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PARAKERATOSIS IN SOME FARM ANIMALS

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Abstract. Due to the continuous growth and renewal of the integument, nutritional deficiencies translate early on into clinical skin signs. Zinc is an essential trace element involved in numerous metabolic processes. Deficiency, induced mainly by inhibition of absorption by some competitors (excess calcium and other bivalent ions, moldy feeds rich in phytic acid and phytates), leads to significant economic losses. Zinc deficiency expresses itself clinically relatively late in the disease, with parakeratosis of the epidermis (the visible part of the iceberg) and of the epithelium of the keratinized mucous membranes: buccal, esophageal, forestomach, esophageal portion of the gastric mucosa, vaginal, immunosuppression, growth retardation, reproductive disorders etc. We have recently encountered an episode of parakeratosis in young pigs, which started 3 weeks after changing the feed with a cheaper (poor quality) one.

Keywords: cattle, pigs, parakeratosis, zinc deficiency, immunosuppression

The integument is a signal for dietary misconduct, responding promptly to both quantitative and qualitative deficiencies in nutrient intake and to disorders of metabolic utilization of nutrients: malabsorption and maldigestion syndromes following chronic intestinal, hepatic, pancreatic diseases, etc. Nutritional and metabolic deficiencies manifest themselves clinically polymorphic.

Parakeratosis is a dyskeratosis due to the inability to form alphakeratins due to lack of keratohyalin sulfation. As a result, nucleated keratinocytes, devoid of keratohyalin, reach the skin surface and become basophilic in colour. The most important cause is zinc deficiency.

In addition to congenital parakeratosis in newborns, acquired nutritional parakeratosis is much more common in practice.

Parakeratosis is of greatest practical importance in piglets and calves, but has also been described in pet carnivores. The primary etiologic factor in the occurrence of parakeratosis is **zinc deficiency**. Even congenital parakeratosis is referred to by some authors as 'hereditary zinc deficiency' or 'hereditary zinc dysmetabolism' (Yager and Scott, 1993). However, the disease is rarely caused by primary zinc deficiency. In the majority of cases, secondary deficiency occurs due to the excessive presence of zinc antagonists in the ration: calcium, phosphorus, copper, molybdenum, molybdenum, iron. These bivalent ions competitively inhibit intestinal absorption of zinc. The tendency of farmers to unduly supplement calcium and phosphorus in the ration in order to prevent rickets appears to be one of the most important causes of parakeratosis (Onțanu et al., 1987). It is reported that at 1.3% calcium in the ration of fattening pigs, weight gain is strongly inhibited and 90% of the animals develop parakeratosis. Phytic acid and phytates - zinc chelators - also play an important role. They are found in large quantities in fresh maize, mouldy feed (some fungi produce phytic acid) and soybean meal. In the case of phytates, there seems to be a vicious circle of zinc deficiency: phytates chelate zinc, and on the other hand, zinc deficiency leads to a decrease in the activity of intestinal phytase, an enzyme that inactivates phytates (Onțanu et al., 1987).

The deficiency exerts its effect through deficiencies in the enzymes in which zinc is contained: RNA polymerase, carbonic anhydrase, serum alkaline phosphatase. Skin epithelial parakeratosis occurs. In severe forms, parakeratosis of **mucosal epithelium** also occurs, especially buccal, pharyngo-esophageal, forestomach and cardia part of the abomasum. In addition, thymus atrophy occurs.

Mucosal lesions and atrophy of the thymus favor the development of life-threatening digestive and respiratory intercurrent infections.

Symptomatology. In calves the disease appears at 4-6 weeks of age. It begins with growth cessation. The hair becomes dull, coarse. The parakeratotic lesions appear initially peribucally around the nostrils, then submandibular, on the ventral aspect of the neck, the inner aspect of the thighs and in the regions of greater mechanical stress. The lesions consist of gray or open-brown, very itchy, gray or brownish crusts, under which the skin is hairless. Removal of the scabs leaves the area bleeding. The thickening and lack of elasticity of the parakeratotic skin causes cracks and fissures in the more mechanically stressed areas, which are entry points for infectious agents. Wound healing is delayed.

In association with cutaneous parakeratosis, parakeratosis of the epithelium of the digestive and respiratory mucous membranes etc. Is produced. This favors the development of bronchopneumonia and gastroenteritis, which hasten the lethal end of the disease. There is also thickening of the joint ends (Anton et al, 2010).

Zinc concentration is low: in serum 30-80 micrograms/100 ml instead of 100-140, in bones 60-80 ppm instead of 90-105, in liver 100-115 ppm instead of 125-170. Serum alkaline phosphatase is also decreased to 17.1 IU instead of 135 IU/l.

