الضعف العضلى فى الحملان

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فحصت ثلاثة حملان من قطيع به أعراض الضعف العضلى بقرية مسرع بمحافظة أسيوط من الناحية المورفولوجيه والناحية الهستو باثولوجيه وكذلك النشاط الأنزيمى فى الأنسجة وكانت النتائج الباثولوجيه تشتمل على ترسيب الكالسيوم فى عضلات القلب وكذلك تتكرز مع نزيف فى عضلات الجسم الحركيه واللسان ـ أما الكبد والكلى والأورطى والعضلات كانت بهم تغيرات فى التمثيل الغذائى للدهون (استحالات شحميه) وكذلك تغيرات فى النشاطات الانزيميه . وقد دونت النتائج ونوقشت من الناحية الباثولوجية .

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ENZOOTIC MYOPATHY IN LAMBS

(With 9 Figures)

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SUMMARY

Three lambs of a flock affected with clinical manifestations corresponding to myopathy were subjected to pathological and enzyme histochemical studies. The results consisted mainly of subendocardial calcification, degenerative changes in the form of coagulative necrosis associated with haemorrhages in the skeletal muscles including the tongue, wide spread fatty changes in the muscles, liver, kidneys and aorta. The enzymatic activity of acid phosphatase, alkaline phsophatase, adenosine triphosphatase and succinic dehydrogenase in the skeletal muscles, heart, liver and kidneys were affected. The pathological findings were greatly similar to white muscle disease.

INTRODUCTION

Myopathy has been frequently described in claves and young sheep. Different factors have been associated with the occurrence of this affection in animals, these are either deficiency of selenium-vitamin E complex (COTCHIN, 1948, HOGUE, 1958, BLAXTER, 1962, BURTON et al., 1962, and GARDINER, 1962) or the presence of substances that could act to precipitate selenium-vitamin E deficiency, as, arsenic, mercury, cadmium and thalium

(VANVLEET, 1976). In addition BOSANQUET et al.,(1955)described a myopathy in male and female sheep in middle age and related it to scrapie disease of sheep. Also congenital origin for a few isolated cases of muscular dystrophy has been postulated by a number of invistigators (ROBERTS,1929, HUTT,1934, KIDWELL et al., 1952, MORLEY, 1954, HARLEY and DODD, 1957).

In Egypt the disease has been also recorded in claves of both foreign (semintal calf) and native breeds (buffaloes) by SOLIMAN et al., 1962 and BAYOUMI, 1973. Recently, preliminary investigation revealed the occurrence of the disease among a private sheep flock in Masrah, a village in the neighburhood of Assiut. The clinical symptoms consisted of weakness, stiffness and inablity to stand associated with loss of appetite and diarrhoea. In the present study pathological as well as histochemical investigations were carried out in a trial to elucidate the nature of this affection.

MATERIALS AND METHODS

The materials consisted of one dead lamb of an average age of 3 weeks and two lambs, one and fifteen days old, which were slaughtered after showing the symptoms. After gross examination, samples from the gastrocnemius and psoas muscles, tongue, heart, lung, liver, kidneys, brain, intestines and testes were processed for paraffin embedding,and sections were stained with haematoxylin and eosin stain. Frozen cryostat sections were prepared for demonstration of lipids and enzyme histochemical reactions of alkaline phosphatase acid phosphatase, (GOMORI, 1952), adenosine triphosphatase (BARKA and ANDERSON, 1963) and succinic dehydrogenase (WACHSTEIN and MEISEL, 1960).

Morphological findings:

At necropsy, the two lambs 15 days and 3 weeks age revealed that the gstrocnemius muscles of both sides showed haemorrhages while the psoas muscle of the lumbar region contained pale muscle bundles contrasted with darker ones. The diaphragm appeared normal. There were petechial haemorrhages along the coronary groove in the heart and subendocardial deposition of flocculent white masses which were numerous in the right than the left ventricles or the septum. The intermediate lobe of the lung showed a relatively large, dark reddish consolidated area . In the liver there were an irrigular subcapsular pale patches. The kidneys were slightly enlarged, pale and soft. There was also a mild catarrhal enteritis.

