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MAIN PATHOMORPHOLOGICAL CHANGES OF LIVER TISSUES IN EXPERIMENTAL METABOLIC SYNDROME

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In terms of morbidity and prevalence, metabolic syndrome belongs to the group of diseases of civilization that is becoming a pandemic. Some researchers mention the formation of metabolic syndrome in childhood. Pathomorphological changes in the structural organization of liver components acquire the manifestation of the main pathological reactions of stromal and parenchymal elements. To determine the main pathological processes and pathohistological changes as criteria for metabolic syndrome we developed the experimental model of metabolic syndrome on mature male Wistar rats which was patented. The semifine sections were made of liver tissues and stained with toluidine blue and examined under the microscope. The histological structure of experimental animals' livers had partial decompensation of hepatic tubules and edema of the parenchyma. Hepatocytes were polygonal in shape and located densely without clear boundaries. They had the form of "stone-block pavement" and significantly swollen with light cytoplasm, the Kraevsky cells were formed. A moderate number of the binuclear hepatocytes, small groups of the liver cells with the nuclei having one and two nucleoli, and signs of nuclear fission were found. Beam-radial structure of lobules was absent against the background of hydropic and fatty degeneration of hepatocytes and small focal necrosis. The central veins were full-blooded, their endothelium was with signs of desquamation, and the basement membrane was thickened. The portal tracts are expanded due to edema. The formation of single necrosis sites was noted in the portal area. Destructive changes in bile ducts, dystrophic changes of a vascular endothelium were noted in the damaged portal tracts. Uneven expansion and edema of the perisinusoidal spaces of Disse were observed. Swelling of Kupffer cell nuclei was noted.

Key words: binuclear hepatocytes, hepatocyte dystrophy, hepatocyte necrosis, Kupffer cells.

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ОСНОВНІ ПАТОМОРФОЛОГІЧНІ ЗМІНИ ТКАНИН ПЕЧІНКИ ПРИ ЕКСПЕРИМЕНТАЛЬНОМУ МЕТАБОЛІЧНОМУ СИНДРОМІ

За показниками захворюваності та поширеності метаболічний синдром відносять до групи хвороб цивілізації що набуває ознак пандемії. Ряд дослідників наголошують на формуванні метаболічного синдрому у дитячому віці. Патоморфологічні зміни у структурній організації компонентів печінки набувають прояву основних патологічних реакцій стромальних і паренхіматозних елементів. Для визначення основних патологічних процесів та патогістологічних змін, як критеріїв метаболічного синдрому ми розробили на статевозрілих щурах-самцях лінії Вістар експериментальну модель метаболічного синдрому, на яку отримали патент України. З тканин печінки виготовляли напівтонкі зрізи які фарбували толуїдиновим-синім та піддавали мікроскопічному вивченню. Гістологічна структура печінки експериментальних тварин мала часткову декомпенсацію печінкових балок та набряк паренхіми. Гепатоцити набували полігональної форми, були розташовані щільно, без чітких меж, у вигляді «бруківки», значно набряклі, із світлою цитоплазмою, з утворенням клітини Краєвського. Знайдена помірна кількість двоядерних гепатоцитів, невеликі групи збережених печінкових клітин, що мають ядра з одним і двома ядерцями та ознаки поділу ядерного матеріалу. Балочно-радіальна будова часточок відсутня на тлі гідропічної та жирової дистрофії гепатоцитів і дрібновогнищевих некрозів. Центральні вени були повнокровні, а їх ендотелій з ознаками десквамації, базальна мембрана потовщена. Портальні тракти розширені за рахунок набряку. В припортальній зоні відзначено формування поодиноких вогнищ некрозів. В пошкоджених портальних трактах відзначалися деструктивні зміни жовчних протоків, дистрофічні зміни ендотелію судин. Спостерігається нерівномірне розширення та набряк перисинусоїдних просторів Діссе. Відмічається набухання ядер клітин Купфера.

