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MORPHOLOGICAL ASPECTS OF THE NORMAL VERSUS PATHOLOGICAL LIVER

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Introduction. Chronic liver diseases stimulate a degree of hepatocyte injury. This previously mentioned modifications, alters the known liver architecture and finally ends in cirrhosis. Liver pathology as cirrhosis develops after a long period of pathological alterations. In this iddea, inflammation is a great point that results in replacement of the healthy liver parenchyma with fibrotic tissue and regenerative nodules. In addition, progressive portal hypertension, systemic inflammation, and liver failure drive cirrhosis outcomes. The management of this liver pathology, is centred on the treatment of the causes and complications. Liver transplantation can be required in some cases.

The aim of this article was to identify the best available evidences analyzing liver samples, normall and pathological.

Material and methods. Were made permanent preparations and used two colors. Hematoxylin–Eosin and also Goldner – Szekely trichrome stains stain for observation at optical microscope with x10 and x40 lens magnification. Samples liver collected during necropsy, from healthy patients and from patients diagnosed with cirrhosis.

Results and discussion. Normal liver with hepatocytes, Kiernann space, connective septa, observations using lens x10 and samples colored with Goldner Szekely trichrome stains. Beside, for comparisions, ill liver images, classic stain H&E. Inflammation is a great point that results in replacement of the healthy liver parenchyma with fibrotic tissue and regenerative nodules. In addition, progressive portal hypertension, systemic inflammation, and liver failure drive cirrhosis outcomes.

Conclusions. Our contribution in the written text, is related to the impact of physical, psychological and physiological factors. All this previously mentioned factors, area great impact on the health-related quality of life of adult patients with liver cirrhosis. The management of this liver pathology, is centred on the treatment of the causes and complications. Liver transplantation can be required in some cases.

Key words: liver, diseases, cirrhosis, diagnosis, management.

INTRODUCTION

Cirrhosis, as a nowadays disease, is characterized by fibrosis and nodule formation of the liver. In the secondary plan, it is known as a chronic injury, which leads to alteration of the normal lobular organization of the liver. A complex of factors, such as life style, or environmentals, can injure the liver, and beside also including viral infections, toxins, hereditary [3, 7, 10]. With each injury, the liver suffer alterations as fibrosis. Finally but after a long-standing injury, liver functionalteration, develop in time cirrhosis as a complex diseases.

Ethiology of the chronic liver diseases usually progress unfortunately in cirrhosis, following pathological mechanisms. In the research, the most common causes of cirrhosis are hepatitis C virus (HCV), alcoholic liver disease, and nonalcoholic steatohepatitis (NASH). Hepatitis B virus (HBV) and HCV are the most common causes [1, 4, 11]. Other ethiological points of cirrhosis include autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis, hemochromatosis, Budd-Chiari syndrome, Wilson disease, alpha-1 antitrypsin deficiency, drug-induced liver cirrhosis, and chronic right-sided heart failure [1].

The cause of morbidity and mortality in cirrhosis is the development of portal hypertension and hyperdynamic circulation. Portal hypertension develops secondary to fibrosis and vasoregulatory alterations [2, 9,14].

Liver fibrosis it is known by excessive synthesis and deposition of connective tissue proteins. Interstitial collagens in the extracellular matrix of the liver has been discovered in this liver pathology. Hepato-

cytes alterations, results of an abnormal wound healing in response to chronic liver injury, previously mentioned in this written pages. The long term stimuli involved in the initiation of fibrosis leads to oxidative stress. Next point that concure to disease include mediators of molecular events involved in the pathogenesis of hepatic fibrosis. These processes lead to cellular injury and initiate inflammatory responses. As a response, cytokines and growth factors play a role as trigger activation and transformation of resting hepatic stellate cells into myofibroblast like cells. At the end of process, start an excessive synthesis of connective tissue proteins, including collagens. Uncontrolled and hepatocyte fibrosis results in distortion of lobular architecture of the liver. Pathologists show the nodular formations in the liver as a diagnosis of cirrhosis. The liver strucure injury and regeneration process could also results in genomic aberrations and mutations. Finally, develop hepatocellular carcinoma. This review try to cover various aspects of the molecular mechanisms involved in the pathogenesis of hepatic fibrosis. A great point of our this scientific orientation, is hepatic fibrosis diagnosis, with special emphasize on N-Nitrosodimethylamine (NDMA; Dimethylnitorsmaine, DMN) as the inducing agent [3, 4, 8, 12].

