Secondary Copper deficiency associated dermatitis in Goats: Insights from hematobiochemical, mineral and histopathological investigation

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Abstract

A goat farm holding forty-five Thanjavur black, female goats of one to nine years old reared under semi intensive system had a history of alopecia, loss of hair pigmentation, dry and crusty skin lesions all over the body in twenty goats. Animals had poor response to treatment with systemic and topical medications in the past six months. Skin scrapings, punch skin biopsy, fecal pellets, whole blood and serum and randomized feed samples were collected for laboratory analysis. Skin scrapings and fecal samples were negative for parasites. Whole blood analysis showed decreased in haemoglobin, packed cell volume and elevated red cell distribution width. Blood profile was suggestive of microcytic hypochromic anaemia. Serum analysis showed elevated ALP, AST and hypoproteinaemia. Histopathology of skin revealed hyperkeratosis, parakeratosis, subacute dermatitis with collagen breakage and lysis in the dermis. Serum copper was low in all the goats. Micronutrients (Cu and Zn) were within permissible level in feed samples. Despite adequate dietary copper levels, reduced serum copper concentration along with clinical, haematological and histopathological findings indicates impaired absorption and/or metabolism of copper is suggestive of secondary copper deficiency.

Keywords: Goat; copper deficiency; Achromotrichia; hyperkeratosis; parakeratosis

Introduction

Copper is a trace mineral essential for iron metabolism, the formation of elastin and collagen, production of melanin and maintaining the integrity of the central nervous system (Mahmoud et al., 2015). Primary copper deficiency arises from inadequate dietary intake, while secondary copper deficiency results from factors that impair copper absorption or metabolism. In small ruminants, shift from extensive to semi-intensive system of management has brought many new conditions (Vaidya et al., 2025) eg. Sulfur-metabolizing bacteria in the rumen exacerbate this condition by amplifying the antagonistic effects of dietary sulfur (Radostits et al. 2016). Neurological disorders are the most commonly reported consequence of copper deficiency in goat kids, while dermatitis occurs less frequently (El-Khaiat et al. 2012). Clinical signs of copper deficiency-associated dermatitis include alopecia, achromotrichia, itching, pale conjunctiva and emaciation (Cerone et al. 2000). The present report records the copper deficiency-associated dermatitis in goats reared under semi-intensive system in an organized farm, which was confirmed through laboratory investigations of blood mineral profiling, feed analysis and histopathology of skin.

Materials and Methods

The present investigation was conducted in a private goat farm located at Orathanadu, Thanjavur, Tamil Nadu (longitude and latitude of Orathanadu is 10.6252° N and 79.2550° E). The goat flock size of 45 Thanjavur black goats of one to nine years old females reared under semi-intensive system. History showed that the twenty herd mates exhibited chronic, intermittent skin lesions and had poor response to treatment with antibiotics, ivermectin and topical ointments since six months. Further, affected animals had recurrence of cutaneous lesions. All animals in the farm were maintained on a regular deworming and vaccination schedule. All the goats were regularly fed with commercial goat feed.

Among the affected animals, seven goats were randomly selected for detailed investigation. Samples such as skin scrapings and faeces were collected for parasitological studies. Whole blood and serum were collected for haematological and biochemical analysis by using automatic haematology (EXIGO®, Sweden) and biochemical auto analyzer (Selectra PRO XS®). Punch skin biopsies were collected in 10% formalin for histopathology. Skin biopsies were processed as per standard paraffin-embedding techniques and tissue sections of 4-5 µm thickness were prepared using manual microtome (Leica) and stained with hematoxylin and eosin (Bancroft and Layton, 2019a). Duplicate sections were subjected for Masson trichrome staining technique to demonstrate collagen (Bancroft and Layton, 2019b). Serum and feed (Randomised feed samples) minerals (copper and zinc) were determined by using atomic absorption spectrophotometer (Thermo Fisher Scientific India) available at Department of Animal Nutrition, Veterinary College and Research Institute, Orathanadu, Thanjavur.