Ключові слова: двоядерні гепатоцити, дистрофія гепатоцитів, некроз гепатоцитів, клітини Купфера.

The study is a fragment of the research project "Influence of environmentally hazardous factors on the mechanisms of civilization diseases development and their correction by physiologically active substances", state registration No. 0122U002033.

The modern civilized world is characterized by a certain list of "benefits", which are attributed by all sanologists to the risk factors for complex diseases with high percentage of fatal consequences: consumption in excess products with high carbohydrates and animal fats, daily stressful lifestyle and a sedentary lifestyle. This basis forms numerous pathological changes in the human body, which lead to impaired structures of the cardiovascular system, liver and pancreas with metabolic disorders and the formation of insulin resistance, visceral obesity and type 2 diabetes mellitus (DM2). Such a symptom complex in modern medicine is called metabolic syndrome (MS), and the processes of liver damage – non -alcoholic fatty liver disease (NFLD) [3].

MS is attributed to a group of civilization diseases that acquires signs of pandemic. The prevalence of MS in the world has catastrophic indices. Thus, according to WHO, 59.1 % of Ukrainians had overweight in 2019, and 24.8 % had obesity.

Approximately the same indices are observed in the USA, Russia, Canada, Europe. In Japan and China – up to 10.6 %, but in the last decade, the figures have increased significantly not only in developed economies, but also countries with middle and below middle level citizens [7, 9, 12].

More and more authors are emphasizing the formation of MS in childhood and long before the clinical manifestations of CD2 or diseases of the CVS (cardiovascular system) organs. Therefore, MS in the modern clinic is also recognized as a pediatric problem, and the frequency of NFLD is observed from 17.5 to 50 % of the child's population [12].

It is well known that the liver is the central organ where all known metabolism ways are crossed and it is in this organ that the first morphological restructuring of structural organization begins under the influence of metabolic factors.

Therefore, the study of pathomorphological changes in the structural organization of liver components creates prerequisites for understanding the key pathophysiological progenetic reactions of stromal and parenchymal elements. On the basis of the above, we developed the experimental MS model on the mature rats of Vistar-males, for which the patent of Ukraine was received [11].

In the available literature, we observed the research of authors who sought to give a typical morphological characteristic of the rat liver after experimental alimentary obesity. Having performed numerous morphometric studies, the authors concluded that there were structural changes in parenchymal cells, accompanied by functional tension of capillary-connective tissue structures, disorders of blood and lymph circulation in the liver [6].

In other works [5], the authors, having carried out morphological analysis, argue for the interconnection of development of fatty dystrophy in the parenchyma of the liver and stimulation of functional activity of hepatocytes on the basis of a certain increase in nuclear-cytoplasmic ratio.

In recent years, the attention of researchers gradually attracted the peculiarities of reverse pathomorphological changes of the liver against the background of correction with various medicines [3], and completeness and complex morphogenesis remain at the general level of coverage.

The study of human biopsy histological material is characterized by pathomorphological changes in the form of large-drop fatty dystrophy of hepatocytes with inflammatory cellular infiltration, balloon dystrophy of hepatocytes and perisinoidal and pericellular fibrosis of varying degrees of expression [6].

In similar studies [13], the author also points out the polymorphism of pathohistological changes with the preserved general structure of the liver in the form of deformation of bodies and nuclei of hepatocytes in different areas due to fatty dystrophy, bridge necrosis and increase in the area of the hepatocyte profile with a decrease in the area of the nucleus profile.

All of the above signs characterize the polymorphism of pathomorphological changes that last for years, and patients do not complain for a long time, in such cases it is impossible to characterize morphological status in the initial stages of MS development. Thus, there is a need to establish a complex of basic signs of pathomorphological changes of liver tissues inherent in the experimental MS.

The purpose of the study was to establish the main pathomorphological processes that lead to changes in liver tissues in experimentally modeled metabolic syndrome in Vistar rats.

