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التكلس الجلدى في حلمات ثدى الأبقار والجاموس

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أجريت دراسة مستغيضة على ثلاثة حالات تكلس تحت جلد حلمات ثدى الأبقار والجاموس فــــى المجمهورية مصر العربية ، تم الدراسة ميكروسكوبيا وهستوكيميائيا ونوقشت نتائج مكان التكلس واسبابــه وكيفية حد وثه وكذا العلاقة بين تكلس النسيج تحت الجلد في حلمات ثدى الا بقار والجامــــوس والتكلس الدائرى في الكلاب وتكلس السرطان والظلهارى والتكلس الشامل والتكلس الشامل والتكلس المستوطن كما نوقش دور النسيج العضلي وعطيات الحليب في نشو عثل هذا التكلس.

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CALCINOSIS CUTIS IN BOVINE TEAT (With 2 Figures)

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SUMMARY

Three cases of calcinosis cutis in subcutaneous tissue of bovine teats are diagnosed. The lesions are micromorphologically and histochemically studied. Location, astiology, Pathogenesis, as well as relation of our findings to calcinosis circumscripta, calcifying epitheliomas, calcinosis universalis, enzootic calcinosis and other similar conditions are discussed

INTRODUCTION

Calcinosis cutis, i.e., calcification of the skin is known to occur in animals in three more or less distinct forms including presternal calcification in cattle and sheep, calcinosis circumscripta and calcinosis universalis in dogs (JUBB and KENNEDY, 1963). Calcium deposition in these three forms may occur in the connective tissue of brisket and sternal cutis in presternal calcification, of the tongue, lip, leg and neck in calcinosis circumscripta (DOUGLAS and KELLY, 1966; HOWELL and ISHMAEL, 1968), and in many parts of the body in calcinosis universalis (JUBB and KENNEDY, 1963). Deposits have also been suggested to occur in epidermal apocrine sweat glands (CHRISTIE and JABARA, 1964; CORDY, 1967, KUNZE, 1966) or epithelioma in the skin (SMITH and JONES, 1957, NIELSEN and COLE, 1966). Subcutaneous muscles are principally affected in calcinosis universalis (JUBB and KENNEDY, 1963).

Pathological conditions known as enzootic calcinosis (DIRKSEN et al., 1970) enteque secs (ECKELL et al., 1960), espichaments (PARADI and DOS SANTOS, 1947, naalehu (LYND et al., 1965), Manchester wasting disease (ARNOLD and FINCHAM, 1950) are known to be associated with widespread calcinosis in different organs of the body in cattle.

In the present work three cases of calcification in the subcutaneous tissue of the teat are studied histopathologically, the aetiology and pathogenesis of this affection is discussed.

MATERIAL and METHODS

The teats used in this study were obtained from cows and buffaloes slaughtered at abbatoirs in asurveyfor studying bovine teat lesions in Egypt. On microscope examination of the teats, three cases showed subcutaneous calcification. Paraffin sections were stained with haematoxylin and eosin, periodic acid-Schiff technique (PAS), Van Giesons, Trichrome, alizarin red, Von Kossa, alcian blue, toluidine blue, as well as decalcified sections were carefully examined.

RESULTS

In the teat of these three animals, lesions could not be detected grossly. Microscopically, straticied squamous epithelium of the epidermis showed mild degree of hyperkeratosis and acanthosis. Areas of calcification occurred in the intermediate layer of the wall of the teat, which is a well vascularized layer rich in collagen bundles and muscle fibers (Fig. 1). Many of muscle fibers and bundles were selectively involved in the process of calcification. According to the degree of affection, deposits in the muscle fibers may appear as amorphous, dustlike particles

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distributed along the length of the myofibrils in the early stage. Calcification commonly occurred in degenerating muscle fibers. Degeneration firstly appeared in the form of perinuclear cytoplasmic vacuoles which enlarge, extend between the myofibrils seperating and disrupting them and eventually leads to fibrinolysis so that only the sarco-lemmal tube remained visible in sections stained with H. and E. when calcification occurred in these muscle fibers the sarcolemmaland the area directly beneath it usually showed dense deposits of dark-stained calcium particles. The contractile substance at the interior of the fibril or its breakdown products did not appear to have an affinity for calcium salts. It did not stained with any of specific stains of calcium applied in the present study, was lightly acidophilic by H. and E., metachromatic and PAS positive.

