Diagnosis of white muscle disease in two goat kids: combining pathological evaluation with ICP-MS based trace element analysis

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ABSTRACT

White muscle disease, commonly observed in young ruminants, is a myopathy caused by selenium (Se)/vitamin E deficiency. In this case, white muscle disease was diagnosed in two goat kids following multiple kid mortalities in a hair goat herd in the Gördes district of Manisa province. The disease was diagnosed based on the farmer's anamnesis, detailed pathological examination, and measurement of Se concentrations in blood and tissue samples using Inductively Coupled Plasma Mass Spectrometry (ICP-MS). In addition to the clinical and pathological findings, the diagnosis of white muscle disease was further supported by the detection of markedly low Se concentrations in the blood, liver, and muscle tissues of the kids. This case report highlights the importance of trace element analysis in the differential diagnosis of white muscle disease from other myopathies in small ruminants.

White muscle disease, also known as nutritional myopathy, is a common metabolic disorder caused by Se and/or vitamin E deficiency in blood, tissues and organs (4, 8, 11, 13). Although it is more common in lambs, kids, and calves, adult sheep and goats may also be affected (14, 15). White muscle disease is characterized by hyaline degeneration and necrosis in skeletal muscle, cardiac muscle and the diaphragm, leading to a pale, dry, opaque appearance and distinct white discoloration of the muscles ranging from yellow to cream white (2, 15). Clinical symptoms such as difficulty standing, reluctance to move, muscle stiffness and spinal curvature are usually observed in sick animals (8, 12). The pathogenesis of the disease involves necrotic lesions occurring in many muscle tissues such as limbs, heart and respiratory system as a result of oxidative stress caused by free radicals (4, 7). Histopathological and laboratory examination of the affected muscles helps differentiate the disease from other possible myopathies. Laboratory tests typically include quantification of Se and vitamin E levels in blood or tissue samples (4, 7, 12). The prognosis varies depending on the severity of Se deficiency and the extent of muscle damage (2, 12). Overall, white muscle disease is a significant nutritional disorder that negatively affects animal health and welfare, particularly in ruminants raised in regions with Se-deficient soils (12, 14).

The case materials consisted of carcasses and pre-mortem blood samples from two 2–3-month-old goat kids, which were submitted to the Bornova Veterinary Control Institute for diagnostic evaluation following a

series of deaths in a herd of 85 hair goats in the Gördes district of Manisa province. According to the anamnesis provided by the farmer, the adult goats grazed on pasture year-round and were supplemented with hay, dry forage, and barley in the evenings. It was reported that kidding began in February 2024, and the kids were fed exclusively with maternal milk during their first month. No treatments other than a polyvalent vaccine (Coglavax®, 2 ml per animal, administered twice subcutaneously) were applied. A total of 18 kids, but no adult goats, had reportedly died. The initial symptoms observed in the kids included a desire to lick soil and foreign objects, with a mud-like accumulation of soil found in the rumen contents of the deceased animals. The clinical course included reluctance to suckle, lethargy, limb stiffness, disheveled hair, difficulty breathing, and death within 2-3 days. After the deaths began, each kid received an intramuscular injection of 3 ml Activate® mineral solution (2.5 mg copper gluconate; 1.25 mg sodium selenite; 5 mg manganese; 5 mg zinc gluconate per mL / ALKE, Istanbul, 50 ml, IM) every 3 days.

The most prominent finding during the necropsy was the presence of white necrotic areas in the heart muscle of both kids (Figure 1. A-B), along with anemia observed in the skeletal muscles particularly in the hind limbs and diaphragm (Figure 1. C). The lungs of Kid 1 were markedly enlarged and dark red, with fluid oozing from the cut surface, and foamy fluid was present in the trachea and bronchi of both kids. Additionally, a blood clot was detected in the left ventricle of the heart in Kid 1 (Figure 1. B). Based on these findings, tissue samples were collected for pathological and biochemical examination to confirm white muscle disease.