Materials and methods. The experiment involved sexually mature rats in connection with the convincing epidemiology of MS in men [3, 7, 8, 9]. For this fragment of the experiment, all animals were divided into an intact group and those who were modulated by the MS. The Patent for a utility model "Metabolic Syndrome Modeling Methodology Method" was registered under No. 122249 of 26.12.2017 [11]. The maintenance and use of animals was carried out in accordance with the developed resolutions "General ethical principles of animal experiments", approved by the VII National Congress on Bioethics in 2019 and the Law of Ukraine of 13.02.2020 [1, 2].

After fulfilling all the necessary conditions for performing the experiment, rats were sacrificed with thiopental anesthesia and further decapitation. For a comprehensive determination of MS morphological features and obtaining histological material, the rat body was cut by a ventral median incision.

The liver tissues were targeted and collected from main four sections of the liver to objectify possible topographic and anatomical interpretations. The pieces of 1×1×2 mm were formed with razor blades and immersed in a solution of 4 % glutaraldehyde for fixing and further manufacturing of epoxy blocks. The latter were used to make semi-thin sections (up to 2 μm), which were stained with toluidin-blue and embedded in polystyrene for constant storage according to conventional methods [4].

For microscopic examination of the resulting material, we used the Optica trinocular microscope Series B510, with a "SIGETA M3 SMOS 8500" digital chamber and image processing software. The resulting images were subsequent to morphometric processing, using the Toup Viwe software ToupTek Version 3.7.13127.2018.1016.

The relative indices of the structural components of the liver tissue were determined: S of hepatocytes' cytoplasm, S the of hepatocytes' nuclei, nuclear-cytoplasmic ratios, the number of single- and two-core hepatocytes, their ratio, the number and area of sinusoid cells. Variation-statistical processing of

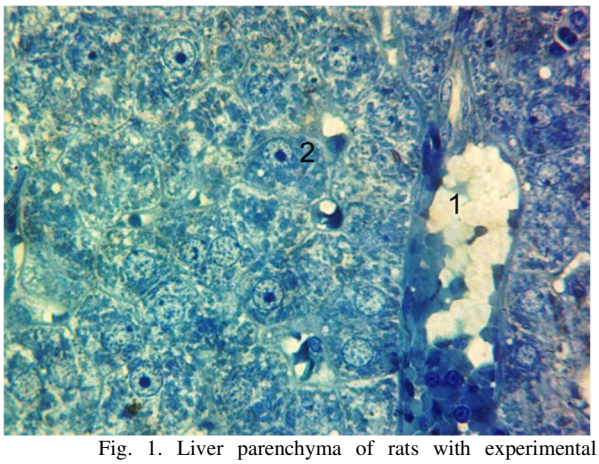

Fig. 1. Liver parenchyma of rats with experimental metabolic syndrome. 1 – vein of moderate blood supply, 2 – polygonal hepatocytes, located concisely in the form of "paving stones" (Kraevsky cells). Semi-thin slice. Staining with toluidine blue. Magnification x100.

digital measurements was carried out using the Microsoft Excel software with the definition of the following algorithm: arithmetic mean (M), meansquare deviation (σ), mean error of arithmetic mean (m).

Results of the study and their discussion. In the conditions of experimental metabolic syndrome in the liver of experimental animals signs of its morphological structure and homeostasis impairment were found. The histological structure of the liver in experimental animals has partial decompensation of liver beams and parenchymal edema. Hepatocytes acquire a polygonal shape, located compactly, in the form of "paving stones", significantly swollen, with a light cytoplasm, which indicates the mobilization of glycogen (the so-called Kraevsky cells) (fig. 1).

Such structural changes indicate that hepatocytes lose glycogen, undergo hydropic dystrophy, resulting in anoxic necrosis in the central region of the liver lobe (centrolobular necrosis). A moderate number of dinuclear hepatocytes was found in histological specimens of experimental MS. Small groups of preserved liver cells with signs of nuclear material division (fig. 2A). The beam-radial structure of the lobes is erased against the background of hydropic and fatty dystrophy of hepatocytes and small focal necroses, preserved hepatocytes have nuclei with one or two nucleoli (fig. 2B).