Microscopically, degenerative changes associated with or without haemorrhages were the prominent features in the muscles of the back, thigh, tongue and heart. In cross section of the skeletal muscles the changes were patchy in distribution within the fasciculi. While coagulative necrosis was more prominent around the ruptured blood vessels (Fig. 1), the other muscle fibres were pale stained and had pyknotic or karryorrhectic nuclei. Other type, probably more advanced,changes were observed in fasiculi showing no haemorrhages. These were characterised by shrinkage of the individual fibres, pyknosis of the nuclei, common occurrence of minute fat globules in the cytoplasm and an increased number of cellular elements in the areolar connective tissue seperating them, (Fig. 2). No multinucleated cells were observed and no cacification was found in the gastrocnemius and psoas muscle or muscles of the tongue

(Fig. 3). In the heart, the disappearance of many subendocardial muscle fibres was marked. Those which were still present were calcified and finely granular deposits were seen on the myofibrils giving the muscle fibre a dark basophilic granulated appearance in sections stained with heamatoxylin and eosin (Fig. 4). No calcification was found in the interstitial connective tissue. The deeply seated muscle fibres showed hyalinization and fragmentation with an increase of mononuclear cells in the interstitial connective tissue, (Fig.5). No haemorrage observed in the heart muscles. In the skeletal muscles, but not in the heart, sarcoblasts, single or arranged in strands, were observed sometimes between the degenerating muscle fibres These cells were smaller than the usuall muscel fibre, having a more esionophilic cytoplasm and a central oval to rounded nucleus.

In the liver, dilatation and engorgement of the central veins associated with centrolobular congestion of the sinsoids and distortion of the hepatic cells columns were found Moreover,perilobular fatty changes were observed. Infilteration of mononuclear cells at the hepatic triads could also be seen. The kidneys showed also degenerative changes. These were characterised by coagulative necrosis of the cells of the proximal and distal convoluted tubules, fatty changes in the cells of the striaght tubules (Fig. 6), hyaline casts and calcereous deposits in the lumina.

The dark reddish lesions found in the intermediate lobe of the lung at necropsy consisted mainly of hyperplastic peribronchiolar lymphoid tissues associated with severe haemorrhages. The surrounding alveolar septa were lined by an irrigular

cuboidal epithelial cells which many of them were desquamated inside the alveolar lumens.

In the aorta, the smooth muscle cells of the tunica media, which are normally elongated, became rounded and reoriented to be radially arranged along the course of the elastic lamellae. The cytoplasm of these cells appeared vacuolated, the nucleus was pushed aside toward the cell membrane, and the whole cell imparted the appearance of the foam cells, (Fig. 7). The elastic lamellae of the middle coat were disarranged and interrupted.

Results of enzyme-histochemical studies:

The degenerated area of the skeletal muscles and the subendocardial area of the heart showed an increased enzymatic activity of acid phosphatase (Fig. 8 a,b), while the activity of succinic dehydrogenase and adenosine triphosphatase was decreased or totally lost in some areas (Fig. 9). In the liver the most severely affected perilobular areas showed no acid phosphatase activity and the activity of succinic dehydrogenase, which is normally strong in this area, together adenosine triphosphatase activity were greatly inhibited. In the kidneys decreased activity of all of the four enzymes studied was observed in the convoluted tubules, while there was more active reactions in the striaght tubules with regard to acid phosphatase.

DISCUSSION

In the present study the lesions in the skeletal and cardiac muscles had a great similarity to those of white muscle disease recorded in sheep by many invistigators to be due to deficiency of vitamin E-selenium complex (BLAXTER et al. 1952, BLAXTER, 1953, HOGUE,1958, MUTH et al.,1959, MUTH and ALLAWAY, 1963). It is of interest to find that the muscles of the tongue also showed similar pathological changes, which is probably the main cause of inablity to eat manifested clinically as loss of apetite.

