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HISTOPATHOLOGICAL FEATURES IN CHRONIC PANCREATITIS

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ABSTRACT. We present the results of a prospective and retrospective study conducted in the last 10 years in three Universities of Medicine (Arad's and Timisoara's Universities of Medicine from Romania and Chisinau's University of Medicine from Republic of Moldova). The results are based on clinical and paraclinical data collected from 53,03% of the entire lot of patients diagnosed with chronic pancreatitis admitted in the surgical departments of the aforementioned universities. The patients were distributed in two groups, according to the aetiology of the chronic pancreatitis: obstructive and non-obstructive chronic pancreatitis. The age of the patients varied from 17 to 65 years, with a mean age of 45 years. Intraoperatory pancreatic tissue was prelevated from all patients and sent for histopathological analysis. All patients signed the informed consent. Structural modifications of the pancreatic acinar cells were noticed, evidence of different degrees of functional activity, dystrophic modifications and cellular dysfunction being also found. Concomitantly, alongside dystrophic intracellular modifications we noticed destructive disorders of the acinar basal membrane and of the cellular membranes.
KEY WORDS : chronic lithiasic pancreatitis, morphological, histopathological changes

INTRODUCTION

Chronic pancreatitis is a controversial disease, showing an increasing frequency, often diagnosed after a bout of acute pancreatitis, being determined either by the presence of gallstones in the Wirsung channel either by stenosis or inflammation (Curca T. Et al., 2002; Gheorghe L et al., 2002; et al., Gheorghita V et al., 2004). Since therapeutic and surgical management is done according to ethiopathogenic factors, whole teams of specialists are trying a multidisciplinary approach in order to achieve a pre- or intraoperatory diagnosis, that would allow a more accurate approach, so that the therapeutic benefit could be as efficient, and allow as much social and professional reintegration as possible for the patient (Geers C et al., 2006; Notohara K et al., 2000).

MATERIAL AND METHOD

105 patients with chronic pancreatitis (CP) and indication for open surgical treatment were included in the study. In all cases intraoperatory bioptic fragments were taken from different regions of the pancreas. The patients were divided in two lots according to the aethiology of CP. I-st lot included patients with CP of obstructive aethiology and the II-nd lot patients with CP of non-obstructive aethiology. The fragments were fixed in formalin and sent for histopathological examination. Haematoxilin-eosin and Van-Ghizon stainig was performed

RESULTS

Intraoperatory pancreatic tissue prelevation could be performed in 81 (77,14%) cases (I lot–35 (81,4%) cases, II lot–46 (74,19%) cases). The fragments had an approximate size of 10/15 mm, excised from the cephalopancreatic region in 35 cases (43,2%) (I lot - 15 (42,8%) cases, II lot - 20 (43,4%)

cases), from the pancreatic body in 30 cases (37,0%) (I lot - 15 (42,8%) cases, II lot -15 (32,6%) cases) and from the tail of the pancreas in 16 cases (19,8%) (I lot – 5 (14,2%) cases, II lot – 11 (23,9%) cases). Histopathological examination revealed extended areas of exocrine parenchyma with minimal inter- and intralobular sclerosis. In 53 (81,5%) cases in the conjunctive and interlobular spaces a persistence of diffuse inflammatory tissue could be noticed. **(fig.1**); there was also an excess of inflammatory process in other areas **(fig. 2**). Interlobular sclerosis signs were found at different levels (**fig. 3**), focal lipomatosis, with lobule fragmentation signs. The inflammatory tissue contained lymphocytes, plasmocytes and macrophages. In the cases of diffuse pancreas sclerosis, there were pronounced signs of inter and intralobular sclerosis, with incipient or definitive fragmentation of lobules in smaller pieces of acinary tissue.

More significant transformations were found in the pancreatic ductal system. Interlobular duct walls were thicker, sclerosed, with dystrophic or atrophic changes in the mucosa, deformation or atrophy of epithelium and its proliferation in the ductal wall, with cystic dilatation of the basal duct (**fig. 4**). Fingerlike and valvular excrescences of fibrous tissue in the ductal walls obliterated almost completely their lumen and made pancreatic juice transit perturbations. (**fig. 5**).

