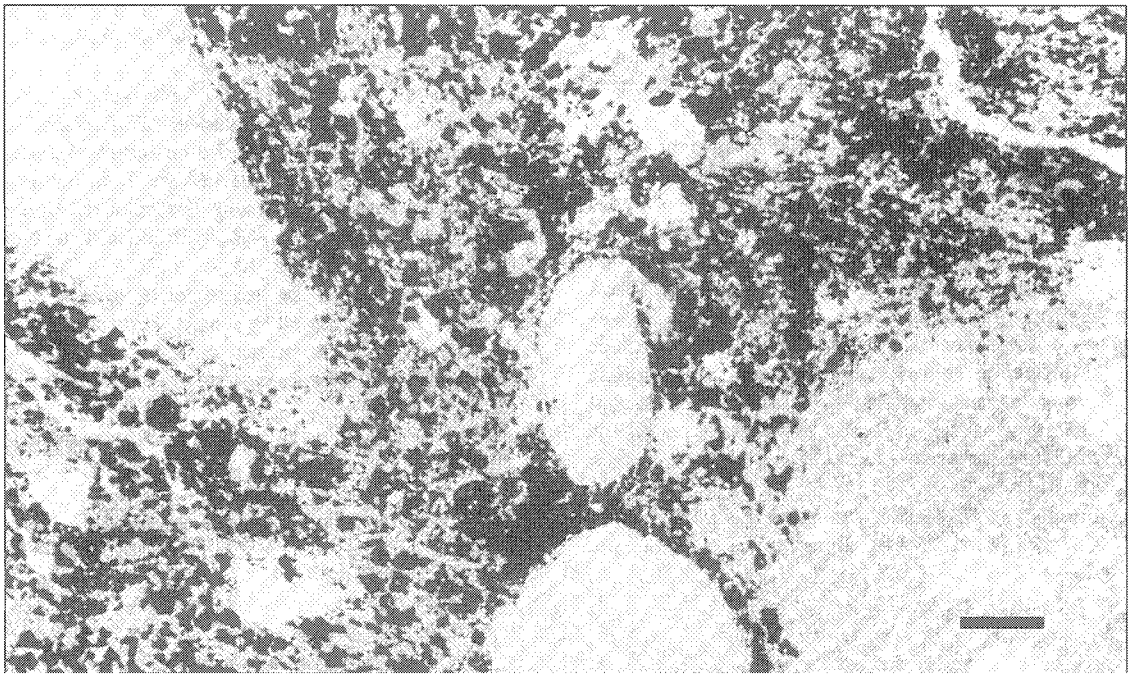

Fig. 4. Spleen tissue from one of the emaciated reindeer, loaded with iron pigment, hemosiderin, seen as darkly stained particles. Prussian blue staining. Bar is in the order of 200 µm.

the second year there was a dramatic change, probably due to overgrazing and grazing competition from the horses. The fact that the reindeer disregarded the protein-rich clover in the paddock might be explained by an altered palatability of the clover.

In conclusion, over the years, there was a lack of appropriate amounts of feed available in the paddock during summer to restore muscle protein reserves lost during winter. The dietary situation with a continuous suboptimal intake of nutrients was not recognised in time to reverse the poor clinical condition of the animals. The resulting emaciation led to death for five out of the six animals in the herd, the first death occuring in the middle of summer. The experiences from this case report emphasises the importance of knowledge on adequate nutrition and the specific requirements of reindeer, especially in non-traditional reindeer management systems. The clinical and pathological investigations could not point out any specific pathogen, but instead strongly points to malnutrition and undernutrition as the cause of the mortality.

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