ISSN 2306-5559 (print)
ISSN 2410-938X (online)

www.vhk.kz

А.Н.Сызғанов
атындағы
ҰЛТТЫҚ
ҒЫЛЫМИ ХИРУРГИЯ
ОРТАЛЫҒЫ

№ 4 (61) 2019

ҚАЗАҚСТАН ХИРУРГИЯСЫНЫҢ ХАБАРШЫСЫ

ВЕСТНИК ХИРУРГИИ КАЗАХСТАНА

BULLETIN OF SURGERY IN KAZAKHSTAN

АЛМАТЫ /ALMATY

№ 4 (61) 2019
Издается с 2005 г.
ISSN 2306-5559 (print)
ISSN 2410-938X (online)

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әр тоқсанда шығып тұратын А.Н. Сызғанов атындағы Ұлттық ғылыми хирургия орталығының ғылыми-тәжірибелік журналы
ежеквартальный научно-практический журнал Научного центра хирургии им. А.Н. Сызганова
a quarterly scientific-practical journal of the National Scientific Center of Surgery named after A.N. Syzganov

Журнал ҚР ақпарат, мәдениет және спорт Министрлігінде тіркелген.
«Қазақстан хирургиясының хабаршысы» ҚР ҒБМ Ғылым және білім саласындағы бақылау комитеті ұсынған Ғылыми еңбектің негізгі нәтижелерін жариялау үшін ұсынатын ғылыми баспалар тізбесі енгізілген.
Тіркеу нөмірі 5564-Ж.
Журналдың иесі – «А.Н. Сызғанов атындағы Ұлттық ғылыми хирургия орталығы» АҚ.
Баспаның мекен-жайы:
050004, Алматы қ., Желтоқсан көш. 62,
тел. 7(727) 2795306
http://vhk.kz, e-mail: kaz.vestnik@mail.ru
Әрленім және беттеу:
«*ДАНиЛЕНКО*» ЖК-де дайындалған,
Алматы қ., Чайковский көш. 23-3,
тел. 7(727) 2780172
Таралымы – 500 дана
Корректор: Нургалиева А.Н.
e-mail: aigul.nur10792@mail.ru
тел: 87786690021
Жарияланатын мақалалардың мазмұнына авторлар жауапты.
ҚАЗПОЧТА жазылу индексі – 75327

Журнал зарегистрирован в Министерстве информации, культуры и спорта РК.
Журнал включен в перечень научных изданий, рекомендуемых Комитетом по контролю в сфере образования и науки МОН РК
Регистрационный номер: 5564-Ж.
Владелец журнала - АО «Национальный научный центр хирургии имени А.Н. Сызганова».
Адрес редакции:
050004, г. Алматы, ул. Желтоксан, 62,
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Дизайн и верстка:
ИП «*ДАНиЛЕНКО*»,
г. Алматы, ул. Чайковского, 23-3,
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Корректор: Нургалиева А.Н.
e-mail: aigul.nur10792@mail.ru
тел: 87786690021
Ответственность за содержание публикуемых материалов несут авторы.
Подписной индекс КАЗПОЧТЫ – 75327

The journal is registered with the Ministry of Information, Culture and Sports of the Republic of Kazakhstan.
The "Bulletin of Surgery in Kazakhstan" is included in the list of scientific publications recommended by the Committee for Control of Education and Science, Ministry of Education and Science of Kazakhstan.
Registration number: 5564-Ж
The owner of the journal - JSC «National scientific center of surgery named after A.N. Syzganov»
Editorial address:
62, Zheltoksan street, Almaty, 050004
tel. 7 (727) 2795306
http://vhk.kz, e-mail: kaz.vestnik@mail.ru
Design and lead out:
«*ДАНиЛЕНКО*» SP,
23, Tchaikovsky street, apt.3, Almaty,
tel. 7 (727) 2780172
Edition – 500 copies.
Corrector: Nurgalyeva A.N.
e-mail: aigul.nur10792@mail.ru
tel: 87786690021
The authors are responsible for the content of published materials.
Subscript index of KAZPOST – 75327

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STRUCTURAL CHANGES IN THE THYROID AND ADRENAL GLANDS IN ACUTE HYPOXIA

МРНТИ 76.29.37

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Abstract

The article provides information about the research work carried out to study the patho- and morphogenetic features of morphofunctional changes occurring in the tissue structures of the thyroid and adrenal glands under the influence of barocamera hypoxia.

