MORPHO-STRUCTURAL ASPECTS CONCERNING THE HEALTHY LIVER COMPARATIVE TO THE PATHOLOGICAL ONE

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Keywords: hepatic affections, degradated hepatocytes, hepatic lobules, fibrosis.

Abstract: The paper analyzes the microscopic modifications of the liver, on a batch of dead patients,suffering from hepato-cellular affections, all from the Districtual Hospital of Botosani, between September-November 2008. The patological aspects of the liver, in its every form, are observed especially in men which are big alcohol consumers. The normal structure of the liver is obviously transforming in hepatic diseases like alcoholic steatohepatitis, hepatic cirrhosis, liver cancer and hydatid cyst.

INTRODUCTION

As we know, the liver is a digestive gland which has an extreme importance in the metabolism of different organic substances. In normal manner, the liver is composed of four lobes: the right lobe, the left lobe, the quadrate lobe and the caudate one (Spigelian lobe). On surface or in exterior, it is covered by a fibrous capsule named Glisson' s capsule; this capsule pervades in the interior of liver mass and separates the liver stroma in hepatic lobules. On transversal sections, we can observe in a healthy liver this hepatic lobules very well contoured, separated betwwen them by Kiernan' s spaces.

The structure of each normal hepatic lobule is composed of a centrilobular vein, portal vein branch and a perilobular vein, suprahepatical vein branch. Between these two vessels it is a very dense system of venous cappilary. In the eyes of this system there are the hepatic cell cordons (or Remak cordons), every hepatocyte having an approximate hexagonal or quadrate shape and in the center two or three nucleus; hepatocyte' s cytoplasma is rich in mitochondrias, lysosomes and endoplasmic reticle, but predominating the mitochondrias. In some places we can observe inclusions like some vacuoles full of fatty content; in a healthy liver, these inclusions are sporadic or very small, sometimes even missing.

The patological aspects of the liver, in its every form, are observed especially in men which are big alcohol consumers. The normal structure of the liver is obviously transforming in hepatic diseases like hepatitis B, hepatitis C; large drugs consumption and also in large alcohol ingestion. The present paper discusses the results of some investigations devoted to microscopic liver modifications in patients suffering from different hepatic affections, such as alcoholic steatohepatitis, hepatic cirrhosis, liver cancer and hydatid cyst, hospitalized in the Mavromati Districtual Hospital of Botosani, between September-November 2008.

MATERIALS AND METHODS

The study starts from the results of the microscopic examination of the liver samples taken over from the dead patients, suffering from hepatic affections (alcoholic steatohepatitis, hepatic cirrhosis, liver cancer and hydatid cyst), in the above-mentioned period. The pieces of liver tissue were fixed for light microscopy in 10% solution of formaldehyde, dehydrated in ethanol and amylic alcohol, embedded in parafin and sectioned at 1-2 microns. They were stained with hemalaun-eosine and examined with a Novex microscope and photographed with a Panasonic TZ1.

RESULTS AND DISCUSSIONS

The first signal indicating a structural modification of the liver behind a large alcohol consumption is alcoholic steatohepatitis; alcoholic steatohepatitis represents a large lipids accumulation in hepatic cells. At first, the lipids are marked out only partially in hepatic lobules, so affecting only certain territories of hepatic lobules (fig. 1.1.), then including extensive areas. In figure 1.1., we can observe the debut of this liver affection, the hepatic lobules not being degraded entiretilly. Every hepatic lobule presents potent vacuolised cells, full with liquid or fatness, degradated hepatocytes situated especially around the centrolobular vein (Pinto *et al.*, 1996; Sheth *et al.*, 1997; Tehli *et al.*, 1995). The Remak cordons appear relatively normal. On this section too, we can observe demotions of the hepatocytes in the periphery of some hepatic lobules (fig. 1.2.).

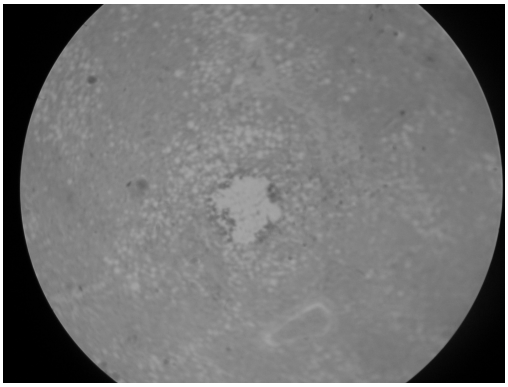

Figure 1.1. Debut of alcoholic steatohepatitis (10x)