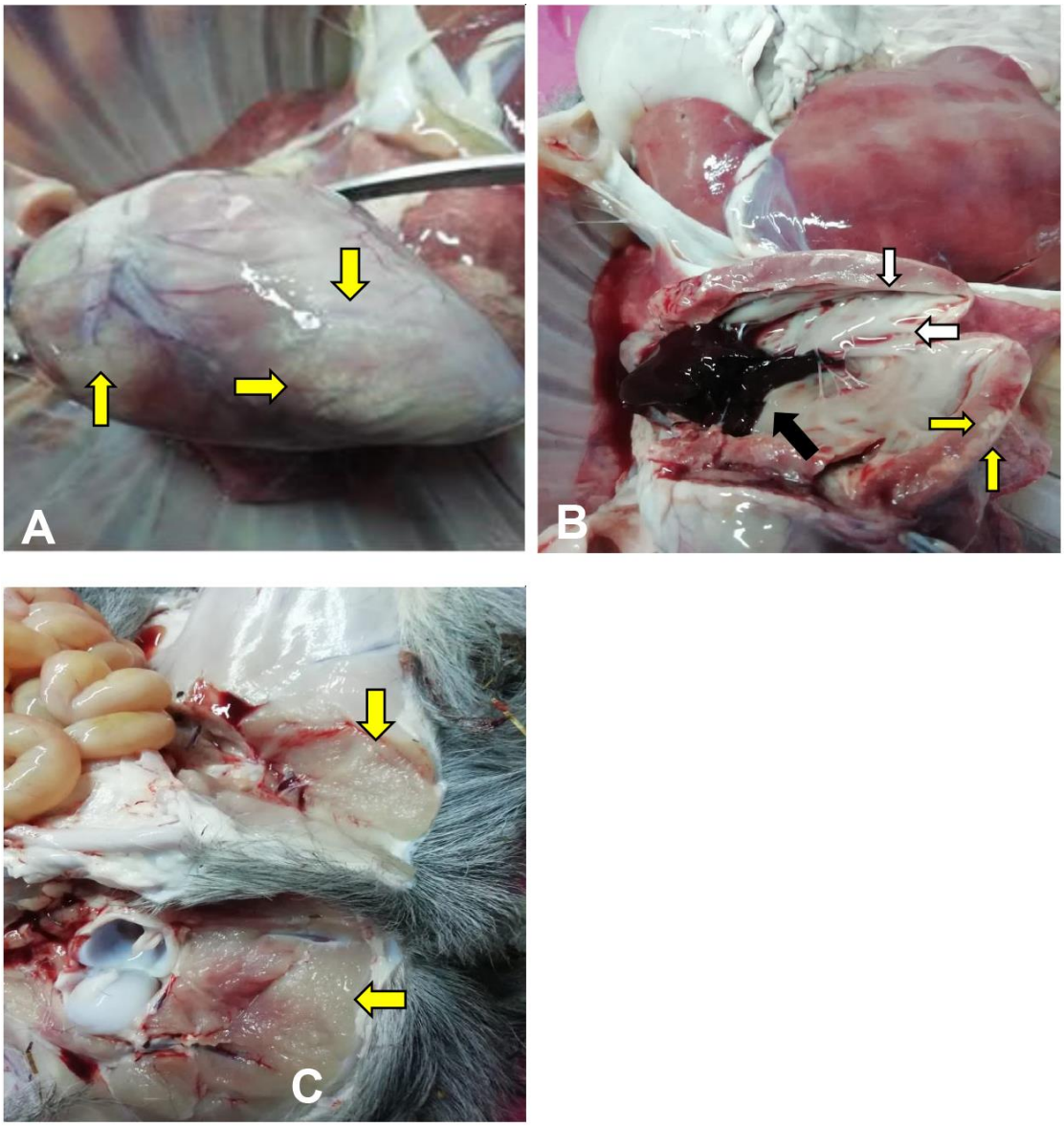

Figure 1. A. Pale areas of calcification in the epicardium (yellow arrows) of the heart. **B.** Extensive white calcified areas in the subepicardial myocardium (yellow arrows) and on the endocardial surface (white arrows), along with a blood clot (black arrow) related to cardiomyopathy in the left ventricule of the heart. **C.** Pale appearance in the gluteal muscles.