The object of the study were the thyroid and adrenal glands of adult male white rats with a mass of 180-200 grams. In the course of the study, anatomic, histological and morphometric examination methods were used.

During the morphological study of the thyroid and adrenal glands parenchyma and stroma of the glands in all animals under the infuence of acute hypoxia, diffuse edema, acute dilatation of the vessels in the microcirculatory bed, fragmentation of the walls, violation of the completeness of the endothelial layer, absorption of blood plasma into the vascular wall were observed. These dystrophic changes are more pronounced in the adrenal gland than in the thyroid gland. This is explained by the fact that the adrenal gland is more sensitive to stress factors and cells are more damaged, and the thyroid gland is more early to hypoxia.

Keywords
thyroid gland, adrenal glands, acute hypoxia, structure

Жіті гипоксия кезіндегі бүйрек үсті безінің және қалқанша безінің құрылымдық өзгерістері

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АВТОРЛАР ТУРАЛЫ

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Аңдатпа

Мақалада барокамералық гипоксияның әсерінен бүйрек үсті безінің және қалқанша безінің тіндік құрылымдарында жүретін морфофункционалдық өзгерістердің пато- және морфогенетикалық ерекшеліктерін зерттеу мақсатында жүргізілген ғылыми-зерттеу жұмыстары туралы мәліметтер келтіріледі.

Зерттеу нысаны ретінде салмағы 180-200 грамды құрайтын ақ түсті ересек егеуқұйрықтардың аталықтары алынды. Зерттеу барысында анатомиялық, гистологиялық және морфометрикалық зерттеу әдістері пайдаланылды.

Бүйрек үсті безінің және қалқанша безінің паренхималарын, бездердің стромаларын морфологиялық зерттеу кезінде жіті гипоксияның әсерінен барлық жануарларда диффуздық ісіну, микроциркуляторлық арнадағы тамырлардың жіті кеңеюі, қабырғалардың фрагментациясы, эндотелиалды қабат үздіксіздігінің бұзылуы, тамыр қабырғаларына қан плазмаларының енуі байқалды.

Бұл дистрофиялық өзгерістер қалқанша безіне қарағанда, бүйрек үсті безінде айқын көрінеді. Мұның себебі, бүйрек үсті безі күйзеліс факторларына аса сезімтал, ал қалқанша безі гипоксияға ертерек ұшырайды деп түсіндіріледі.

Түйін сөздер
қалқанша безі, бүйрек үсті безі, жіті гипоксия, құрылым

Структурные изменения щитовидной железы и надпочечников при острой гипоксии

ОБ АВТОРАХ

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Аннотация

В статье приводятся сведения о научно-исследовательской работе, проведенной с целью изучения пато- и морфогенетических особенностей морфофункциональных изменений, происходящих в тканевых структурах щитовидной железы и надпочечников под влиянием барокамерной гипоксии.

Объектом исследования служили щитовидная железа и надпочечники взрослых самцов белых крыс массой 180-200 грамм. В ходе исследования использовались анатомические, гистологические и морфометрические методы исследования.