Results and Discussion

On general clinical examination, the affected goats were thin with poor body condition and pale mucous membranes. Dermatological examination revealed generalized, scattered patchy alopecia in most of the animals, along with a rough hair coat, dry, scaly, crusty skin lesions and depigmentation of hair (Fig. 1 & 2). Examination of skin scrapings revealed no evidence of parasites/ parasitic stages and or bacteria and fungus suggestive of pathogenic significance respectively.

Whole blood analysis showed reduced hemoglobin and packed cell volume in all seven goats (Table 1) which was in consistent with anemia. Copper plays an indispensable role in incorporation of iron molecule into the heme during haemoglobin synthesis. Conversely, copper deficiency disrupts heme synthesis which resulted in hypochromia and microcytosis (Smith and Sherman, 2009; Almeida et al. 2022). Additionally, impaired maturation of erythroid precursors, defective erythropoiesis and increased oxidative stress, exacerbates anemia in copper deficiency (Ahmed et al. 2009). Elevated red cell distribution width (RDW) observed in the present study indicated the marked anisocytosis of erythrocytes and strongly suggested that the compensatory regenerative response by the bone marrow secondary to anemia impelled by copper deficiency as previously reported by Cerone et al. (2000). These findings were in accordance with earlier reports which emphasized that the copper as a micronutrient to confer essential role in hematopoiesis and the regulation of oxidative stress. Leukogram abnormalities such as leukocytosis and neutrophilia observed in the study suggested that an underlying inflammatory and immune response might contribute for pooling of leucocytes in peripheral blood. The above findings might be due to the dermatitis, associated with copper deficiency (Mahmoud et al., 2015 and Almeida et al. 2022).

Serum liver enzymes such as alkaline phosphatase (Mean and SE: 433.04±39.68 U/L) and aspartate aminotransferase (Mean value of 182.01±78.61U/L) were increased and serum protein (5.9mg/dl) and calcium (8.1 g/dl) level was found to be decreased in one goat. However, other serum biochemical values BUN, creatinine,

Table 1. Hematological changes recorded in goats with secondary copper deficiency associated dermatitis.

S. No.	Animal no.	RBC (10^6/µl)	HGB (g/dl)	HCT (%)	MCV (fl)	MCH (pg)	MCHC (g/dl)	RDWc (%)	WBC (10^3/µl)	LYM (%)	MON (%)	NEU (%)	Interpretation
1.	ONF 96	**7.96**	**4.1**	**10.21**	**13**	5.2	40.6	**33.2**	**17.9**	23.5	0.5	**76**	Low Hb, PCV & elevated RDW Microcytic hypochromic anaemia Leukocytosis, neutrophilia
2.	ONF127	**4.69**	**3.3**	**8.36**	18	6.9	38.9	**45.1**	12.93	80.4	1.1	18.5	Low Hb, PCV & elevated RDW Microcytic hypochromic anaemia
3.	ONF100	**1.99**	**3**	**8.76**	44	15.1	34.3	24.9	7.21	80.5	0.5	19	Anaemia
4.	ONF110	11.04	**6.4**	**16.97**	**15**	5.8	37.6	**33.1**	**22.65**	7.3	0.6	**92.1**	Anaemia, leucocytosis, neutrophilia
5.	ONF116	8.29	**6.5**	**18.79**	23	7.9	34.8	**33.3**	**17.63**	53.8	1.1	**45.1**	Anaemia Leukocytosis, neutrophilia
6.	ONF124	12.86	**6.1**	**12.81**	**10**	4.7	47.3	**40.8**	7.38	89.2	0.7	10.2	Low Hb, PCV and elevated RDW Normocytic hypochromic anaemia
7.	ONF164	10.84	**7.5**	**13.71**	**13**	6.9	54.8	**39.8**	9.71	47.1	0.5	**52.3**	Low Hb, PCV and elevated RDW Microcytic hypochromic anaemia.