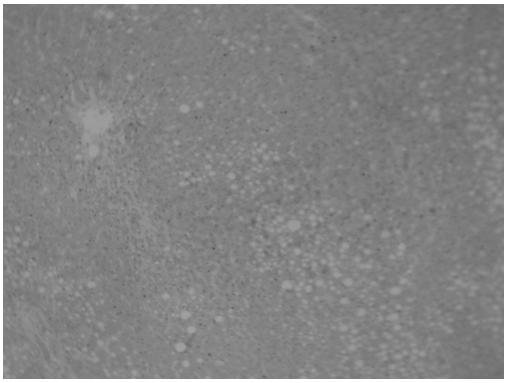

Figure 1.2. Demotions of the hepatocytes in the periphery of some hepatic lobules (10x)

This is the debut of a steatosis. In case of a long alcohol consumption, obesity, high level of cholesterol and triglycerides (dislipidemy), sugar diabetes, excessively drugs ingestion (estrogen hormones, corticoids), major intestinal rejections or genetical diseases with defect of lipids metabolization it cames to hepatic cirrhosis, the final stage of hepatic affections evolution.

In figure 2.1., a section by a human liver, male type, 47 years old, we can observe some aspects of advanced cirrhosis. In this case, the hepatic lobules have no contour (they lost the hexagonal contour), the liver having an aspect of uniform mass, punctuated some places with centrolobular veins (Geller, S.A., 2000); round the centrolobular veins, on extensive areas, the hepatocytes are very vacuolised, with lipids inclusions. In this degradated mass of the liver we can also observe unfunctional hepatocytes, organised in relatively round nodules (Mazzaferro *et al.*, 1996), separated by fibrous bands (fig. 2.2.).

Figure 2.1. Aspects of advanced hepatic cirrhosis (10x)

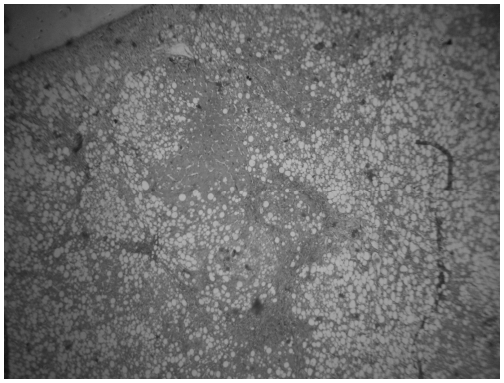

Figure 2.2. Hepatocytes organised in round nodules), separated by fibrous bands (10x)

Gradually, these nodules having functional degradated cells extende, including the whole mass of a hepatic lobule or the whole mass of the liver. In hepatic cirrhosis, we can observe an interlobular and intralobular conjunctive tissue hyperplasia, which separates the parenchyma in microscopic ″islands″. Fibrosis invades and modifies profoundly the portal space, and sometimes draws real septa. Regeneration nodules represent fragments or hepatic lobules themselves with regeneration aspects. Hepatocytes bays are profoundly modified (Cassiman *et al.*, 2002). Inside the regeneration nodules there are no sinusoidal capillaries (Chedid, A., 2000). Hepatic nodules have a reduced metabolic activity, the hepatocyte - portal blood exchanges being compromised. Hepatic architecture is profoundly alterated in cirrhosis. Hepatocitary necroses, fibrogenesis, the forming of regeneration nodules and the lobular fragments surrounded by fibrosis bands are accompanied by portal spaces arranging perturbation, as well as centrilobular veins and hepatic cells arranging perturbation. Some hepatic nodules are lacked of the centrilobular vein, while inside other nodules two or more centrilobular veins are conglomerated. Sometimes, there are more portal spaces, conglomerated all together, in the conjunctive bands which separate the hepatic cells islands, and this fact is testified by the portal triads presence (Gherasim, 2000). The radial hepatocyte cordons arranging disappears, the hepatic cells presenting dystrophic lesions and necroses (Chejfec, G., 2000; Desmet *et al.*, 2004).

Previously, we brought to mind that the hepatic viruses (B, C and D), the aflatoxines, the cirrhosis, the alcohol, the anabolisant steroids, the hemochromatosis, α-1 antitripsine deficiency and the tirosemia lead to hepatic oncogenesis (Benvegnu *et al.*, 1994; Calvert *et al.*, 1990; Mukunda, B.R., 2000; Okuda *et al.*, 1985). In figure 3.1., a section by a human liver with cancer debut, male type, 60 years old, we observe the Remak cordons, composed of powerfull turgid hepatocytes (Wanless *et al.*, 2000); every hepatocyte presents numerous nucleuses, some of them on the way of splitting (fig. 3.2.). The hepatic lobules lost their contour, the Remak cordons are majority free, rounded by lymphocitary infiltrations (fig. 3.3.), so the liver becomes spongious and it is degradating gradually until disappearance (Tsukuma *et al.*, 1993).