При морфологическом исследовании паренхимы щитовидной железы и надпочечников и стромы желез у всех животных под влиянием острой гипоксии наблюдались диффузный отек, острое расширение сосудов в микроциркуляторном русле, фрагментация стенок, нарушение непрерывности эндотелиального слоя, пропитывание плазмы крови в сосудистую стенку. Эти дистрофические изменения более выражены в надпочечниках, чем в щитовидной железе. Это объясняется тем, что надпочечники более чувствительны к стрессовым факторам, а щитовидная железа более рано подвергается гипоксии.

Ключевые слова

щитовидная железа, надпочечники, острая гипоксия, строение

Currently, hypoxia – oxygen deficiency is one of the main problem in medicine, an integral part of modern human life [1]. Hypoxia is the basis of various pathological processes in a number of serious diseases and extreme conditions and is often found in the clinic. Hypoxia affects the development of ischemia in the tissues of the respiratory system, nervous system, organs of the cardiovascular system, as well as organs of the endocrine system, especially the thyroid and adrenal glands, causes polyorgan deficiency, as well as stress conditions in the body [2,3].

Thyroid and adrenal glands, which are the main organs of the neuroendocrine system with a complex biological structure, play an important role in regulating the level of basal metabolism of all cells of the body, normal development and growth of the body, as well as in the formation of compensatory and adaptive processes in the body against various stress factors [4,5]. These glands have different origins, structure and shape. Thus, the thyroid gland consists of the central and peripheral parts, and the adrenal gland consists of the cortex and medulla, which is 70-80% of the volume of the organ.

Corticosteroids synthesized and secreted by the adrenal cortex affect the immune system, the course of inflammatory processes and metabolism, and catecholamines secreted by the adrenal medulla affect the activity of the cardiovascular and nervous system, glandular epithelium, mainly carbohydrate metabolism and thermogenesis [5]. Hormones (thyroxine, triodtrionine) synthesized and secreted by follicular epithelial cells of the thyroid gland are also important for normal growth and development of the body [6,7,8,9].

A group of authors identified barometric pressure and oxygen levels at various altitudes from sea level, as well as describing the symptoms of «mountain diseases», and found that the effect of oxygen deficiency on the body, most diseases of the thyroid [10,11] and adrenal glands, and stress conditions associated with these diseases are directly or indirectly related to oxygen deficiency. Other authors note that morphological changes occurring in the glands under the influence of hypoxia are a nonspecific reaction to stress-syndrome caused by compensatory-adaptive reorganization of the structure of the gland and brain hypoxia.

Analysis of literature data shows that despite numerous microscopic studies devoted to the structure of the tissues of the thyroid and adrenal glands, which play a key role in the life processes of the organism, the study of the hypofunctional state of organs, a number of questions still remain unresolved, contradictory and unanswered in the literature [12,13,14].

The aim of the study was to study the pathogenic and morphogenic properties of morphofunctional changes in the tissue structures of the thyroid and adrenal glands under the influence of acute hyperbaric chamber hypoxia.

Materials and methods

The object of the study was healthy adult male white rats weighing 180-200 grams. Research on animals was conducted in the Department of Phar-

macology and Experimental Surgery of the Scientific Research Center of AMU on the basis of ethical rules specified in Protocol No. 31 of the ethics rules Commission and bioethics committee under the Ministry of health of the Republic of Azerbaijan on 21.04.2008.

Animals are divided into 2 groups – control and experience groups. The animals included in the control group were not intervened, and the second group of experimental animals were experimented in the daytime (about 10-15). To this end, they were put into the barocamera for 2 hours and created a model of acute hypoxia, 5 times a week with a break of 1 hour, 2 times a day and 2 hours every other day. In the barocamera, the temperature was 19-20əC, atmospheric pressure was equal to the pressure 2000-3000 m above the sea level, the particles of natron lime (Ca(OH)2 81%+NaOH 3,4%+H2O 15,6%) were used to absorb the CO2 generated during respiration. The animals removed from the barocamera were provided with water and food and kept under control in standard vivarium conditions. On the 2nd and 5th day of the experiment, intraperitoneal anesthesia was performed by introducing 2-2.5% theopental-sodium solution (100mg/kg) into the peritoneal cavity of animals. Preparations for histological and morphometric examination were taken from the thyroid and adrenal glands of decapitated animals.