Conglomerates, which were occasionally lamellated, involving a muscle bundles were seen in this layer. Fibers at the periphery of the bundles were severely affected and in cross sections these bundles appeared to be surrounded either completely or in completely with dark basophilic masses of calcified bibers (Fig. 2). Decalcified sections stained with H. & E. and undecalcified ones stained with Trichrome stain, in which the deposits took a light purple colour, clearly demonstrated that the calcified tissue was mainly muscle fibers. Collagen or elastic fibers in this layer of the dermis and glandular structures in the wall of the teat were free from any calcium deposits. Wall of blood vessels, as well did not show any calcification. No inflammatory cell reaction was observed in the affected areas or covering epithelium.

DISCUSSION

Various factors are responsible for calcification of soft tissue. In presternal calcification, the process is a dystrophic calcification of chronically inflammed connective tissue, probably as a result of repeated trauma (JUBB and KENNEDY 1963). Aetiologically the essential nature of calcinosis circumscripta in dogs is unknown.

SANDERSLEBEN (1958), CHRISTIE and JABARA (1964) and HOWELL and ISHMAEL (1968) differentiated between two conditions. The first, a calcinosis circumscripta, (kalkgicht, calcium gout, multilocular subcutaneous granuloma), is characterized by multilocular calcified foci surrounded by giant cells and fibrosis. The second type, calcifying epithelioma (hair matrixoma, epithelioma of Malherbe, trichoepithelioma), consists of sheets resembling basal cells or hair matrix epithelium. THOMPSON et al., (1959) thought that prolonged, repeated trauma was the most probable cause of the lesion. CHRISTIE and JABARA (1964) and CORDY (1967) suggested that the lesion was the result of dystrophic calcification in cystic and disorganized apocrine glands, minor trauma has been considered as a factor which evokes the atypical hyperplastic and secretory response of the apocrine glands. HOWELL and ISHMAEL (1968), however, found no evidence of transition from normal or cystic glands to calcinosis circumscripta. In the present study, no features of chronic inflammation were found in the calcified area at the wall of the teat and, moreover, calcified tissue consisted of muscle fibers having no relation to structuresdescribed in calcinosis circumscripta or calcifying epithelioma.

Implication fo poisonous plants has been implicated as a cause of chronic affections associated with wide-spread calcification of body organs. Poisoning with Solanum malacoxylon, whose leaves contain an active principal with an effect similar to dihydrotachysterol, was suggested as the cause of enteque seco in Argentine, CARILLO and WORKER, 1967). As well, poisoning with a solanum was thought to be the cause of espichamento in Brasil and poisoning with cestrum diurnum, whose leaves contain a factor (s) have an action similar to that of 1,25 dihydroxycholecalciferol which is the biologically active form of vitamin D3, was implicated tobe a cause of similar condition in Florida-cattle (WASSERMAN et al., 1975, KROOK et al., 1975). Naalehu disease in Hawii was related to an imbalance of mineral intake, i.e., deficiency of magnesium and phosphorus associated with high intake of calcium and pottasium *. Manchester wasting disease was suggested to be due to a combind effect of feeding a substance having a vitamin D action with imbalance of calcium, manesium and phosphorus (ARNOLD, 1969). No similar conditions were reported to occur either in cattle or buffaloes in Egypt. Moreover, calcification of the skin or subcutaneous tissue was not described in these diseases.

^{* (}MANSTON, PAYNE 1964).

CALCINOSIS CUTIS IN BOVINE TEAT

In the present study, the process of calcification was found mainly to be restricted to muscles in the intermediate layer of the wall of the teat. This lesion is thought to be a dystrophic calcification in which calcium is opt to be deposited in degenerated and necrotic muscle fibers. Mild trauma through continuous mechanical action during milking may be responsible for these degenerative changes and necrosis. Infection and inflammatory changes can be excluded as a probablecause on the basis of absence of inflammatory exudate in the area. The role of generalized disturbance of calcium metabolism either due to imbalanced ration or other factors, is hardly to be stated and further studies are essential. Calcification of the teat wall is an important lesion due to pain that the animal may suffer during the process of milking and that the lesion may initiate inflammatory changes in this part of the teat that may lead to serious complication to the potency of the teat canal and orifice.

