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Brief Communication

MUSCULAR AND MYOCARDIAL DEGENERATION IN RAPIDLY GROWING MALE MINK KITS

Muscular degeneration (dystrophy) occurs in a variety of animal species (*Scott* 1980), including the mink (Mustela vison) in which the condition has been known for many years in association with "yellow fat" ("steatitis"). When related to "yellow fat", the muscular lesions are, as a rule, of less wide distribution, and frequently less conspicuous than the changes in the adipose tissue.

During recent years sudden deaths have occurred in August/September in rapidly growing male mink kits. In some of these cases widespread muscular degeneration are found in the skeletal muscles (Fig. 1), in other cases less evident degenerative lesions, together with microangiopathy, in the myocardium. These instances have been interpreted as somewhat different manifestations of the same syndrome, related to vitamin E/selenium deficiency. It has, however, not been possible to explain why the skeletal muscles are affected in animals from some farms, and the myocardium in animals from others.

Acute muscular degeneration, in the absence of "yellow fat" was recognized in Norway for the first time in the autumn of 1969 (*Nordstoga* 1969). During the first years the muscular involvement was frequently accompanied by extensive haemorrhages in the subcutaneous tissue and/or internal organs, especially in the sweetbread. In some farms the haemorrhages were the dominating feature of the necropsy picture, and the association with muscular lesions became first evident after observation of the farms for a considerable time, the bleedings being so extensive that a disturbance in the coagulation mechanism was suspected. Platelet count, screening tests for the extrinsic (Thrombotest and Normotest) and intrinsic (cephalin time) coagulation mechanism, fibrinogen assay and fibrinolysis (plasma clot lysis time) were, however, not much different from normal* (*Stormorken*, personal communication). Platelet or coagulation defects did not therefore seem to be the cause of the bleedings, and the conclusion was drawn that the widespread

* Tests performed at the Institute for Thrombosis Research, University Hospital, Oslo.

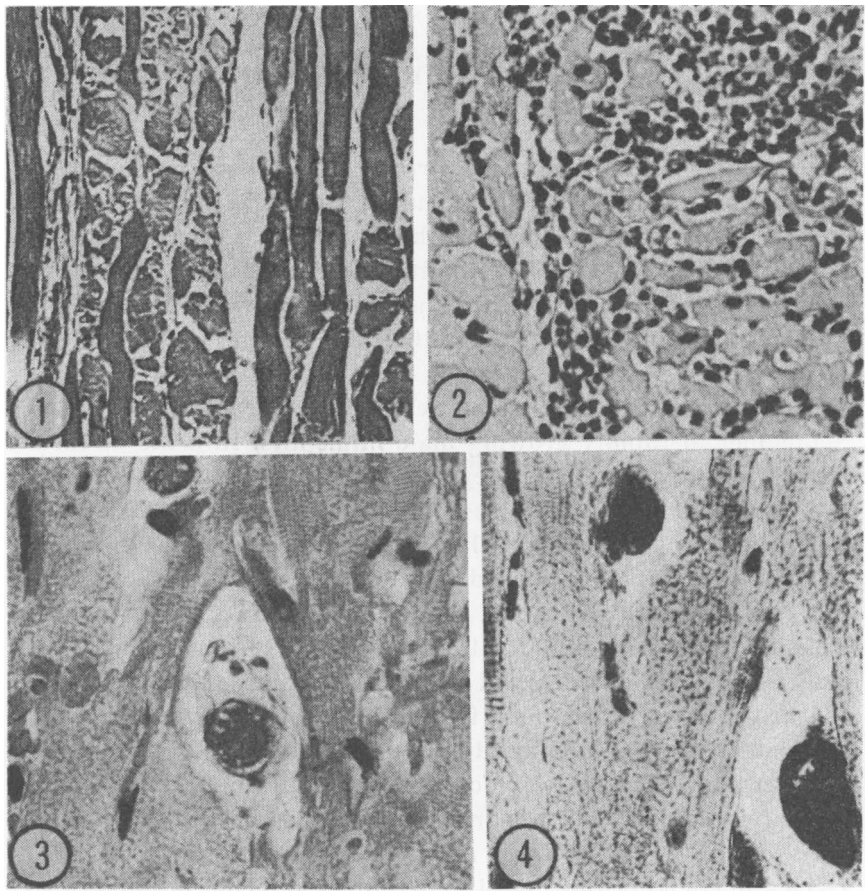

Figure 1. Skeletal muscular degeneration, with hyalinization and fragmentation of muscle fibres. Haematoxylin & eosin, × 170.

Figure 2. Myocardial degeneration, with inflammatory reaction. Haematoxylin & eosin, × 300.

Figure 3. Swollen myocardial muscle fibres, with loss of cross striation and myolysis. Two thrombosed arterioles, surrounded by oedema, are visible. Haematoxylin & eosin, × 480.

Figure 4. Degenerated area of myocardium, with two thrombosed vessels, surrounded by oedema. Phosphotungstic acid haematoxylin, × 480.

haemorrhages were caused by vascular injury and thus associated with the same etiological factors as those responsible for the muscular degeneration, which often was observed in the same animals.

In mink dying of acute muscular degeneration one cannot

expect evident macroscopical changes in the skeletal muscles, although some animals have a dilated urinary bladder, containing coffee brown urine (myoglobinuria). The necropsy picture indicates an acute circulatory collapse, the predominating findings being marked congestion of internal organs, moderate amounts of serous fluid in the body cavities, and pulmonary oedema. Some animals appear anaemic, have an enlarged spleen, and frequently incipient fatty change of the liver.

When the myocardium is affected, the accumulation of serous fluid in the body cavities is more pronounced. Sometimes haemorrhages of macroscopical sizes, and/or pale degenerated areas are visible on the cut surface; the lungs are heavily congested ("shock lungs"). A marked hepatic congestion is also present, while evident extracardial haemorrhages are, as a rule, not found in these cases. Microscopically are myocardial injury with haemorrhages, oedema and myolysis constantly found, occasionally are incipient cellular infiltrations also present. Mural degeneration sometimes occurs in myocardial arterioles, together with hyaline thrombi in small vessels, including minor veins (Figs. 2—4). Thus, in these cases, the cardiac alterations are very similar to porcine "mulberry heart" disease (*Grant* 1961, *Nafstad* 1971).

When comparing with corresponding conditions in other species of domestic animals, it is difficult to imagine other causal factors than vitamin E/selenium deficiency, although the feed used for fur animals in Norway usually contains excess of these substances. A possible hypothesis could be a genetic predisposition, in animals selected for rapid growth and body size. It is an experience that the deaths in the farms decrease after preventive treatment with vitamin E/selenium of animals supposed to be especially predisposed. Preliminary vitamin E and selenium analyses seem to indicate normal values of selenium in organs of animals with myocardial alterations, while the vitamin E-content is sometimes reduced (*Loftsgaard,* personal communication). This observation corresponds to the situation in pigs, in which myocardial lesions are more likely to occur in association with lack of vitamin E than with lack of selenium. There seems also to be a rather low absorption of tocopherol from the intestine in mink, compared to other species (*Eskeland & Rimeslåtten* 1979).

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