The sections are stained with hematoxilin-eosin and PAS, covered with encrusted glass through Canadian balm. Microscopic examination was performed under x8, x20 and x40 magnification. Microphotography of the structural elements of the thyroid and adrenal glands was performed by a digital camera of the microscope "Olympus BX-41", and morphometric parameters were calculated by Microsoft Excel computer program.

Results and discussion

In the rats included in the macroscopically controlled group, the thyroid gland is located at the front of the neck, in front of the trachea and the larynx, and the adrenal glands in the peritoneal area, on the upper poles of the kidneys, under the diaphragm. The relative weight of the thyroid gland of rats included in the control group is 19.5 mg/g, and the relative weight of the adrenal gland is 13,4mq/q. During the visual examination of histological preparations taken from animals, the capsule consisting of dense connective tissue covering both glands from the outside, thin septums going from the capsule to the inside of the gland, the lobules of the thyroid gland separated from each other by the trabeculae, the cortex and medullaryn substances of the adrenal gland are clearly visible. The connective tissue capsule consists of a small amount of glucosaminoglycan and other organic substances.

In the norm, the main structural components of the parenchyma of the thyroid gland are closed corpuscles or slightly tightened follicles of different sizes with a cavity inside, and the main mass of the follicles is thyrocytes. The main cellular elements of the parenchyma of the adrenal glands are the rounded, mainly small-sized adrenocytes (fig.1).

During the microscopic examination, it is possible to see follicles and a few columnar epithelial cells covering them, as well as cuboid tyrositis, round nuclei in the cytoplasm of tyrosites stained with hematoxylin-eosine. In histological sections, in the center predominate follicles made up by prizmatic cells, and follicles made up by cuboid cells in the periphery. In comparison with the periphery in the central part of the gland, the follicles differ in their small size. The size of the follicles and the thyrocytes that make them varies in normal physiological conditions. Microscopically, follicular komplexes (microvillies) consisting of a

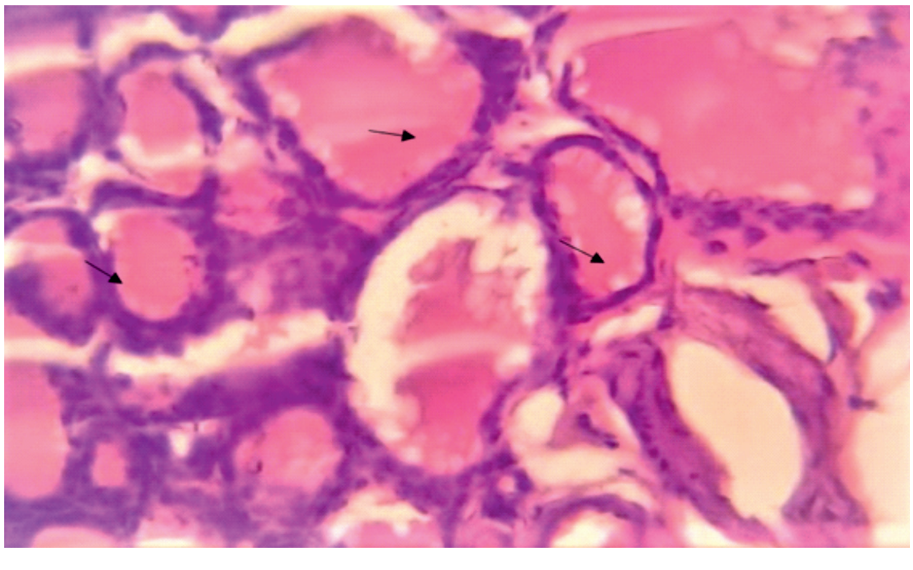

Figure 1.
Normal histological structure of the thyroid gland. Stain: Stain: Hematoxilin-eosin: x40.

group of follicles surrounded by a thin connective tissue capsule are distinguished in the lobules of the thyroid gland.