Prevention and treatment of liver cirrhosis are best done by an interdisciplinary medical team. This scientifically team, include a pathologist, a gastroenterologist, liver surgeon. Beside, a medical team include nurse practitioner, primary care provider and an internist. Liver cirrhosis is associated with systemic complications that can cause death of pacients. A liver transplant is not an option from different causes.

Weight loss of at least 7 % is good for reneval pathological alterations of the liver structure.

Therapy methods and drugs, are more important, including antiviral medications in viral hepatitis, steroids, and immunosuppressant agents in autoimmune hepatitis, ursodeoxycholic acid and obeticholic acid in primary biliary cholangitis, copper chelation in Wilson disease, and iron chelation and phlebotomy in hemochromatosis [4].

The aim of this article was to identify the best available evidences analyzing liver samples, normall and pathological.

MATERIALS AND METHODS

In order to analize morphological structural particularities, samples liver collected during necropsy, from healthy patients and from patients diagnosed with cirrhosis.

Following this purpose, were made permanent preparations that were stained with hematoxylin and eosin for observation at optical microscope. The process of the permanent microscopic preparations was based on prior knowledge of the steps from the classical method, using a standard H&E staining technique. Also normally liver samples were observed by optical microscope using Goldner Szekely trichrome stains. Optical microscope examination was used lens with magnification x10 and x40.

RESULTS AND DISCUSSION

The functional unit of the liver is the lobule with hexagonal form. Kienann space is specific for liver strucutre, including a portal triad (portal vein, hepatic artery, bile duct) sits at each corner of the hexagon. Mitochondri as points observing with lens x40. Portal vein with enlarge lumen (fig. 1).