We found epithelium dysplasia in the form of mucous cylindrical cells, developing adenomatous excrescences
on some areas. Different sectors showed focal proliferation of ductal epithelial wall with genesis of structures like polistratified epithelium. Cells located in the superior layers of this proliferation had a cylindrical form with preserved mucus secretion function. The epithelium of the greater pancreatic duct showed also dystrophic changes, from cylindrical to prismatic epithelium. The cylindrical epithelium of small pancreatic ducts kept its secretion function, but the greater ducts epithelium didn't. This was demonstrated by the presence of mucus inhibitors, which disturbed the drainage function of the ducts. The concentration of unzymogen protein increased, with his coagulation and genesis of microlithiasis (**fig. 6**).

In 3 (3,7%) cases (I lot – 1 (2,8%) case, II lot – 2 (4,3%) cases) Ca accumulation was found in the pancreatic parenchyma **(Fig.7**). In advanced cases, the exocrine parenchyma was destroyed, with lobules sclerosis and presence of limfoplasmocitar infiltration (**Fig.8**).

In all 16 (19,8%) biopsies (I lot – 5 (14,2%) cases, II lot – 11 (23,9%) cases) from the pancreatic tail area, there were histological signs of pronounced interlobular sclerosis, with different levels of atrophy of acinar tissue and duct proliferation. The lobules remained isolated between large bands of hard microcellular conjunctive tissue, with isolated areas of exocrine parenchyma. Also, on some sectors, we found their fragmentation with almost total substitution of pancreatic tissue with conjunctive tissue. In cases with acinar tissue atrophy, multiple focal duct proliferation areas were observed, or the cystic dilatation of the ducts. Associated, there were small areas of endocrine tissue, made from big cells with light colored cytoplasm, and small hypertrofic nuclei. In cases of acinar parenchyma atrophy, the pancreatic endocrine tissue did not show signs of atrophy, but positive signs of hyperplasia (**fig. 8**). In these islands, besides endocrine cells we met also CA without membranes and clear secretor cells, characteristic for both endocrine and exocrine cells. So, in some atrophied lobules we met large islands of endocrine tissue, sometimes exceeding in dimensions the exocrine tissue sectors. We found unheterogenic cell mixture in the Langherhans islands. In the structure of some isolated islands, there were mainly bog cells with picnotic cytoplasm and nucleus. This different content of endocrine cells indicate that at PCC patients there is a change in endocrine tissue function with the increase of different endocrine hormones.

DISCUSSION

Histopathological examination of pancreatic tissue prelevated from patients with PCA revealed some interesting morphological data (Adams AL et al., 2008; Carmen Neamtu et al.,2007). They permitted a more detailed look into the structure changes in CA, which indicate different levels of functional activity, dystrophic changes and cell destruction. We also found shape and dimensions modification of the acines of pancreatic lobules. The exocrine parenchyma kept the synthesis function of pancreatic enzimes, with an increased amount of secretor grains in CA cytoplasm and decreased synthesis of intralobular enzimes. We found big acins with dilated ducts, full with mucosal secretions. In CA cytoplasm, zymogene grains were accumulated both apically and the rest of cytoplasm sectors. In pancreatic lobules there were dystrophic changes outside the intraductal sclerosis areas, with loss of the orientation of cells.

In peripheral sectors, there were signs of interacinar sclerosis with collagen fascicles

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penetrating into the acines causing its fragmentation in small pieces. Dystrophy and cell destruction is followed by glandular sclerosis and lipomatosis. We found no signs of periductal sclerosis. Intralobular ducts were constituted by an epiteliocyte layer, without liquid content.

In PCA we found thickened arterial wall, with small lumen.

Nervous trunks passing through the conjunctive tissue were also hypertrophied. We found also a nervous dystrophic adenopathy. Number of nervous cells were decreased. These explain the continuous algic syndrome.

CONCLUSIONS

Structural modifications of the pancreatic acinar cells were noticed with evidence of different degrees of functional activity, dystrophic modifications and cellular dysfunction.

Concomitantly, alongside dystrophic intracellular modifications we noticed destructive disorders of the acinar basal membrane and of the cellular membranes.

In the case of chronic pancreatitis the following histopathologic peculiarities of structural modifications were found:

- the process of atrophy of the acinar parenchyma
- the substitution fibrosis (vessels with narrow coherent lumen), intravascular fibrosis with or without intraacinar sclerosis
- the hypertrophy of nerve fibers, modifications which explain the contiguous pain syndrome.

CONTRIBUTIONS

All authors contributed equally to the present work.

