ULTRASTRUCTURE OF BOVINE LIVER TELANGIECTASIS

Ultraestructura de la telangiectasia hepática bovina.

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ABSTRACT

Bovine liver telangiectasis is one of the most common liver lesions found in cattle throughout the world. Liver samples from 49 bovines with telangiectasis and from 29 normal animals were examined by electron microscopy. Ultrastructural changes consisted of dilation of sinusoids and Disse’s space, a decrease in the number and size of hepatic cell microvilli, which were swollen or transformed into cytoplasmic blebs of hepatocytes and vacuolar degenerative changes. These changes were regarded as ischemic or hypoxic ones. Endothelial and Kupffer cells were normal. Theories about the pathogenesis of telangiectasis were reviewed and analyzed in relation to our ultrastructural findings. It is concluded that ischemia of hepatocyte theory is well supported by electron microscopic findings.

Key words: Bovine, liver, telangiectasis, ultrastructure

INTRODUCTION

Bovine liver telangiectasis, a focal dilation and congestion of a group of hepatic sinusoids, is the most common liver lesion in cattle from Venezuela [5,19] and other countries of the world [2, 5, 9, 13, 16, 20, 24]. This lesion causes direct economic losses due to liver condemnation by sanitary inspectors and, in severe cases, due to impairment of liver function, indirect losses related to a decrease in milk yield or meat production [15].

The etiology and pathogenesis of bovine liver telangiectasis is still unknown, although several theories have been proposed [1, 2, 3, 6, 8, 9, 10]. There are no previous reports of ultrastructural studies on bovine liver telangiectasis(40,316),(953,438). The purpose of this research was to study the ultrastructure of this lesion in order to get information about its etiology and pathogenesis.

MATERIALS AND METHODS

Liver samples from 49 bovines with hepatic telangiectasis, and from 29 normal bovine livers, were taken between 20 to 35 minutes after death at a local slaughterhouse, fixed by immersion in cold 3% glutaraldehyde in 0.1 M phosphate buffer, post-fixed in 1% osmium tetoxide, dehydrated in alcohol, embedded in PolyBed 812 (Polysciences Inc., Warrington, Penn., USA), cut, stained with lead citrate and uranyl acetate, and observed at the electron microscope.

RESULTS

Hepatocytes from the telangiectatic areas showed clear changes of swelling and degeneration characterized by the formation of electron-lucent, finely granulated, intracytoplasmic vacuoles, swollen and round mitochondria, diminished glycogen granules, slight dilation of the endoplasmic reticulum and lysis of the cytoplasmic matrix; nuclei did not show apparent changes (Fig. 1). Other hepatocytes showed true cytoplasmic blebs which contained cellular organelles, glycogen granules and free ribosomes (Figs. 1, 2, 3, 5). Microvilli were absent or diminished in number and length, becoming broader at their base; some microvilli persisted as cytoplasmic blebs in the space of Disse, with microtubules and filaments diminished in numbers or absent (Figs. 1, 2, 3, 5, 7). Disruption of intercellular junctions between hepatocytes were observed and free hepatic cells were seen at Disse’s space (Fig. 4).

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Palabras claves: Bovino, hígado, telangiectasia, ultraestructura.
Fig. 1. Hepatocyte showing a round, swollen, mitochondria (M) with diminished cristae and a cytoplasmic vacuole (V). Disse's space (D) is widen, containing swollen microvilli (arrowheads). Bar = 0.5 μm.

Fig. 2. Disse's space showing scant, swollen, widen microvilli (arrowheads) from a hepatocyte (H) and a cytoplasmic projection or bleb (B) from another hepatocyte. A normal endothelial cell (E) is observed at the sinusoidal wall. Basal lamina is pointed out by arrows. Bar = 0.5 μm.

Fig. 3. A cytoplasmic projection or bleb (B) and swollen microvilli (arrowheads) are observed at Disse's space. Endothelial cell (E); basal lamina (arrow). Bar = 0.5 μm.