The absence of the lesions in the one day old lamb indicates that the disease develops mainly postantally. It has been stated that in a lamb born by a vitamin E deficient mother,the relative high proportion of unsaturated fatty acids in milk fat plays a considerable role, as the requirment of both selenium and vitamin E is greatly increased under such a condition (KOLB, 1969). One of the prominent features in the present case was the occurrence of fatty changes in the skeletal muscles, liver, kidneys and aorta. This may be correlated to the fact that the consistent protection of vitamin E is apparently related to its central role in protection of cellular memrane from lipoperoxidation, espically membranes rich in unsaturated lipids such as mitachondria, endoplasmic reticulum and plasma membranes (COMBS et al., 1975). Disturbances at the level of these membranes is shown in our study by changes of the enzymatic activities.

Extravasation of erthrocytes was observed in the present in the lung, liver and muscles, In consistence with (SOLIMAN et al., 1962), these haemorrhagic changes appeared to be due

to vascular disturbances and not to trauma as has been suggested by (BLAXTER et al., 1952). This can be emphasized by changes in large blood vessels, as the aorta, described in our study.

In chickens deficiency of vitamin E associated with the presence of high amount of unsaturated fatty acids may result in degenerative change in the central nervous system manifested in the form,of encephalomalacia. As stated by (KOLB,1969). insufficient supplementation of vitamin E leads to increased formation of peroxide radicles causing harmful effect in the organs espicially the brain which has normally a high metabolic activity for phospholipids, sphingomyelin, cholestrol, etc. The changes in the brain which were observed in the present study may be attributed to a similar mechanism.

No abnormalities could be found in the epithelia lining of the semineferous tubules of the testes in contrast to experimental hypovitaminosis in rodents in which necrosis of these cells had been recorded (SMITH and JONES, 1974).

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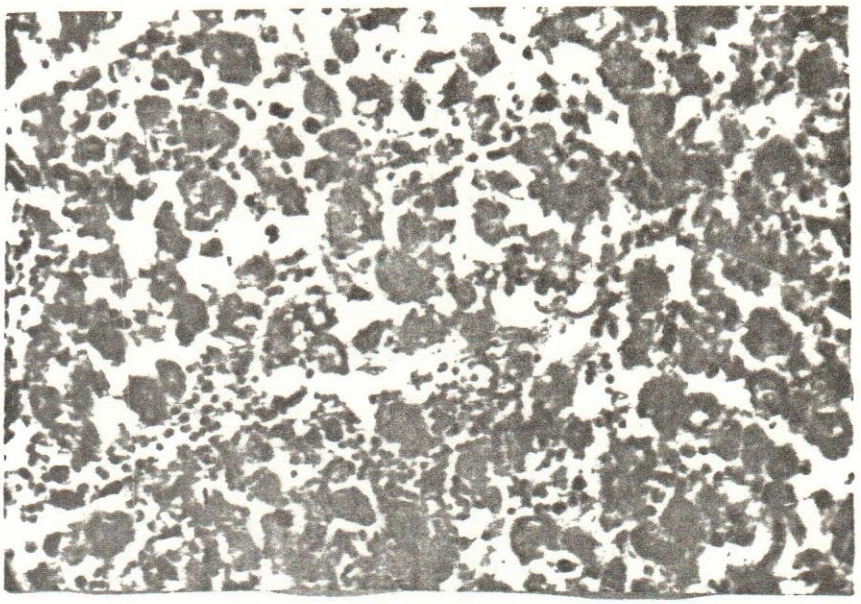

Fig.(1): Cross section of skeletal muscle showing coagulative necrosis and haemorrhages. (H. and E. X 400).