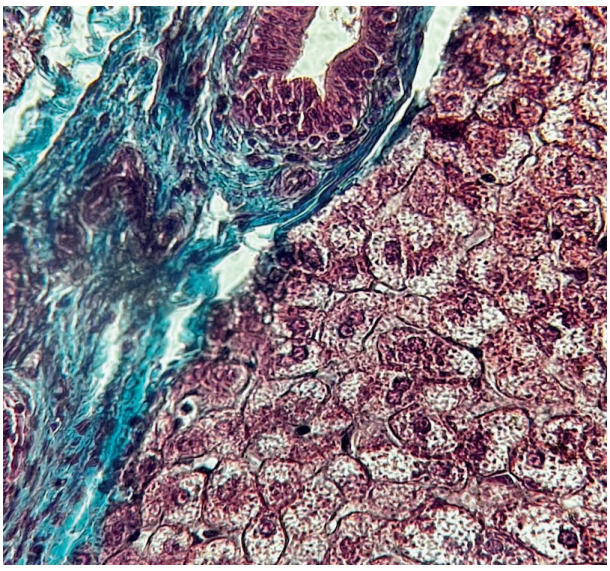

Figure 1 – Normal liver x40 Goldner – Szekely stain

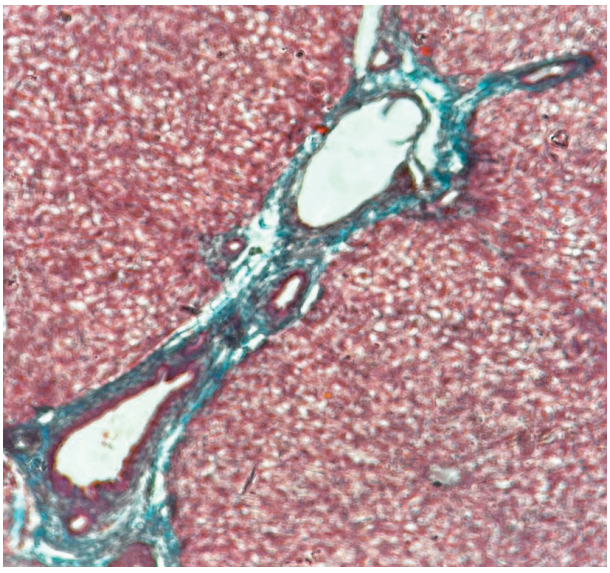

Figure 2 – Normal liver x10 Goldner – Szekely stain

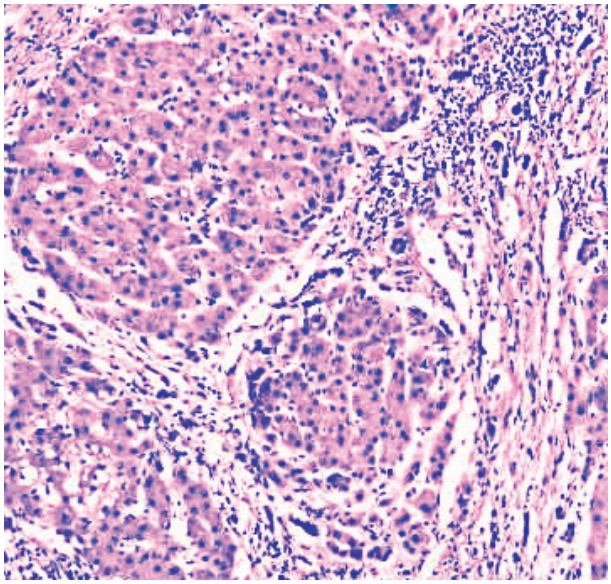

Figure 3 – Cirrhosis liver x10 H&E stain

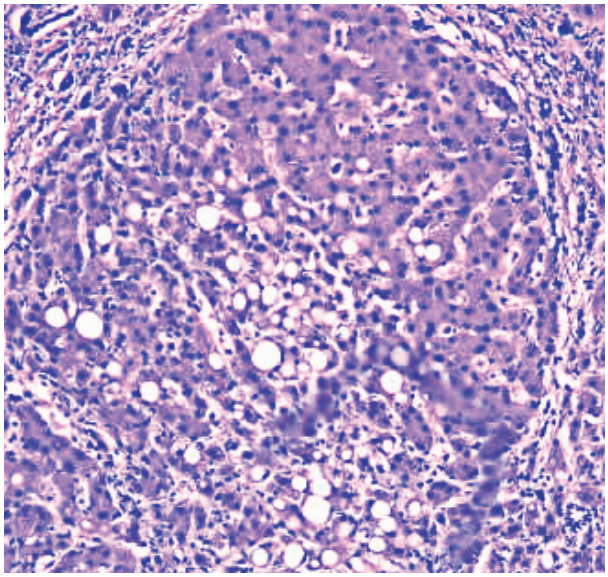

Figure 4 – Cirrhosis liver x10 H&E stain

Based on function and perfusion, hepatocytes are divided into three zones.

1. Zone I is considered to be the periportal region of hepatocytes and are the best perfused and first to regenerate due to their proximity to oxygenated blood and nutrients. Implication in oxidative metabolisms.

2. Zone II is defined as the pericentral region of the hepatocytes.

3. Zone III has the lowest perfusion due to its distance from the portal triad. Implication role in detoxification.

Also Kiernann space, hepatocytes, connective septa, in normal liver (fig. 2).

Cirrhosis is a result of continuous liver injury, inflammation, fibrosis, and necrosis. Commonly cause cirrhosis are chronic hepatitis B and C and also life style including alcoholism. The fibrosis present in cirrhosis occurs from the secretion of TGF-beta from the Ito cells in the space of Disse (fig. 3).

Cirrhosis usually represents with end-stage liver disease. Hepatitis C is the most damaging. Cirrhosis develops after a period of inflammation. The ill liver has parenchyma with fibrotic tissue and regenerative nodules (fig. 4).

Liver fibrosis impairs hepatic function and causes structural change with different types of dameges [5]. Clinically, liver cirrhosis is the severe period of chronic liver diseases. Early prevention and treatment of the causes of develop-