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Figure legends:

Fig. 1. Inflammatory reaction in pancreatic parenchyma (hematoxilin and eosin x 63))

Fig. 2. Conjunctive tissue on the place of pancreatic tissue necrosis. (hematoxilin and eosin x 63)

Fig. 3. Pancreatic parenchyma sclerosis (hematoxilin and eosin x 63 colored)

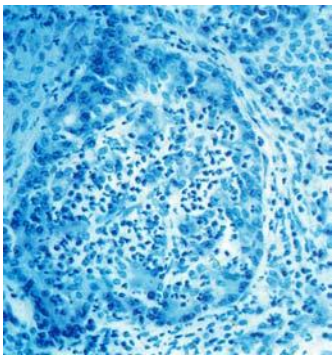
Fig. 4. Ulcerations on pancreatic duct mucosa (colored with hematoxilin and eosin x63)

Fig. 5. „Fibrotic valve" of pancreatic duct (colored with hematoxilin and eosin x63)

Fig. 6. Microlithiasis in pancreatic duct lumen (colored after Van-Ghizon x 63)

Fig. 7. Ca accumulation in pancreatic parenchyma (colored after Van-Ghizon x 63)

Fig.8. Pieces of exocrine pancreatic parenchyma. Sclerosis of pancreatic lobules and limfoplasmocitar infiltration (colored with hematoxilin and eosin x 63)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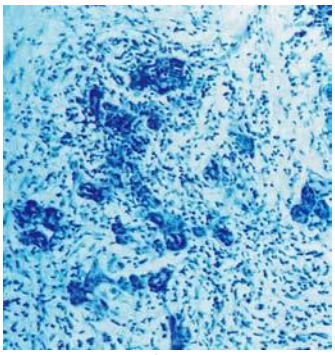
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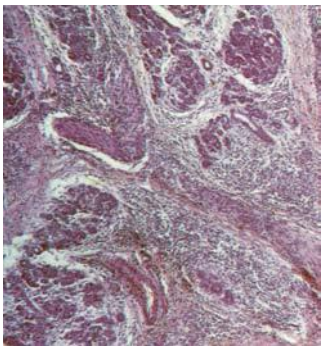
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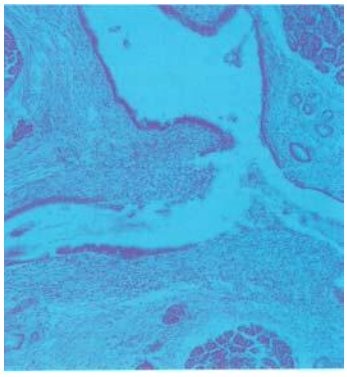
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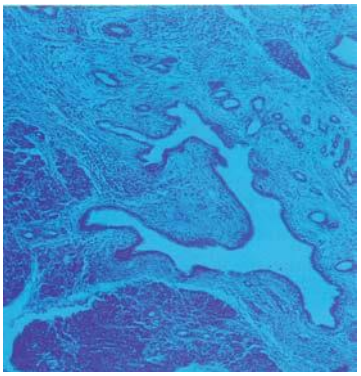
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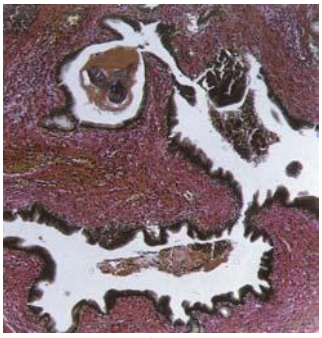
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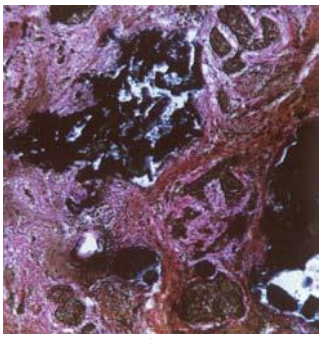
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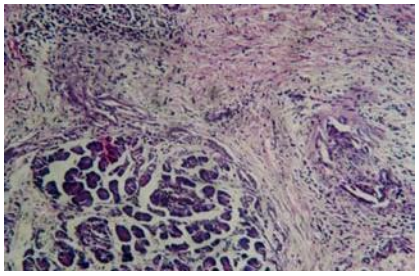
Fig.8