Table 2. Serum and feed copper and zinc levels and skin histopathology of goats with associated dermatitis**.**

S. N.	Animal no.	Serum copper (mg/L)	Serum zinc (mg/L)	Histopathology
Serum				
	Normal	0.67mg/L	0.65-2.50mg/L	
1.	ONF 96	0.644	1.25	Hyperkeratosis
2.	ONF127	0.206	2.58	Hyperkeratosis with subacute dermatitis
3.	ONF100	0.453	1.27	Hyperkeratosis and acanthosis, subacute dermatitis with mixed inflammatory cells
4.	ONF110	0.228	0.83	Hyperkeratosis, parakeratosis, subacute dermatitis with mixed inflammatory cells
5.	ONF116	0.532	0.66	Hyperkeratosis, subacute dermatitis with mixed inflammatory cells
6.	ONF124	0.513	1.19	Hyperkeratosis with inflammatory cells
7.	ONF164	0.375	1.32	Hyperkeratosis with mixed inflammatory cells
Feed				

Type of feed		Feed Copper (ppm)	Feed Zn (ppm)
Goat Adult	:	12.88 ppm	38.1 ppm
		10 to 25 ppm on a dry matter basis – as per NRC recommendation for goats	30 to 40 ppm on a dry matter basis per million (ppm) on a dry matter basis – as per NRC recommendation for goats

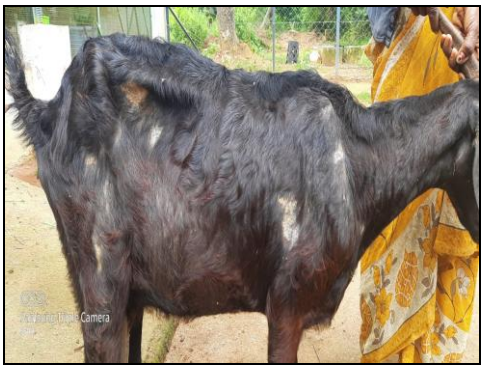

Fig. 1. Dry and rough hair coat, scattered patchy areas of hair loss with scaly and crusty skin lesions in Thanjavur black goat due to Secondary copper deficiency.

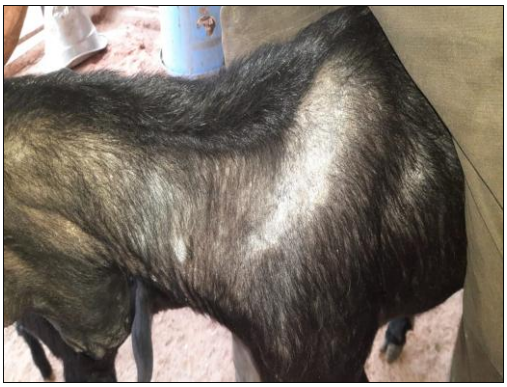

Fig. 2. Thin hair coat, dry scaly and crusty skin lesion over the shoulder blade, lateral neck, achromotrichia in Thanjavur black goat due to Secondary copper deficiency.

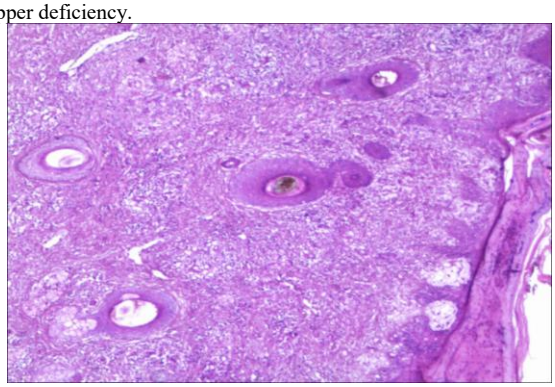

Fig. 3. HP: Skin: Diffuse sub-acute dermatitis in Thanjavur black goat due to Secondary copper deficiency, H&E x40.

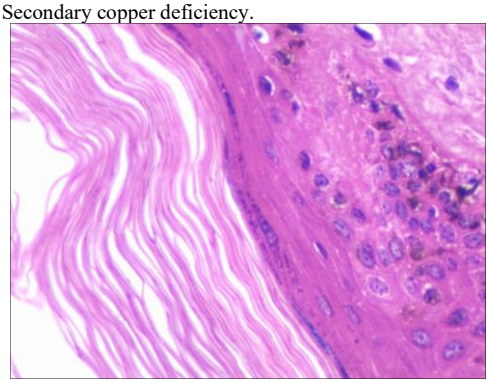

Fig. 4. HP: Skin: Orthokeratotic in Thanjavur black goat due to Secondary copper deficiency, H&E x400.