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The collected tissue samples were fixed in 10% buffered formalin, processed routinely, embedded in paraffin, sectioned at 4 µm, stained with hematoxylin and eosin (H&E), and examined under a light microscope (Olympus BX51). For biochemical analysis, trace element levels in blood, liver, and muscle were determined by inductively coupled plasma mass spectrometry (ICP-MS; Agilent 7700).

Histopathological examination revealed extensive hyaline degeneration and necrosis in the muscle fibers of the skeletal muscles (gluteal muscles, tongue, diaphragm) and the myocardium of both kids, along with mononuclear cell infiltrations around the blood vessels and in the interstitial tissue (Figure 2). Additionally, focal areas of calcification were observed in the necrotic regions of the heart (Figure 2. A). There was edema and congestion with a small number of erythrocytes in the alveoli. The liver also showed signs of congestion.

Blood Se concentrations were significantly lower in both Kid 1 (0.73 µg/dL) and Kid 2 (0.94 µg/dL) compared to the normal range of 30-70 µg/dL. Analyses of muscle and liver tissues confirmed markedly reduced Se concentrations, supporting a diagnosis of Se-deficient white muscle disease (Table 1). Additionally, both kids had elevated blood Cu and Zn levels compared to reference intervals (10, 14).

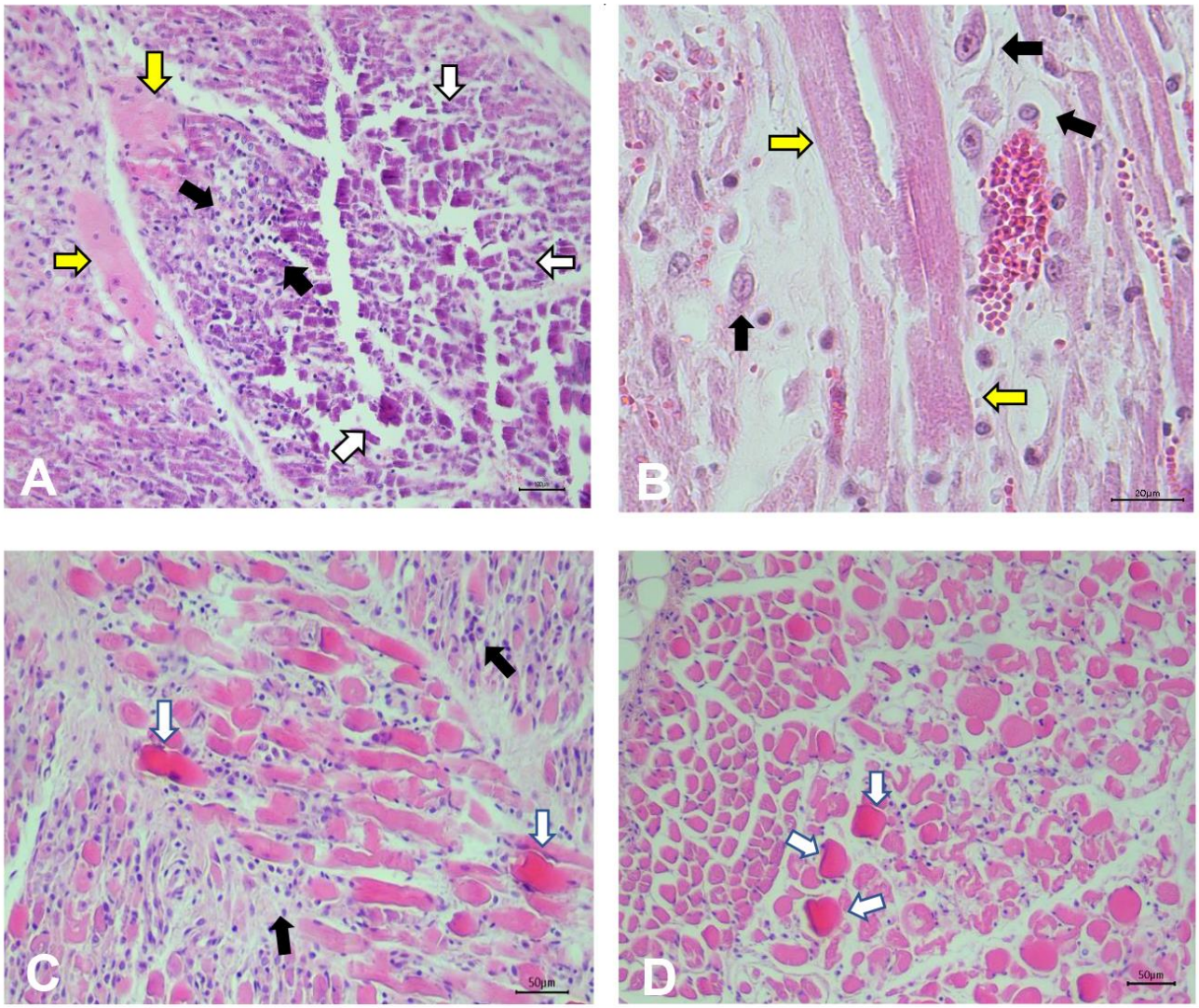

Figure 2. A. Homogeneous pink degenerative muscle fibers (yellow arrows) with focal calcification (white arrows) and mononuclear cell infiltrations (black arrows) in the subepicardial myocardium. H&E stain. **B.