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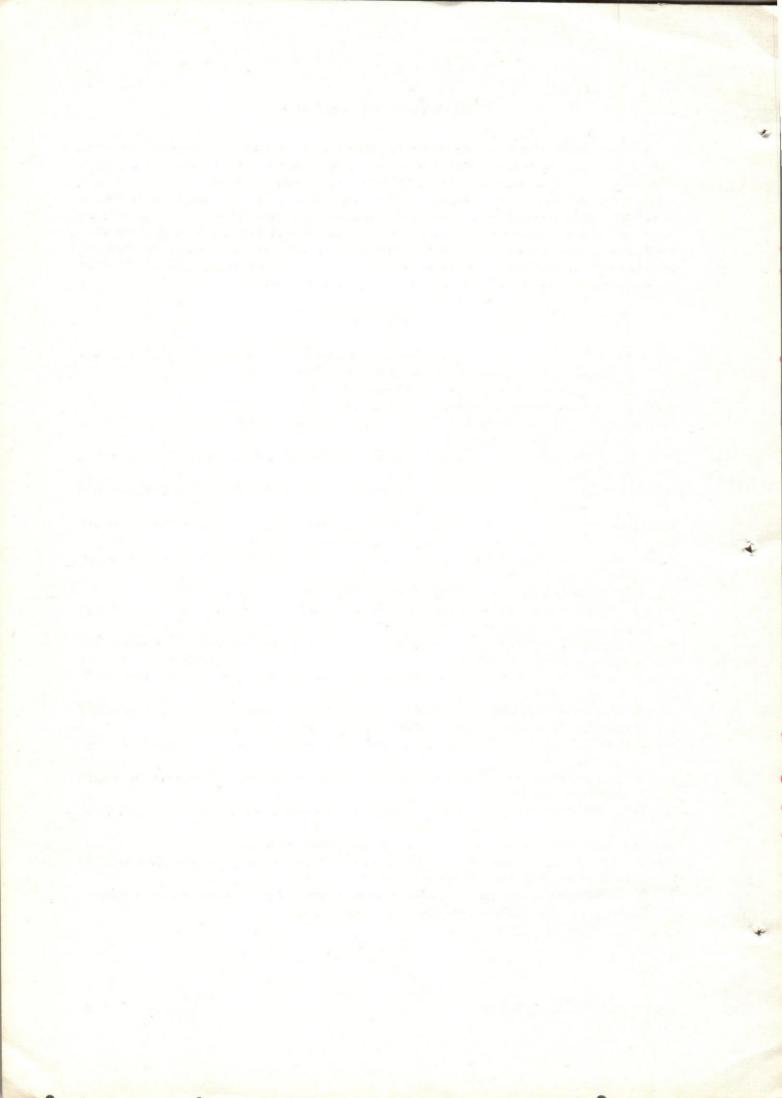
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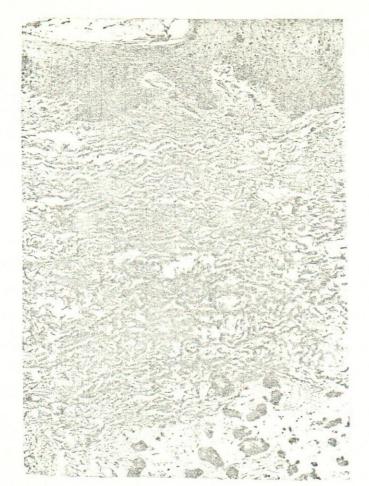


Fig. (1)

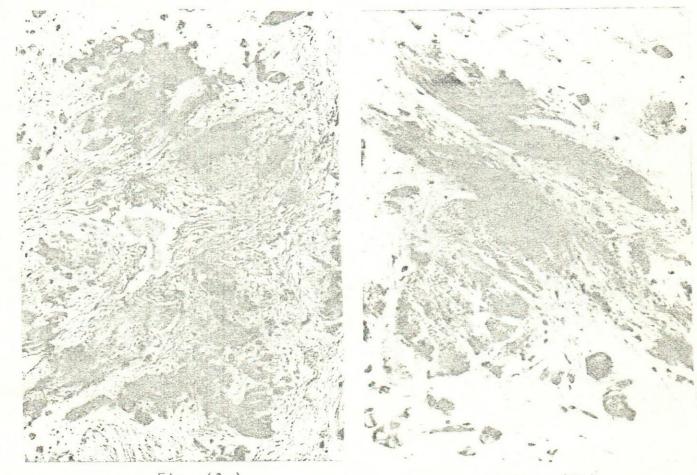


Fig. (2a)

Fig. (2b)

