HISTOPATOLOGICAL ASPECTS IN HEPATIC CIRRHOSIS

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Abstract: The paper analyzes the macroscopic and the microscopic modifications of the liver, on a batch of dead patients,suffering from hepatic cirrhosis,all from the Districtual Hospital of Botosani, between February-June 2006. Hepatic cirrhosis is histologically characterized by an association with fibrose, regeneration nodules and hepatocytary necroses, therefore, alteration of the hepatic and vascularization architecture. The microscopic examination also revealed an interlobular and intralobular conjunctive tissue hyperplasia, which separates the parenchyma in microscopic ″islands″.

INTRODUCTION

Hepatic cirrhosis ranks among the ten fatal maladies, the most pregnant causes of its installation being the hepatic viruses and the alcohol (Malik *et al*., 2002; Haruta *et al*., 2007). Besides these, a faulty alimentation, precarious living conditions, auto-imune hepatitis, biliary stasis, the drugs, heart insufficiency may also provoke hepatic cirrhosis (Martín and de Las Heras, D., 2004; Nacoulma *et al*., 2007).

Hepatic cirrhosis is an extremely severe disease, in which the functions of the hepatic cells are blocked by the intracytoplasmatic cerrides, structurally characterized by an association with fibrose, regeneration nodules and hepatocytary necroses, therefore, alteration of the entire hepatic architecture. Hepatic lobules are ″dislocated″ through by fibroses bridges, resulting hepatic lobule fragments (pseudolobules). They present regeneration aspects (up to regeneration nodules constitution), having variable diameters, from 2-3 mm (micronodular cirrhosis) up to several cm (macronodular cirrhosis). Hepatocitary necroses, fibrogenesis and the regeneration nodules constitution, lobular fragments surrounded by fibrosis bands, are accompanied by the portal canals, centrilobular veins and hepatic cells arranging perturbation (Cassiman *et al*., 2002; Desmet *et al*., 2004; Geller, 2000; Gheban, 2000). Some hepatic nodules are lacked of the centrilobular vein; inside other nodules, two or more centrilobular veins are conglomerated. The radial hepatocyte cordons arranging disappears inside the hepatocyte ″islands″, the hepatic cells presenting dystrophic lesions and necroses.

The present paper discusses the results of some investigations devoted to macroscopic and microscopic liver modifications in patients suffering from hepatic cirrhosis, hospitalized in the Mavoromati Districtual Hospital of Botosani, between February-June 2006.

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 cirrhosis, 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

Hepatic cirrhosis is a chronic affection of the liver, having a progressive and an irreversible evolution, histologically characterized by an association with fibrose, regeneration nodules and hepatocytary necroses, therefore, alteration of the hepatic and vascularization architecture (Wanless *et al*., 2000). The tridimensional structure of the hepatic lobule is profoundly altered, leading to the vascular, lymphatic and biliary polarity debasement. Regardless its causes, hepatic cirrhosis associates various manifestations of hepato-cellular insuficiency to portal hypertension syndrome.

The macroscopic aspect: the liver weight can increase, from 1,5 kg (regular weight) up to 2,5-3 kg. In certain evolutional or advanced phases, the liver reaches 7-800 g. The colour of the liver diversifies to etiology. Its aspects is oftenely spotty; posthepatitic cirrhosis has a puce colour, the biliary one is bottle green with red. Its surface is irregular, with nodes. The liver consistency is tinny, and its inferior margin is sharp (Mukunda, 2000). There is a micronodular cirrhosis (alcoholic cirrhosis) and a macronodular one (postnecrotic cirrhosis caused by hepatitis viruses).

The microscopic aspect: 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. It was remarked port-portal, center-portal and center-central fibroses bridges. Regeneration nodules represent fragments or hepatic lobules themselves with regeneration aspects. Hepatocytes bays are profoundly modified. In post-viral cirrhosis, regeneration nodules are bigger (from 2-3 mm up to some cm) and surrounded by a large quantities of conjunctive tissue, which separates the nodules in parenchyma ″islands″ during regeneration. Inside the regeneration nodules there are no sinusoidal capillaries. Hepatic nodules have a reduced metabolic activity, the hepatocyte - portal blood exchanges being compromised.

Hepatocitary necroses are upkept by toxic factors and by sinusoidal obstructions (Chedid, 2000; Chejfec, 2000); hepato-cellular insuficiency is perpetuated by fibrogenesis in spaces of Disse, even if the inflamation is missing.

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.

Micronodular cirrhosis (alcoholic cirrhosis, portal cirrhosis or Laënnec's cirrhosis) is characterized by the numerous regeneration nodules presence; they have small dimensions (diameter of 2-3 mm), relatively equals, separated by the excessively conjunctive tissue bands. The lesions are extended to all lobules and the liver becomes small, tinny and nodular. There is a perinuclear eosinophilic material (″alcoholic hyaline″ or Mallory bodies) inside the hepatic cells, which represents a complex protein with antigenic properties.