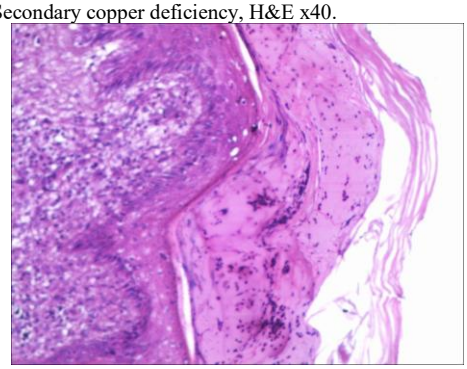

Fig. 5. HP: Skin: Parakeratosis in Thanjavur black goat due to Secondary copper deficiency, H&E x400.

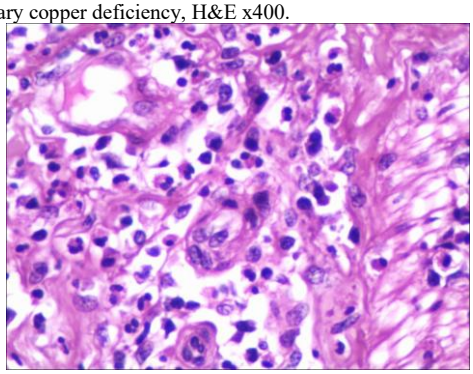

Fig. 6. HP: Skin: Perivascular inflammatory cell (eosinophils and lymphocytes) infiltrations – Subacute dermatitis in Thanjavur black goat due to Secondary copper deficiency, H&E x400.

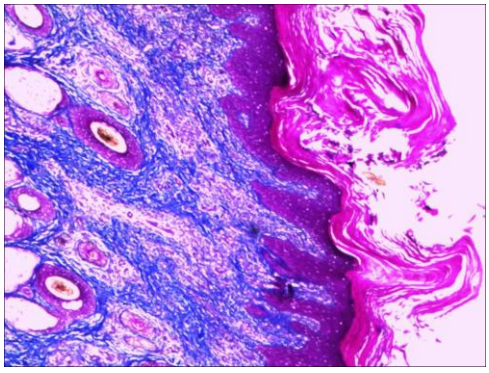

Fig. 7. HP: Skin: Orthokeratotic hyperkeratosis and subacute dermatitis in Thanjavur black goat due to Secondary copper deficiency, Masson's trichrome stain x40.

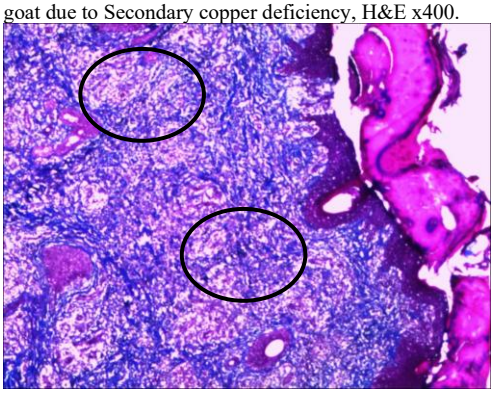

Fig. 8. HP: Skin: Parakeratosis and subacute dermatitis with areas of collagen breakage and lysis (circle) in Thanjavur black goat due to Secondary copper deficiency, Masson Trichrome stain x100.

protein, albumin, ALT, calcium, phosphorous and magnesium were within reference range. Aupperle et al. (2001) reported that liver damage (hepatitis and or cirrhosis) could impair copper metabolism which often leads to decreased ceruloplasmin synthesis, impairing copper transport and hindering its absorption in the gastrointestinal tract. Eventually copper deficiency and subsequent anemia supervenes.