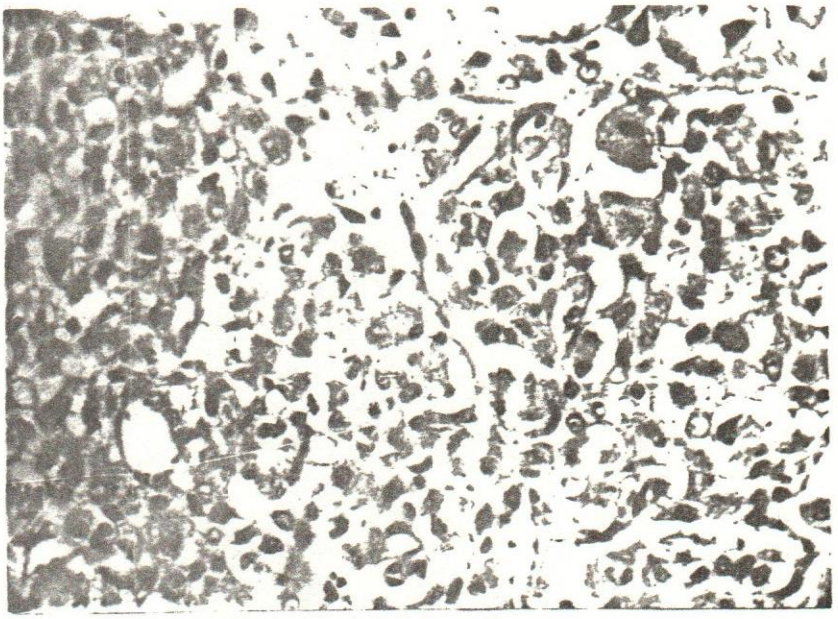

Fig.(2): Skeletal muscle showing shrinkage of individual fibres, pyknosis of their nuclei, minute fat globules in their cytoplasm and the cellular elements in the areolar connective tissue are increase ed.(H&E X 400).

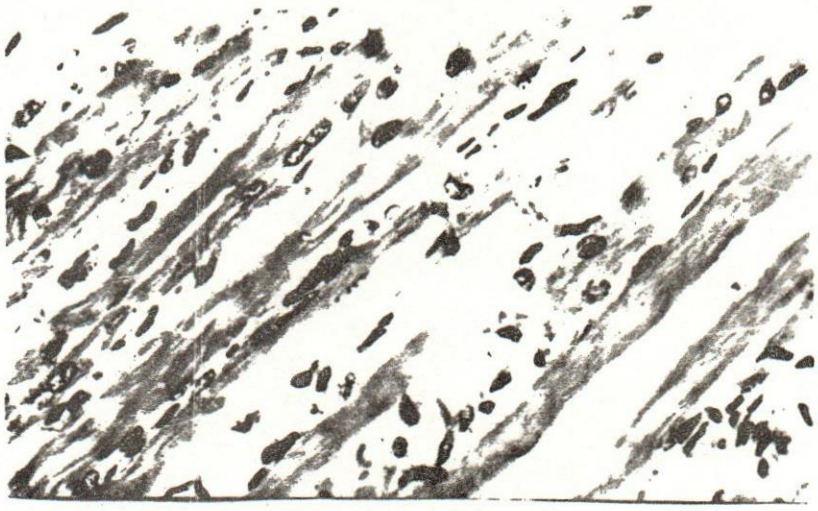

Fig.(3): Tongue showing coagulative necrosis without calcification.(H.&E. X 400)

Fig.(4): Heart showing dystrophic cacification (H.&E. X 400).

Fig.(5): Heart showing hyalinization and mononuclear cellular proliferation in the interstitium. (H.&E. X 400).

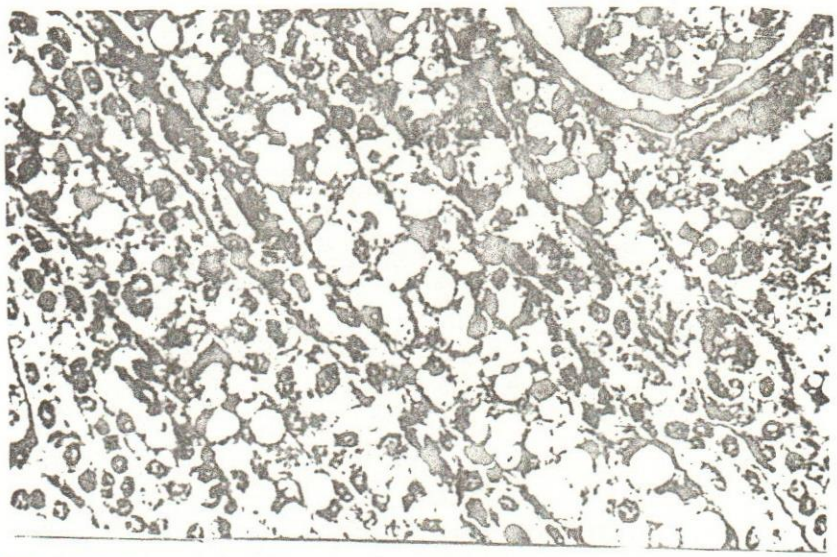

Fig.(6): Kidney showing fatty change in the striaght tubules. (H.&E. X 400).