In the cavity of the follicles marked colloid, collected in liquid form. Depending on the degree of colloid filling, the volume of follicles increases. Colloid is a product of follicular endocrinocytes, mainly composed of thyroglobulin, fills the lumen of follicles in the center as a homogeneous mass, positively stained with PAS. In the peripheral part of the gland there are follicles covered with a single layer cuboid epithelium, in which the cavity is not filled with colloids. In the peripheral part of the gland, there is a single layer of gobaban-like epithelium-covered follicles with some formation of which the cavity is not filled with colloids. In histological preparations, a very elongated shape follicles filled with colloid are also detected.

In the microscope, the well-developed interlobular and interfollicular connective tissue of the thyroid gland is poorly noticeable. Angioarchitectonics of the gland is characterized by a thick network of capillaries, forming strange plexuses, which are in close contact with thyrocytes from all sides.

In the microscopic preparations taken from the adrenal glands and stained with hematoxylin-eosin, thin trabeculae going from the capsules to the thickness of the cortical substance and having the vessels and nerves inside and reticular fibers arising from them are clearly distinguished. Reticular fibers are directed towards the depth of the organ, forming a thin network around the cortex and medulla of parenchyma. Microscopically 3 cortical zones, separated from each other by a not very clear boundaries, are distinguished - the outer glomerular zone; the middle – the fascicular zone, which forms the main part (mass) of the cortical substance, and the inner thin reticular zone, resting to the medulla. The cells of the glomerular and fascicular zones are large, the nuclei are large and rounded, and the cells of the reticular zone are relatively small (fig.2).

The glomerular zone consists of cells that have a uniformly colored cytoplasm, forming arches («glomeruli»), the cells are grouped in the form of small clusters, separated by capillaries from each other. In the cytoplasm of this zone compared with the fascicular zone little fat droplets are noticeable. Under the glomerular zone, the cells of the fascicular zone with a more homogeneous cytoplasm are visible. The cells of the fascicular zone have a radial structure perpendicular to the surface of the gland. Fascicular zone consists of a large vacuolated oxyphilic cells – spongiocytes; they form a radially oriented trabeculae («bundles») having a sinusoidal capillaries. Spongiocytes have bright cytoplasm and seem vacuolated. The cells of the reticular zone are smaller compared to cells of the fascicular zone, attracting the attention by the presence of dark and light adrenocytes inside. In this area, cells subjected to apoptosis are also visible. Microscopic examination in the cytoplasm of the reticular zone found lipofuscin granules. Capillaries of the reticular zone consist of epithelial trabeculae, going in different directions and forming anastomoses with each other.

The amount of lipids in the glomerular and reticular zones of the adrenal gland is less and in the fascicular zone is more. Therefore, cells with transparent cytoplasm forming the fascicular zone are not stained with PAS. This is explained by the fact that in spongiocytes there are fewer glycogen grains, and on the contrary a large amount of fat drops.

The reticular zone of the adrenal cortex is separated from the adrenal medulla by a thin connective tissue covering. This cover like the continuation of connective tissue located perpendicular to the sur-