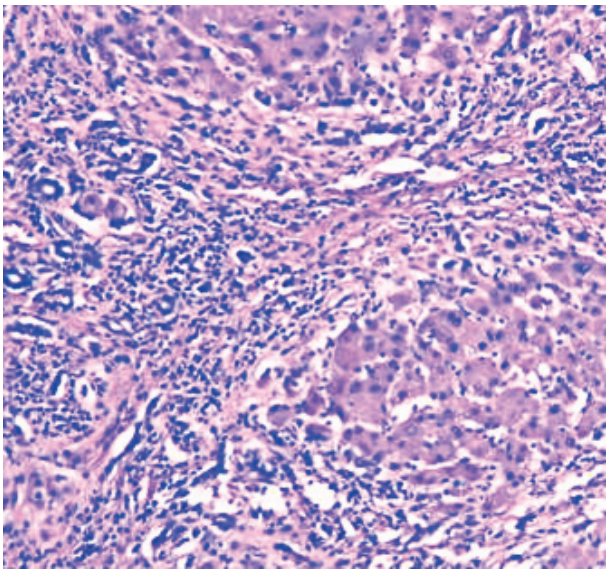

Figure 5 – Cirrhosis liver x10 H&E stain

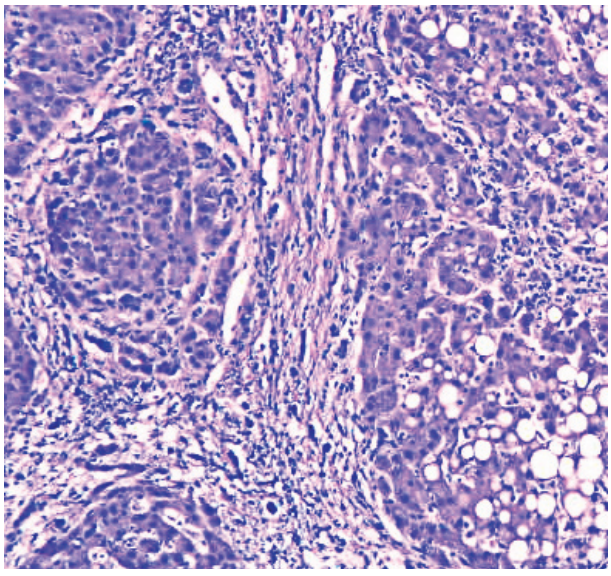

Figure 6 – Cirrhosis liver x10 H&E stain

ment and progression and pathogenic mechanism may slow down or reverse liver cirrhosis and its severe complications. Decompensated liver cirrhosis and its complications, take attention to the clinicians. Various clinically signs as ascites, esophagogastric variceal bleeding, hepatic encephalopathy, acute kidney injury, and hepatocellular carcinoma, could be observing at the medical examination. Clearly that patients' quality of life is affected in liver cirrhosis.

Liver fibrosis score, standard ultrasonography, and transient elastography are important for practicum. Also useful in identifying ill patients with no to minimal fibrosis or advanced fibrosis., medical tests [6]. In this medical direction, chronic liver disease management includes directed counseling, laboratory testing, and ultrasound monitoring.

The management of liver cirrhosis is centred on the clinical part. The proper treatment of the causes and complications and liver transplantation could be prioritaire.

The Child-Pugh score and model for end-stage liver disease (MELD) score are both used to assess and determine prognosis in cirrhotic patients. The MELD score uses creatinine, bilirubin, and INR. While both are used to create a predictive model for cirrhotic patients, the MELD score is the scale of choice for the evaluation of liver transplant patients.

Patient lifestyle changes, unfortunately cannot cure cirrhosis. Complications accompanying hepatic cirrhosis include, portal hypertension, edema in the abdomen and lower extremities, splenomegaly, infections, hepatic encephalopathy.

Behavioral modifications can prevent or at least delay disease progression and provide symptomatic relief.

Lifestyle changes, include factors, as eliminating ethanol consumption and dietary interventions as possible low-sodium diet, in order to reduce water retention. Regulate protein intake according to their doctor's directions and some medical recommandations, will be proper in the treatment of cirrhosis.

Diferential diagnosis of cirrhosis include research directions reffering **to** neonatal iron storage diseases, HELLP(hemolysis, elevated liver enzymes, low platelets) syndrome of pregnancy, idiopathic drug reaction. More than, other diseases are included in the diferential diagnosis of cirrhosis. This are Tyrosinemia, Galactosemia, Fructose intolerance

CONCLUSIONS

HCC is the known common primary cancer in the liver. HCC has nowadays an incidence in increasing [5]. Cirrhosis secondary to HBV and HCV is one of the common risk factor for liver degeneration in cirrhosis.Practically monitoring of cirrhotic patients is recommended, with at least six monthly screenings. For monitorisation of liver disease, abdominal ultrasonography is better [6]. Liver biopsy is the gold standard technique highly promising non-invasive methodology under development, that are used in diagnosis. Liver transplantation (LT) is also an effective therapeutic option for the management of cirrhosis end-stage.Relatively recently research investigations try to elucidate the signal transduction pathways that link hepatocytes alterations including cellular disfunctionality.

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Conflict of interest. No conflict of interest has been declared.

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МОРФОЛОГИЧЕСКИЕ АСПЕКТЫ НОРМАЛЬНОЙ И ПАТОЛОГИЧЕСКОЙ ПЕЧЕНИ

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Введение. Хронические заболевания печени активируют степень повреждения гепатоцитов. Патология печени по типу цирроза развивается после длительного периода патологических изменений. Важным моментом является воспаление, которое приводит к замене здоровой паренхимы печени фиброзной тканью и регенеративными узелками. Наряду с этим прогрессирующая портальная гипертензия, системное воспаление и печеночная недостаточность приводят к развитию цирроза печени. Менеджмент настоящей патологии печени сосредоточен на этиотропной терапии и лечении осложнений. В отдельных случаях может потребоваться трансплантация печени.