** Degenerative muscle fibers (yellow arrows) with mononuclear cell infiltrations (black arrows) in the perivascular and interstitial areas of the subendocardial myocardium. H&E stain. **C.** Muscle fibers exhibiting Zenker's necrosis (white arrows) and mononuclear cell infiltrations (black arrows) between these muscle fibers in the tongue. H&E stain. **D.** Necrotic (white arrows) muscle fibers of irregular shape and size in the gluteal muscle. H&E stain.

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Table 1. Trace element levels in blood, liver and muscle of two kids.

Traits	Trace Elements	Kid 1	Kid 2	Ref Interval
Blood (µg/dL)	Selenium (Se)	0.73	0.94	30-70
	Manganese (Mn)	27.23	23.37	20-40
	Copper (Cu)	196.9	186.7	80-120
	Zinc (Zn)	1505.1	1351.1	80-120
	Cobalt (Co)	0.35	0.37	-
Liver (ppm)	Selenium (Se)	0.036	0.041	0.1-0.3
	Manganese (Mn)	3.349	3.732	3-6
	Copper (Cu)	11.338	15.48	< 150
	Zinc (Zn)	57.523	107.18	20-100
	Cobalt (Co)	0.027	0.039	0.1-0.5
Muscle (ppm)	Selenium (Se)	0.029	0.017	0.1-0.3
	Manganese (Mn)	0.443	0.481	-
	Copper (Cu)	3.569	2.648	2-10
	Zinc (Zn)	48.45	30.40	20-100
	Cobalt (Co)	0.017	0.004	-

*Reference values adapted from, Underwood and Suttle (14), and Puls (10).

White muscle disease is a metabolic disorder associated with Vitamin E and Se deficiency (8, 15). The disease is commonly observed in small ruminants grazing on Se-deficient soils, and its severity can vary depending on the degree of Se deficiency (2). Diagnosis can be achieved through necropsy findings combined with direct measurements of Se and Vitamin E levels (2, 5, 6, 15). In this case report, white muscle disease was diagnosed in two kids based on anamnesis, necropsy, histopathology, and Se level analyses.