Fig. 4. A free, unattached hepatocyte (H) and a cytoplasmic projection (arrowhead) from another hepatocyte is observed at a widen Disse's space (D). Arrows are pointing out the basal lamina. Bar = 0.5 μm.

Fig. 5. Disse's space showing several swollen, deformed microvilli (arrowheads). Part of an endothelial cell (E) and an ill-defined basal lamina (arrow), are also observed. Bar = 0.5 μm.

Sinusoids were dilated. The space of Disse was widen, containing scant, broaden, microvilli, cytoplasmic blebs of hepatocytes, free hepatocytes, rests of organelles, and, in a great number of cases, erythrocytes and leucocytes (Figs. 1, 2, 3, 4, 6, 7).
The basal lamina was normal or slightly widen (0.1μm) and its structure was fuzzier than normal; only in few cases it was lost, and, in these cases, there also was a total destruction or lost of the sinusoidal wall (Fig. 6).

The endothelial cells showed a slight increase in the number of their pinocytotic vesicles, otherwise were normal (Fig. 7). Kupffer cells were normal.

Our findings have revealed hepatocyte changes characterized by a reduction or lost of microvilli, formation of cytoplasmic blebs into Disse's space and slight dilation of the endoplasmic reticulum, changes that are characteristics of cellular hypoxia. Similar changes have been observed in rats experimentally subjected to hypoxia [11, 12].

Autolysis of hepatocytes can also cause similar changes to those above mentioned [11]. However, in our cases, these changes were disregarded as autolytic because they were not seen in the livers of normal animals subjected to the same method of sacrifice, equal elapsing time between death and fixation and similar electron microscopy technique.

Hepatic cell necrosis and infiltration of leucocytes were not observed in our cases, so our results allow to point out that neither hepatic necrosis nor inflammation were the initial cause of bovine liver telangiectasia. In regard to glycogen, we observed a decrease of it in the hepatocytes, probably due to fasting of animals before sacrifice, and did not see its accumulation in the microvilli, the cytoplasmic blebs, or underneath endothelial cells; consequently, these findings do not support Andersen's theory. Reid et al [15] have demonstrated that fasting is a cause of glycogen depletion in hepatocytes.

Ischemic changes of hepatocytes followed by hydropic or vacuolar degeneration, as seen in our cases, cause a swelling of the affected cell, bringing about a shortening or disappearance of microvilli and can produce a greater tension upon the cell membrane with disruption of intercellular junctions, releasing the hepatocytes into Disse's space or the sinusoidal lumen, if the sinusoidal wall was broken; these changes were observed in few cases.

Dilation of sinusoids and Disse's space is an ultrastructural finding described in cases of liver telangiectasis (or Pseudois) in humans [17], it was a constant finding in our cases. Dilation of Disse's space is due to shrinkage and reduction of hepatic microvilli, retraction of degenerating hepatocytes or to loosening and release of hepatic cells from the liver trabecula. In some cases we observed erythrocytes and leucocytes into Disse's space which indicated breakage of the sinusoidal wall, allowing the blood to enter this space. Destruction of the endothelial lining and the sinusoidal wall have also been observed in human liver telangiectasia[14, 17].

Changes in endothelial or Kupffer cells that could explain the breakage of the sinusoidal wall were not observed; for this reason, we think that sinusoidal wall breakage was just a physical event, probably due to a pressure increase by plasma in the space of Disse.
The changes observed correspond to a lesion initially produced by hypoxia or ischemia of hepatocytes, which brings about, as a consequence, a dilatation of Disse’s space, shrinkage and release of hepatocytes and breakage of the sinusoidal wall probably due to an increased pressure at the space of Disse.

Our ultrastructural findings support Jensen et al.’s [10] theory which states that ischemia of hepatocytes is the basic lesion of bovine liver telangiectasis.

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REFERENCES