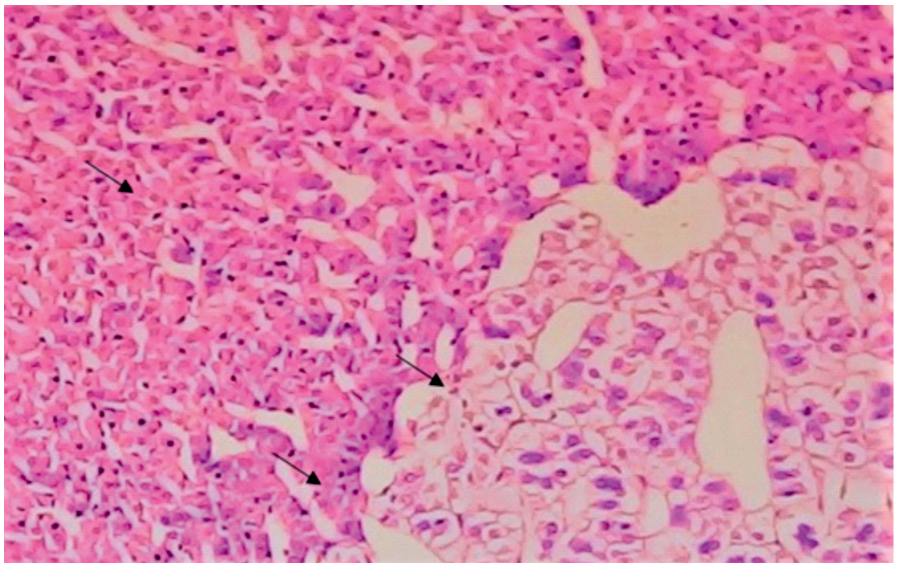

Figure 2. Normal histological structure of the adrenal gland. Stain: Hematoxilin-eosin: x20.

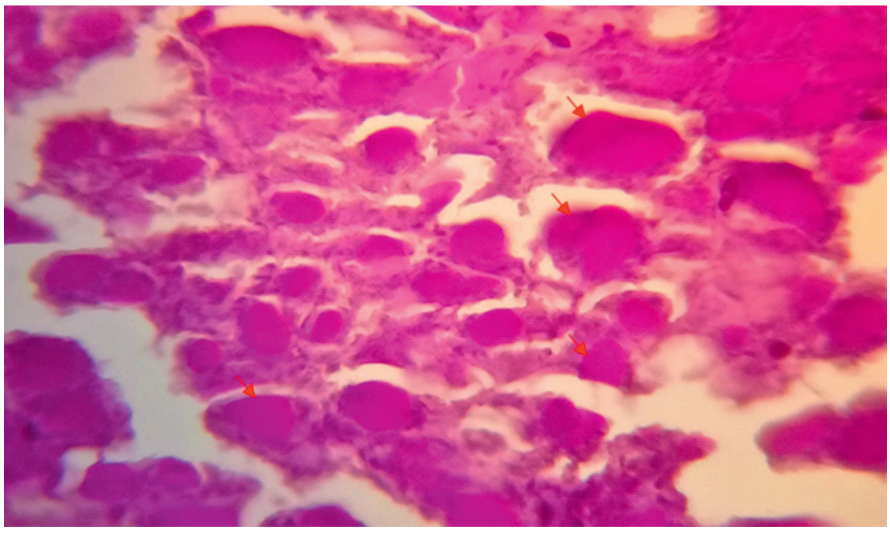

Figure 3.
2nd day of acute hypoxia model. Pathohystological structure of the thyroid gland. Stain: PAS x40

face of the gland. The cytoplasm of the adrenocytes of the medullary substance is pale stained in comparison to the cytoplasm of the cortical substance. The medullary substance is made up of chromaffin, gangliose and supporting cells that have a round or oval shape. Chromaffin cells are the main cells of the medullary substance, which are in the form of «throws» and «nests», have a large polygonal shape nuclei, fine-grained and vacuolated cytoplasm. Ganglious cells are few in number and are vegetative in nature; supporting cells have processes, there are glial in origin and contain chromaffin cells.

In the microscopic examination, the interzonal, intracellular connective tissue of the adrenal gland is poorly noticeable, and capillaries covering the adrenocytes from all sides and forming thick plexuses are clearly visible. The lumen of the capillaries in the medulla is wide, compared with the cortical substance.