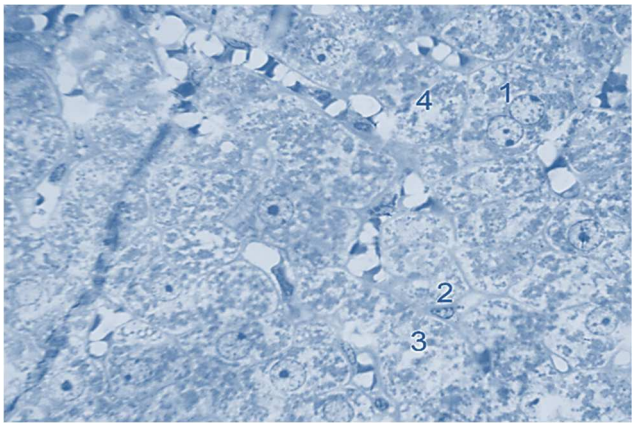

Fig. 2A. Parenchyma of rat liver with experimental MS. 1 – dinuclear hepatocytes. 2 – Kupffer cells. 3 – necrosis of hepatocytes. 4 – small cells with the nuclear material division. Semi-thin slice. Staining with toluidine blue. Magnification x100.

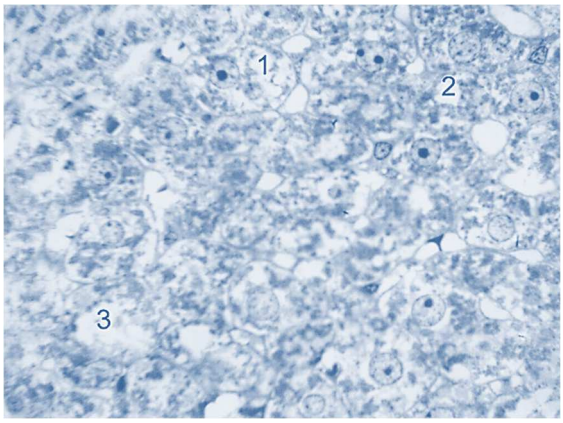

Fig. 2B. Parenchyma of rat liver with experimental MS. 1 – hydropic dystrophy of hepatocytes, 2 – fatty dystrophy of hepatocytes, 3 – necroses of hepatocytes. Semi-thin slice. Staining with toluidine blue. Magnification x100.

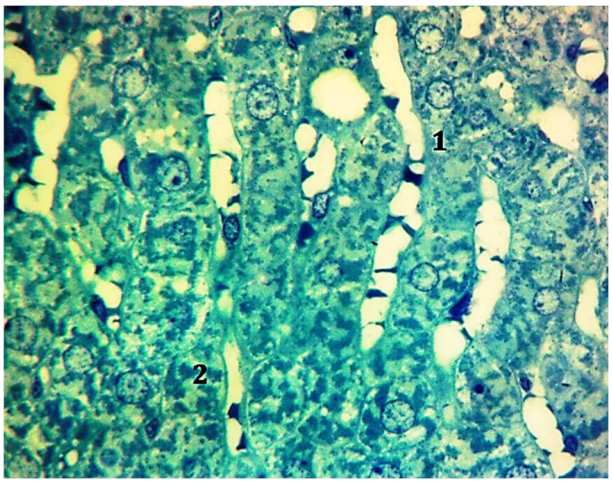

Fig. 3A. Liver parenchyma of rats with experimental MS. 1 – uneven expansion (edema) of the perisinusoidal spaces of Disse. 2 – necrosis of hepatocytes. Semi-thin slice. Staining with toluidine blue. Magnification x100.