Macronodular cirrhosis (postnecrotic cirrhosis, posthepatic cirrhosis or multilobular cirrhosis) is characterized by the hepatic tissue nodules presence, with a diameter over 3 mm; some nodules reaches a dimension of cm order. Regular hepatic nodules can be founded in these macronodules. A voluminous regeneration nodule can suggest initially the neoplasm. Macronodular cirrhosis develops a much more important splenomegaly than micronodular cirrhosis.

CONCLUSIONS

Hepatic cirrhosis is structurally characterized by an association with fibrose, regeneration nodules and hepatocytary necroses, therefore, alteration of the entire hepatic architecture. Hepatocitary necroses, fibrogenesis and the regeneration nodules constitution, lobular fragments surrounded by fibrosis bands, are accompanied by the portal canals,

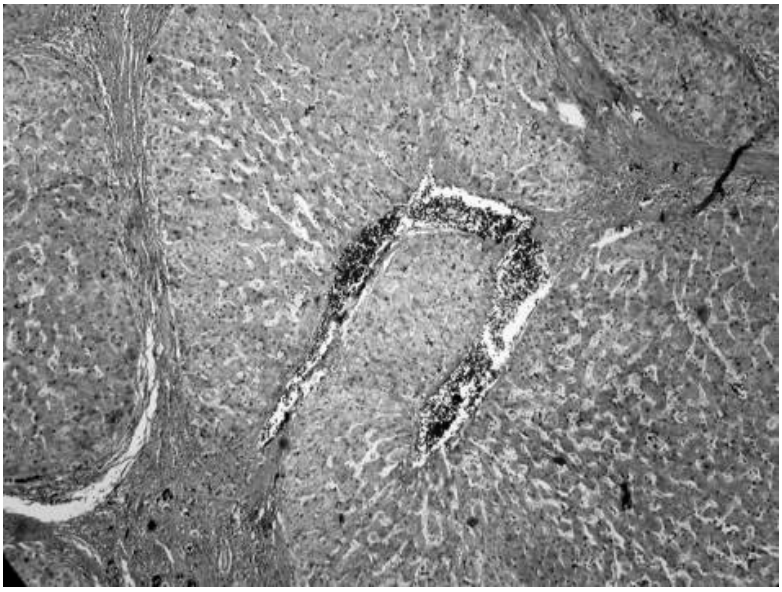

Figure 1. Portal-centrilobular shunt, wide open, a major contributor for hepatic insuficiency

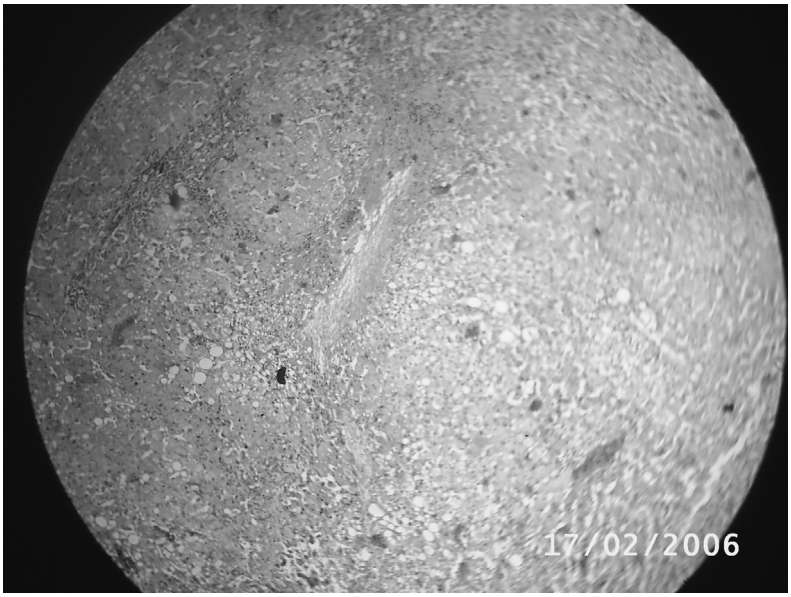

Figure 2. Acute hepatocitary necrosis in hepatic cirrhosis

centrolobular veins and hepatic cells arranging perturbation. The hepatic cells present dystrophic lesions and necroses.

There is an inflammatory lympha-plasmocytary infiltrate inside the conjunctive tissue, and the limiting cellular plate, situated to the hepatic parenchyma islands periphery, is perforated by inflammatory-conjunctive fronts, which progressively advance, causing the hepatocyte's death. There are numerous regeneration nodules, with small dimensions, relatively equals, separated by the excessively conjunctive tissue bands, and lesions extended to all lobules in micronodular cirrhosis. In macronodular cirrhosis, hepatic tissue nodules reach dimensions of cm order; the macronodules can contain regular hepatic lobules.

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