The disease is particularly characterized by degeneration in the heart, gluteal, diaphragm, tongue, and intercostal muscles (2, 3, 8). In the present case report, lesions were observed in these muscle groups, consistent with literature findings. Additionally, the pale calcified areas in the heart muscle, more prominent in the epicardium and endocardium, are consistent with previous reports (9, 15) (Figure 1. A). The presence of a blood clot in the left ventricle of one of the kids is also indicative of heart failure due to cardiomyopathy (9) (Figure 1. B).

Histopathologically, similar to previous reports, the degenerated and necrotic muscle fibers in both the heart and skeletal muscles appeared swollen, pink, and irregular in shape. Dark pink-purple dystrophic calcification areas, along with mononuclear cell infiltrations in the interstitium and around the blood vessels, were also observed (8, 9) (Figure 2. A-D). The mild congestion and edema observed in the lungs, as well as the liver congestion, in our study have also been linked to heart failure in previous studies (9).

According to the anamnesis provided by the farmer, the affected kids exhibited typical clinical signs of white muscle disease, including reluctance to suckle, lethargy, limb stiffness, disheveled hair, and difficulty breathing. Additionally, in this case, the necropsy and histopathological findings, combined with trace element analyses of blood, liver, and muscle tissues, clearly indicated white muscle disease due to Se deficiency. Both kids had significantly low Se levels in their blood, liver, and muscle tissues compared to the reference ranges (Table 1). These markedly low Se trace element levels are consistent with results reported in other studies on white muscle disease (1, 5, 11). Interestingly, both kids had elevated blood Cu and Zn levels. Although the Cu and Zn concentrations in liver and muscle tissues were within normal ranges, the elevated blood levels might be explained by the mineral injections (Activate®) administered to the kids over three days during the disease process.

The white muscle disease diagnosed in the kids in this case is likely due to insufficient Se levels in the region where the goat herd grazed or in their diet (2, 5). As mentioned in the anamnesis, the adult goats were only supplemented with hay, dry forage, and barley in the evenings and were otherwise left to graze on pasture. Early diagnosis and Se supplementation are crucial for the survival of affected animals (5, 13). In this case, although mineral supplementation was attempted through injections after the onset of deaths in the kids, this intervention appears to have been insufficient to correct the severe Se deficiency, as evidenced by the total of 18 kid deaths in the herd.

Differential diagnoses for the presented case include infectious, traumatic, and other nutritional myopathies.

Although the diagnosis of white muscle disease is traditionally based on clinical signs, necropsy, and histopathological findings, the low Se levels determined using ICP-MS in this case are critical for distinguishing the disease from other causes of myopathy. In particular, oral or parenteral Se supplementation to pregnant animals raised on Se deficient soils significantly reduces the risk of neonatal white muscle disease (10, 13).

In conclusion, this case represents the first report to definitively diagnose white muscle disease in two kids based on anamnesis data, necropsy and histopathological findings, and highly sensitive Se level analysis using the ICP-MS method. Therefore, this study provides valuable contributions to the literature on the diagnosis of white muscle disease in small ruminants. It also underscores the importance of integrating clinical, pathological, and laboratory data for the accurate diagnosis of white muscle disease.

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Ethical Statement

This study presents cases of white muscle disease diagnosed through pathological examinations and Se analyses in two kids submitted to the Bornova Veterinary Control Institute for diagnostic purposes. This study did not involve any experimental procedures on live animals. The carcasses and associated samples were submitted by the owners for diagnostic purposes.

Conflicts of Interest

The authors declared there is no conflict of interest.

Author Contributions

CO and EG conceived and designed the project. MK and DT acquired data. CO, EG, MK, and DT analysed and interpreted data. CO, and EG wrote the paper.

Data Availability Statement

The data supporting this study's findings are available from the corresponding author upon reasonable request.

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