Serum copper levels in the affected goats were recorded at lower than that of normal reference value of 0.67 mg/L for goats whereas serum zinc levels (Table 2) were within the reference range (0.65–2.50 mg/L) (Ivans et al., 1990). Copper was a vital micronutrient involved in numerous enzymatic processes, including iron metabolism, connective tissue formation and immune function. Copper deficiency in goats has been associated with anemia, ataxia and reduced growth rates in Omani goat breeds (Osman et al. 2008). El-Khaiat et al. (2012) observed that experimentally induced copper deficiency in goats resulted in hair depigmentation, steely appearance and easy detachment of black hair. They also reported emaciation and significant weight loss which became evident after 12 weeks of induced copper deficiency. Tyrosinase, a copper dependent enzyme involved in melanin production, which results achromotrichia (Frank, 1998; Aupperle et al., 2001). The above documented evidence appeared to be similar with the clinical findings of the present investigation and corroborate that the observed skin lesions were attributable to copper deficiency.

Histopathology of skin biopsy samples revealed acanthosis (Fig. 3), orthokeratotic hyperkeratosis (Fig. 4) and parakeratosis (Fig. 5) of superficial stratified epithelium. Further, dermis revealed subacute dermatitis with mixed inflammatory cells. Inflammatory cells were throng around the blood vessels and composed predominantly of eosinophils followed by lymphocytes and neutrophils (Fig. 6). Macrophages and plasma cells were fewer in the lesions. Collagen in the dermis were haphazardly arranged and showed scattered areas of breakage and lysis. Masson trichrome stain revealed massive collagen bundles which were interspersed by inflammatory cells (Fig. 7). Multiple foci of collagen breakage and lysis were evidenced (Fig. 8). The above histopathological changes were in accordance with earlier reports (Aupperle et al. 2001; Varghese et al. 2021). Aupperle et al. (2001) reported that the experimental copper and chromium deficiency in black haired goats showed achromotrichia and dermatitis with orthokeratotic hyperkeratosis and eosinophilic perivascular infiltrations. Though the above documented observations were in line with the present investigation, parakeratosis recorded in the present study might be due to chronic deficiency of the illness induced by copper.

Non-pruritic lesions with loss of collagen reported by Varghese et al. (2021) in human beings with copper deficiency were in parallel with the present study. Further the role of copper containing enzymes particularly lysyl oxidase mediate cross-linking of connective tissue proteins such as dermal collagen and elastin fibers (Aupperle et al., 2001) was found to maintain structural integrity of skin. Deficiency in lysyl oxidase leads to weakened connective tissues, epidermal atrophy and reduced skin elasticity. Foci of dermal collagen breakage and lysis with inflammatory cell accumulations of the present study suggested that lysyl oxidase play pivotal role in collagen synthesis, maturation and maintenance of the skin. Additionally, copper deficiency found to impairs keratinocyte maturation and differentiation, leading to incomplete keratinization and hyperkeratosis (Aupperle et al., 2001). Reduced copper-dependent ceruloplasmin and superoxide dismutase activity contributes to oxidative tissue damage and inflammation which further exacerbating skin lesions and parakeratosis (Cerone et al. 2000). Feed analysis revealed copper (12.88 ppm) and zinc (38.1 ppm) levels were within the permissible ranges (copper: 10–25 ppm; zinc: 30–40 ppm on a dry matter basis as per NRC recommendations for goats) shown in table 2. Despite adequate dietary copper levels, reduced serum copper concentration along with clinical, hematological, and histopathological findings indicates impaired absorption and metabolism of copper. Therefore, the present case was diagnosed as secondary copper deficiency associated with dermatitis in goats.

Conclusion

This study investigated chronic dermatitis associated with secondary copper deficiency in Thanjavur Black goats reared on a semi-intensive system. Affected goats were showed chronic skin lesions, including alopecia, depigmentation and poor body condition, alongside anemia. Diagnostics revealed microcytic hypochromic anemia, low serum copper levels (<0.67 mg/L), and elevated liver enzymes. Histopathology identified hyperkeratosis and subacute dermatitis, while feed analysis showed adequate dietary copper, suggesting impaired copper absorption or metabolism. This study highlights the clinical and pathological implications of copper deficiency in goats, emphasizing the need for regular trace mineral monitoring and management to prevent similar outbreaks.

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Conflict of interests There is no conflict of interest.

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