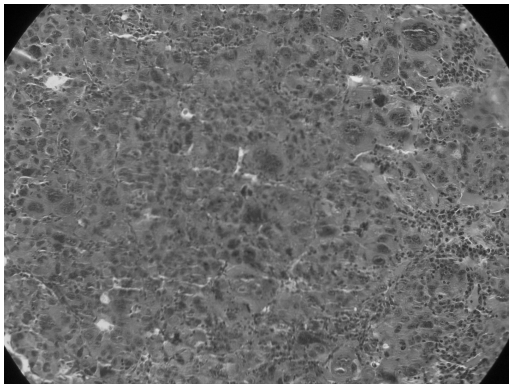

Figure 3.1. Human liver with cancer debut (10x)

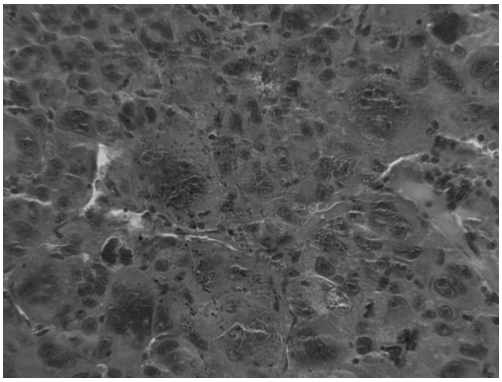

Figure 3.2. Hepatocytes with nucleuses on splitting (40x)

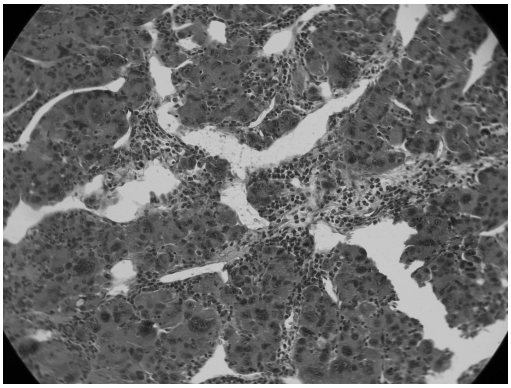

Figure 3.3. Remak cordons rounded by lymphocitary infiltrations (40x)

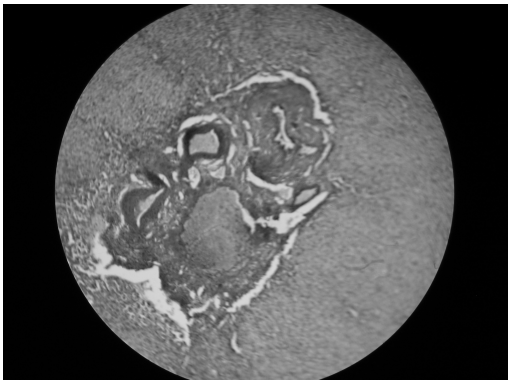

Figure 4.1. Hydatid cyst, ensemble (20x)

Finally, we present the aspect of a liver invaded by a hydatid cyst, female type, 69 years old (fig. 4.1., ensemble and detail). On this section we can observe the hepatic tissue round the cyst looking like a uniform mass without contours, the hepatic lobules being marked only by the presence of centrolobular veins. It is observe a succession of scolexes positioned inside the cyst (fig. 4.2., fig. 4.3.). In this case, the hepatic tissue is degradating gradually, the hepatic cordons loose their contour and direction, the hepatocyte have in majority a series of inclusions, especially pygmentary; because of these pygmentary inclusions the nucleuses almost disappear, they can not being visible (fig. 4.4., ensemble and detail). Gradually, the Remak hepatic cordons are clear away between them creating numerous spaces, thus the liver acquires an network aspect (fig. 4.5.).

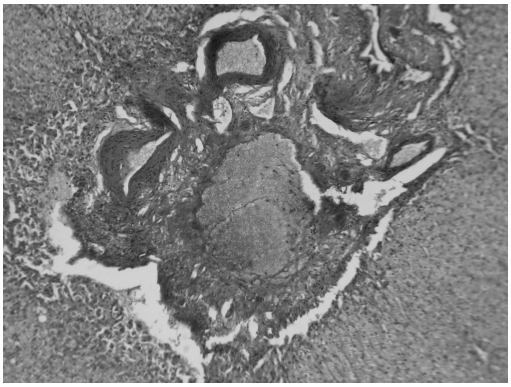

Figure 4.1. Hydatid cyst, detail (40x)