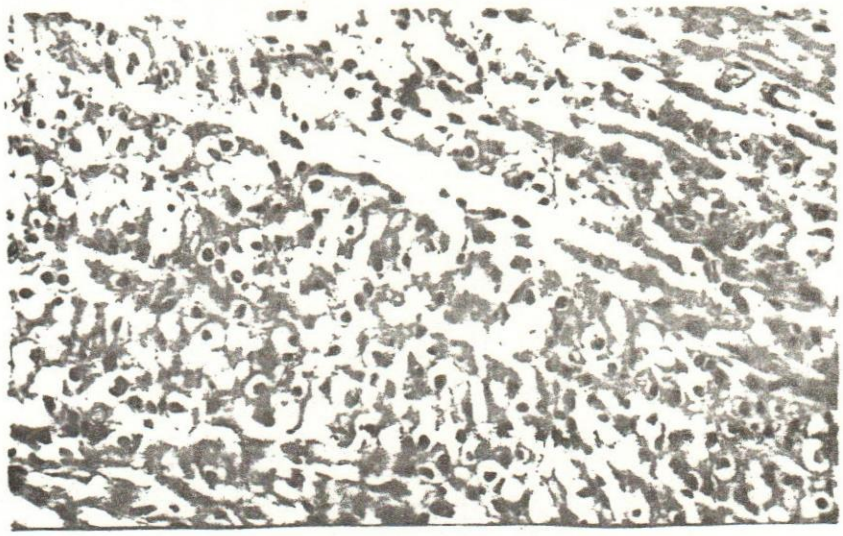

Fig.(7): Aorta showing reoriented smooth muscle cells which are laden with lipid; foam like cells. (H.&E.)X400).

Fig.(8a): Degenerated skeletal muscle area with increased activity of acid phosphatase. (X 125).

Fig.(8b): Degenerated subendocardial muscle area with increased activity of acid phosphatase. (X 125).

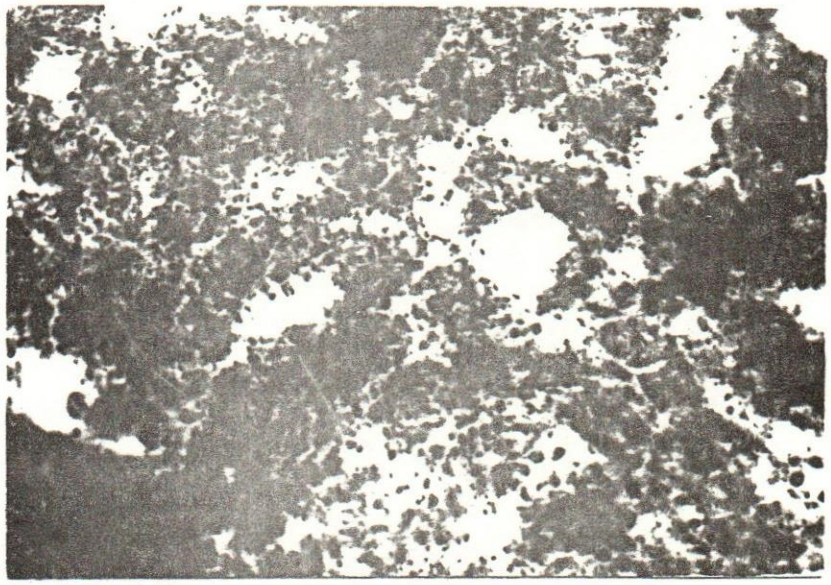

Fig.(9): Loss of succinic dehydrogenase activity in the affected lesions of the liver. (X 125).