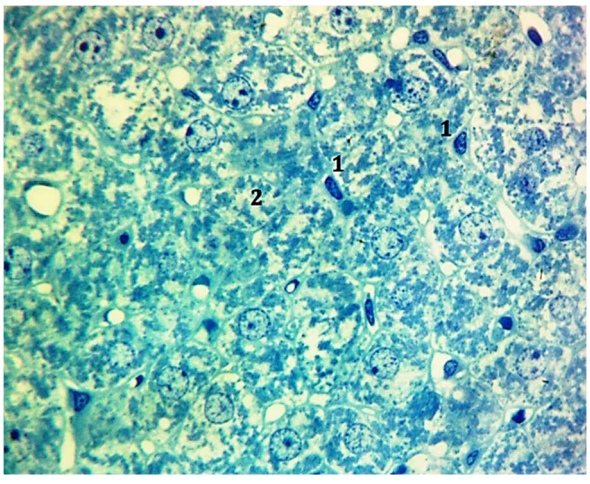

Fig. 3B. Liver parenchyma of rats with experimental MS. 1 – swelling of the nuclei of Kupffer cells. 2 – necrosis of hepatocytes. Semi-thin slice. Staining with toluidine blue. Magnification. 100.

Central veins are full-blooded, their endothelium has signs of desquamation, and the basement membrane is thickened. Portal tracts are expanded due to edema. The formation of single foci of necrosis was noted in the portal zone. Destructive changes of bile ducts, dystrophic changes of a vascular endothelium were noted in the damaged portal tracts. These changes are spreading in the port-portal direction. Uneven expansion (edema) of perisinusoidal Disse spaces is noted (fig. 3A). Most sinusoids are dilated, swelling of Kupffer cell nuclei is noted (fig. 3B).

In the study of morphometric parameters of liver tissue structures, the following results were obtained presented in Table. 1.

Table 1

Morphometric parameters of liver cells

Indices		Intact liver	Metabolic syndrome
Cross-sectional area, mμ	Hepatocyte	400.25 ± 49.68	488.08 ± 44.51
	Nucleus	59.88±10.59	59.69 ± 8.48
	Cytoplasm	340.37±45.77	428.39±40.97
Nuclear-cytoplasmic ratio		0.18±0.03	0.14±0.02
Number of hepatocytes in the field of view of the microscope at magnification ×400	Mononuclear	26.21±3.56	20.36±2.46
	Binuclear	4.57±1.14	4.29±1.08
Ratio of the number of binuclear hepatocytes to the number of mononuclear hepatocytes		0.18±0.04	0.22±0.07
Area of sinusoidal cells		20.19±4.58	21.36±5.12
Number of sinusoidal cells		39.93±4.36	19.36±3.46
Ratio of the sinusoidal cells number to the number of mononuclear hepatocytes		1.54±0.15	0.22±0.07

The cross-sectional area of the hepatocyte is characterized by an increase in the experimental group to 488.08±44.51 μm compared to hepatocytes in the intact group of animals 400.25±49.68 μm. This increase is due to an increase in cytoplasmic area from 340.37±45.77 μm in the intact group to 428.39±40.97 μm in experimental MS, and the cross-sectional area of the nucleus remained almost the same in both groups. Therefore, the nuclear-cytoplasmic ratio decreased in the experimental group to 0.14±0.02 compared to the intact 0.18±0.03.

The next morphometric manifestation of MS was a general decrease in the number of mononuclear hepatocytes to 20.36±2.46 compared to intact liver 26.21±3.56, and the mean indices of binuclear remained almost the same in both groups.

Fluctuations in the area of sinusoidal cells are insignificant, but their number decreases significantly from 39.93±4.36 in intact tissue to 19.36±3.46 in the experimental group.

The next main morphological component of experimental MS manifestation was a significant decrease in the ratio of the number of sinusoidal cells to the number of mononuclear hepatocytes, which was in the experimental group 0.22±0.07 at 1.54±0.15 in the intact group.