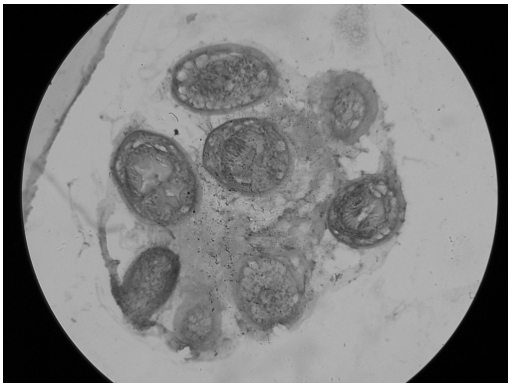

Figure 4.2. Succession of scolexes inside the cyst (20x)

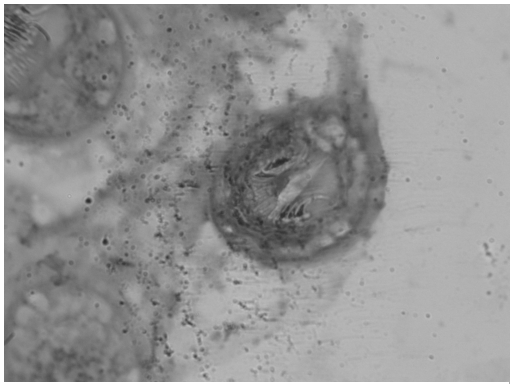

Figure 4.3. Succession of scolexes inside the cyst (40x)

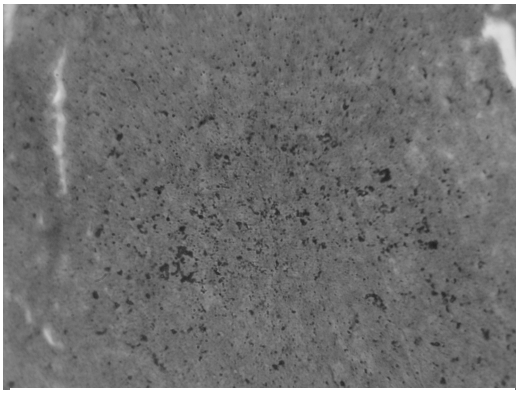

Figure 4.4. Hepatocytes with pygmentary inclusions and the nucleuses can not be visible, ensemble (40x)

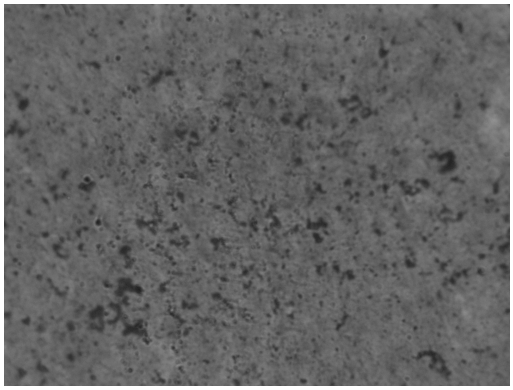

Figure 4.4. Hepatocytes with pygmentary inclusions and the nucleuses can not be visible, detail (40x)

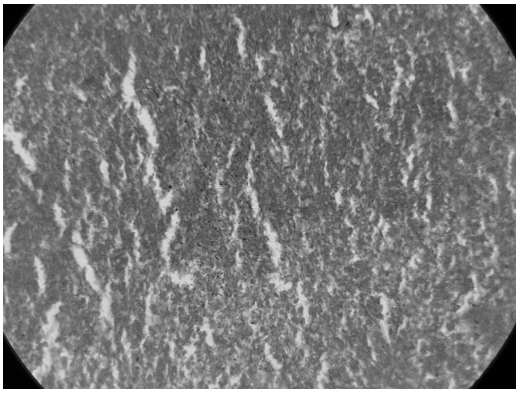

Figure 4.5. Degradated liver with network aspect (40x)

CONCLUSIONS

The patological aspects of the liver, in its every form, are observed especially in men which are big alcohol consumers. The normal structure of the liver is obviously transforming in hepatic diseases like hepatitis B, hepatitis C; large drugs consumption and also in large alcohol ingestion. The investigations of the patients (in majority male) suffering from different hepatic affections, such as alcoholic steatohepatitis and hepatic cirrhosis revealed microscopic liver modifications, characterized by lipidic inclusions, degradated hepatic cells, fibrosis, regeneration nodules, hepatic architecture profoundly alterated, hepatocitary necroses. In hepatic oncogenesis, it was observed powerfull turgid hepatocytes having numerous nucleuses, some of them on the way of splitting, lymphocitary infiltrations, thus the liver became spongious and it was gradually

degradated until disappearance. In the case of hydatid cyst we can also observed degradated hepatic tissue, hepatocytes with pygmentary inclusions, the disappearance of the nucleuses and the network aspect of the liver.

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