Цель данной статьи – выявить наилучшие доступные доказательства, анализирующие образцы печени, нормальные и патологические.

Материалы и методы. Были приготовлены фиксированные препараты, окрашенные двумя способами. Использовали гемоатоксилин и эозин, а также проводили трихромное окрашивание по Гольднеру – Секели. Микроскопировали под оптическим световым микроскопом при увеличении х10 и х40. Образцы печени отбирали при аутопсии от пациентов без патологии печени и от пациентов с диагнозом цирроз печени.

Результаты и обсуждение. Микроскопия х10 в нормальных образцах печени, окрашенных трихромными красителями по Гольднеру – Секели видны гепатоциты, пространство Кирнана, соединительные перегородки. Наряду с этим, для сравнения представлена микроскопия печеночной ткани пациентов с патологией печени, окрашенных классическим методом H&E. Воспаление является важным моментом, приводящим к замещению здоровой паренхимы печени фиброзной тканью и регенеративными узелками. Кроме того, прогрессирующая портальная гипертензия, системное воспаление и печеночная недостаточность приводят к развитию цирроза печени.

Выводы. Наш вклад в данной статье связан с воздействием физических, психологических и физиологических факторов. Все эти ранее упомянутые факторы оказывают большое влияние на качество жизни взрослых пациентов с циррозом печени. Лечение этой патологии печени сосредоточено на лечении причин и осложнений. В некоторых случаях может потребоваться трансплантация печени.

Ключевые слова: печень, заболевания, цирроз печени, диагностика, лечение.

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ҚАЛЫПТЫ ЖӘНЕ ПАТОЛОГИЯЛЫҚ БАУЫРДЫҢ МОРФОЛОГИЯЛЫҚ АСПЕКТІЛЕРІ

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Кіріспе. Бауырдың созылмалы аурулары гепатоциттердің зақымдану дәрежесін белсендіреді. Цирроз сияқты бауыр патологиясы ұзақ уақыт бойы патологиялық өзгерістерден кейін дамиды. Маңызды сәт – бұл сау бауыр паренхимасын талшықты тінмен және регенеративті түйіндермен ауыстыруға әкелетін қабыну. Сонымен қатар прогрессивті порталдық гипертензия, жүйелі қабыну және бауыр жеткіліксіздігі бауыр циррозының дамуына әкеледі. Ағымдағы бауыр патологиясын басқару этиотропты терапияға және асқынуларды емдеуге бағытталған. Кейбір жағдайларда бауыр трансплантациясы қажет болуы мүмкін.

Бұл мақаланың мақсаты – қалыпты және патологиялық бауыр үлгілерін талдайтын ең жақсы дәлелдемелерді анықтау.

Материалдар мен әдістер. Бекітілген препараттар екі әдіспен боялған: гемоатоксилин мен эозин жане Голднер – Секели трихромды бояумен. Микроскопия х10 және х40 үлкейту оптикалық жарық микроскопымен жүргізілді. Бауыр тінінің үлгілері бауыр циррозы бар және бауыр патологиясы жоқ пациенттерден аутопсия кезінде алынды.

*Нәтижелер және талқылау***.** Объективті үлкейту х10 микроскопия нәтижелері: Голднер-Секели трихром әдісімен боялған қалыпты бауыр үлгілерінде гепатоциттер, Кирнан кеңістігі және дәнекер қабықшалары көрінеді. Сонымен қатар, салыстыру үшін классикалық H&E әдісімен боялған бауыр патологиясы бар науқастардың микроскопиясы ұсынылған. Қабыну сау бауыр паренхимасының фиброзды тінмен және регенеративті түйіндермен алмастырылуына әкелетін маңызды фактор болып табылады. Сондай-ақ, прогрессивті порталдық гипертензия, жүйелі қабыну және бауыр жеткіліксіздігі цирроздың дамуына әкеледі.

Қорытынды. Бұл мақаладағы біздің үлес физикалық, психологиялық және физиологиялық факторлардың әсеріне қатысты. Жоғарыда аталған факторлардың барлығы циррозы бар ересек пациенттердің өмір сүру сапасына үлкен әсер етеді. Бұл бауыр ауруын емдеу себептер мен асқынуларды емдеуге бағытталған. Кейбір жағдайларда бауыр трансплантациясы қажет болуы мүмкін.

Кілт сөздер: бауыр, аурулар, бауыр циррозы, диагностика, емдеу.