The issue of experimental metabolic syndrome in time is quite relevant due to the high prevalence of liver damage among the population of Ukraine, the consequences of which destroy the cardiovascular system and affect glucose uptake causing diabetes 2. Therefore, the study of the main signs of histopathological changes in liver tissue – diagnostic criteria for the MS pathogenesis and a reasonable etiopathogenetic approach to treatment.

Having developed an experimental model on male Vistar rats, we investigated the relative indices of liver tissue structural components: cytoplasmic area of hepatocytes, area of their nuclei, nuclear-cytoplasmic ratios, number of mononuclear and binuclear hepatocytes and their ratios, number and area of sinuses. In addition to morphometric characteristics, we identified a number of pathomorphological signs of inflammatory and destructive-degenerative processes in liver tissue that accompany the alternative influence of pathogenetic factors.

Among the main pathohistological manifestations the change in the shape of hepatocytes to polygonal with a dense location, which takes the form of “paving stones” was determined. Such changes occur due to the increase in the size of hepatocytes in MS due to the increase in its cytoplasm. Microscopically, they are significantly swollen, have a light cytoplasm, which indicates the mobilization of glycogen with the formation of Kraevsky cells. When glycogen is lost, hepatocytes undergo hydropic dystrophy, resulting in anoxic centrilobular necrosis.

Fatty dystrophy of hepatocytes is observed, which in combination with the above processes and edema of the parenchyma tissue structure loses the beam-radial structure of the lobules. Therefore, in morphometric analysis, we observe a general decrease in the number of mononuclear hepatocytes compared to intact liver.

Pathomorphological changes in the structure are also observed on the part of the stroma components. Thus, plethora is determined in the central veins, their endothelium shows signs of desquamation, and the basement membrane thickens. Portal tracts are expanded due to edema. In the peri-portal area there is the formation of single foci of necrosis and destructive changes in the bile ducts and dystrophic changes in the vascular endothelium. Uneven expansion due to edema of the perisinusoidal spaces of Disse was noted. Therefore, the fluctuations in the area of sinusoidal cells were insignificant, but their number decreases significantly with the ratio of the number of sinusoidal cells to the number of mononuclear hepatocytes in the experimental group.

The structural organization of liver tissues in intact rats did not differ fundamentally from the structure of this organ, which was presented in scientific papers previously published in available scientific sources [5, 6, 9, 11] indicating specific formations such as central vein, endothelial cells, liver beams, hepatocytes and Kupffer cell. Therefore, we consider it inexpedient to cover this issue, and focus on the peculiarities of the liver tissue reorganization in experimental animals.

These changes spread to all parts of the liver and indicate diffuse chronic inflammatory processes, and the constancy of the number of binuclear hepatocytes in both groups indicates moderate regeneration of the liver parenchyma at the intracellular level in MS [5].

Conclusions

1. The main pathological processes occurring in the liver tissue in the experimental metabolic syndrome include edema of the parenchyma, hydropic dystrophy, anoxic centrilobular necrosis, fatty degeneration of hepatocytes.

2. The main histopathological changes occur both in the parenchyma and in the stroma of the organ. In the parenchyma: change in the shape of hepatocytes, their dense location (appearance of “paving stones”), light cytoplasm, the presence of Kraevsky cells, fatty dystrophy, centrolobular necrosis, signs of edema, loss of beam-radial structure of the lobules. The components of the stroma: in the central veins plethora is determined, the endothelium has signs of desquamation, the basement membrane is thickened, portal tracts dilated due to edema, in the peri-portal zone – formation of single necrosis foci and destructive changes in bile ducts, dystrophic changes of vascular endothelium, dilation of perisinusoidal Disse spaces.

3. The change in morphometric parameters is manifested in the form of an increase in the cytoplasmic area of hepatocytes, a decrease in the number of mononuclear hepatocytes, a significant decrease in the number of sinusoidal cells and a decrease in the ratio of sinusoidal cells to the number of mononuclear hepatocytes.