In adult cattle a parakeratosis of the base of the tail and the dorsal surface of the hindlimbs is described. It can be confused with chorioptic mange (Scott, 2007). Parakeratosis of the epithelium of the vaginal mucosa causes reproductive disorders. Zinc defficiency is also involved in pathogenesis of subclinical mastitis (increasing of somatic cells from milk) (Anton et al., 2007).

Spontaneous parakeratosis is rare in lambs. It has been produced experimentally and is characterized by: thin, fragile, easily depilated wool and dystrophies of the horny tissues, resulting in horn droop or keratinous buds at the horn site in hornless breeds (Yager and Scott, 1993).

In swine the disease occurs most commonly in the 2-4 month-old fattening piglets, but may also occur in the neonatal period due to sow hypozynchosis. Initially erythematous macules appear on the abdomen, inner surface of the thighs, periocular, peribuccal, ears, scrotum, tail, periarticular. Progressively, papules covered with dry, brownish-black, dry scabs appear. Due to pruritus, in some areas the crusts are removed, but they create entry portals for bacteria and fungi.

We have recently encountered an episode of parakeratosis in young pigs, which started 3 weeks after changing the feed with a cheaper (poor quality) one. We presume the intervention of zinc antagonists, including from moldy corn, since in addition to cutaneous parakeratosis (Fig. 1, 2), digestive disorders (some with lethal end) and some manifestations of estrogenism, characteristic of mycotoxicosis produced by zearalenone, elaborated by *Fusarium* spp. (Paul, 2001), were also observed.

Mucosal parakeratosis, associated with intercurrent digestive infections causes vomiting, diarrhea, anemia, weakness, taste perversion. Predisposes to esophago-gastric ulcer.

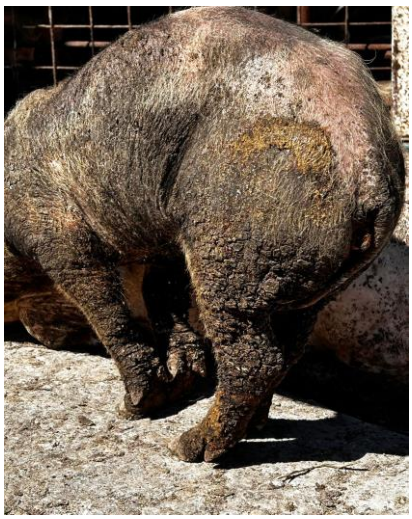


Figure 1. *Generalized cutaneous parakeratosis and deep scratching lesions*

Histologic examination is made from unulcerated areas. Moderate or severe acanthosis and multifocal or diffuse parakeratosis, both epidermal and follicular, are found. Extension of parakeratosis into the follicular infundibulum is considered characteristic for zinc deficiency. In the dermis, superficial perivascular dermatitis with polymorphic, often eosinophil-rich infiltrate is evident (Gross et al., 1992). Bacterial complications cause pyoderma lesions.

The **diagnosis** of parakeratosis is based on the corroboration of anamnestic and clinico-pathological data with chemical analysis of the ration, determination of zincemia, serum alkaline phosphatase and zinc in various tissues. Histologic examination of the skin after biopsy provides the most useful diagnostic data. Interpretation of the lesions must, however, be done with care, as parakeratosis of various degrees occurs secondary to most chronic dermatitis.



Figure 2. *Generalized cutaneous parakeratosis. Deep grooves leaving bleeding wounds exposed to infection*

Differential diagnosis from hyperkeratosis - which clinically manifests similarly to parakeratosis - is based on histopathologic examination.

Of particular practical importance is the differential diagnosis of parakeratosis from parasitic pruritic and hyperkeratotic dermatitis (mange, dermatomycoses), which is based on direct parasitologic examination after scraping off the crusts.

Prophylaxis and treatment. To prevent parakeratosis, it is necessary to ensure an optimal balance between zinc and the factors conditioning its bioavailability.

In calves, the administration of 100 mg zinc/animal/day cures parakeratosis within 14 days. In adult bulls, parakeratosis is cured when 250-500 mg zinc/animal/day is administered daily or when salt lick containing 1-2 % zinc is administered.

In piglets, neonatal parakeratosis can be prevented by feeding sows 1 g of zinc daily during the last 4 weeks of gestation. In fattening sows, in addition to avoiding excess zinc antagonists in the feed, 500 mg zinc sulphate/animal/day can be added to the diet. The dose is also effective for curative purposes (Mitrea et al., 2006).

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