Prospects of further research in this direction will include morphological and biochemical studies of the liver in experimental models of MS and the determination of cyto-histological characteristics in the correction with polyphenols.

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PECULIARITIES OF INNERVATION OF MUSCLES AND SKIN OF THE ANTERIOR AND LATERAL ABDOMINAL WALLS IN HUMAN FETUSES

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Considering theoretical and practical importance of data regarding fetal anatomical variability of intercostal nerves, iliopsoas and inguinal nerves, the study was performed on 44 preparations of human fetuses of 231.0–375.0 mm parietococcygeal length using macromicroscopic preparation, superficial staining of dissected nerves, and morphometry. In some cases, in the innervation of the muscles and skin of the anterior and lateral abdominal walls took part VI (V)–XII intercostal nerves and iliohypogastric nerve, and in other – VII–XII intercostal nerves, iliohypogastric and ilioinguinal nerves. The formation of the intramuscular nerve plexus of the abdomen involves branches that begin directly from the main trunks of the intercostal nerves, and also from the lateral, anterior and additional musculocutaneous branches of the intercostal nerves, and the branches of the iliohypogastric and ilioinguinal nerves. In human fetuses, there are areas of overlap between the innervations of the muscles and skin of the anterior and lateral abdominal walls.

Key words: intercostal nerves, iliohypogastric nerve, ilioinguinal nerve, muscles of abdomen, skin of abdomen, fetus, human.

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ОСОБЛИВОСТІ ІННЕРВАЦІЇ М'ЯЗІВ І ШКІРИ ПЕРЕДНЬОЇ І БІЧНИХ СТІНОК ЖИВОТА У ПЛОДІВ ЛЮДИНИ

З огляду на теоретичну і практичну важливість даних щодо фетальної анатомічної мінливості міжребрових нервів, клубово-підчеревного і клубово-пахвинного нервів дослідження проведено на 44 препаратах плодів людини 7–10 місяців 231,0–375,0 мм тім'яно-куприкової довжини за допомогою макромікроскопічного препарування, поверхневого забарвлення відпрепарованих нервів, а також морфометрії. У плодів людини встановлено, що в одних випадках, в іннервації м'язів та шкіри передньої і бічної стінок живота беруть участь VI(V)–XII міжреброві нерви і клубово-підчеревний нерв, а в інших – VII–XII міжреброві нерви, клубово-підчеревний і клубово-пахвинний нерви. М'язам передньої і бічних стінок живота притаманна сегментарна іннервація. В утворенні внутрішньом'язового нервового сплетення ділянки живота беруть участь гілки, які починаються безпосередньо від основних стовбурів міжребрових нервів, а також від бічних, передніх і додаткових м'язово-шкірних гілок міжребрових нервів, та гілки клубово-підчеревного і клубово-пахвинного нервів. У плодів людини існують зони перекриття іннервації м'язів і шкіри передньої і бічних стінок живота. Встановлені зв'язки між шкірними гілками суміжних міжребрових нервів вказують на відсутність чіткої метамерності в іннервації шкіри передньо-бічних стінок живота.

Ключові слова: міжреброві нерви, клубово-підчеревний нерв, клубово-пахвинний нерв, м'язи живота, шкіра живота, плід, людина.

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Until now, the question of making incisions on the anterior abdominal wall in order to access the abdominal organs remains relevant. We have to mark that during laparotomy and lumbotomy are often damaged the intercostal nerves (IN) and their branches, and also the branches of the iliohypogastric and ilioinguinal nerves, which take part in the innervation of the muscles and skin of the abdominal wall. The above mentioned can lead to such complications as atrophy of the abdominal muscles, formation of postoperative hernias, development of trophic ulcers, pain syndrome, traumatic neuritis and neuroma of damaged nerves, etc. [9, 15].

In the area of the anterior abdominal wall, the number of IN varies from 1 to 4 pairs, but most often there are two pairs of IN (60 % of observations) [3]. INs penetrate the rectus abdominis muscles usually