2 days after the creation of the hypoxia model, the morphological and morphometric changes associated with hypoxia in histological preparations made from the thyroid and adrenal glands of animals are noted. However, compared with the control group, attention is drawn to the presence of weak dystrophic and destructive changes in both gland cells. In the morphometric indicators obtained as a result of the study, a significant difference is also not noted in the control group, only the relative weight of the glands is increased.

The thyroid and adrenal glands of the experimental animals included in this group have grown in volume, in particular, the consistency of the adrenal gland has softened, the color is pale. The capsule of the glands, which has a large number of collagen fibers, is slightly loosened, its lobules are relatively swollen, the border between the central and peripheral parts of the thyroid gland is clearly visible with the naked eye, and the cortex and medullary substances of the adrenal gland are poorly distinguished from each other.

Microscopically, no noticeable changes in the structure of the thyroid gland – parenchyma – follicles, epithelial cells of thyrocytes are observed; weak dystrophy of stroma – interlobular and interfollicular connective tissue, as well as endothelial cells covering the vessels is noted. Follicles as a whole have not changed their structure properties and normal staining properties. Prismatic epithelial cells of the follicles of the central part of the gland are poorly swollen, but do not lose their shape. In this case, the intrafollicular colloid has a liquid and foamy consistensy and is pierced by numerous resorption vacuoles, the cytoplasm of the cell is foamy, slightly edematous, the nuclei appear in the direction of the edge of the cytoplasm. The colloid, which fills the nucleus of the cell and the cavity of the follicle, is poorly stained. Morphometric parameters of follicles are relatively changed (fig.3).

In the peripheral parts of the thyroid gland, the follicles have a large size, flattened epithelium, thickening and stagnation of the colloid, a significant increase in the diameter and volume of the follicles is noted. As a result of this, the thyrocytes of the follicles get a flattened shape. Unlike the control group, by the action of hypoxia colloid is partially replaced with edema fluid in the cavity of peripheral follicles and pronounced PAS-positive reaction noticeable. The nuclei of thyrocytes are stretched parallel to the surface of the follicle and poorly stained with hematoxylin-eosin. In the cytoplasm of both the central and peripheral part of the gland, the individual micro-sized fat drops are clearly visible.

On the second day of hypoxia, the interlobular connective tissue of the thyroid gland is poorly visible. Cellular elements of connective tissue – fibroblasts, reticular cells, collagen fibers, mainly fibrous structures of the interstitial substance, are slightly swollen, but have not been targeted. Endo-

thelial cells that cover the walls of the capillaries are bulging towards the lumen, edema does not cover wide areas, although weak interstitial edema is detected in the vascular areas. However, dystrophic changes in glandular stroma relative to parenchyma are noticeable. The microcirculatory bed of the thyroid gland attracts attention with its weak plethora. In connection with an increase in vascular permeability, signs of plasmorrhagia, focal diapedesis bleeding are detected.

Microscopically, the boundaries of the cortical substance of the adrenal gland appear weak, the capsule becomes loose, loosening and irregular placements of the trabeclae arising from the capsule into the cortical substance are noticeable. The sinusoid capillaries of the cortical substance are enlarged and filled with blood. The size of the fascicular zone has decreased, and the glomerular and reticular zones have not changed. The cells of the glomerular zone are of small size, have a «matte» cytoplasm and a hyperchrome nucleus.

In the fascicular zone, in particular, in its outer layer (subzonal part), foam «transparent» cytoplasm cells, in the area close to the reticular zone (inner subzonal part), «matte» cytoplasm cells predominate. The «matte» cytoplasm and the small sizes nuclei of of the reticular zone appear weak. The nucleus of the adrenocorticocytes of the cortical substance is located not in the center of the cytoplasm, but slightly outside and poorly stained with hematoxylin-eosin. An unequal amount of lipid drops are noted in the cytoplasm of the fascisular and reticular zones.

The connective tissue covering separating the medulla from the cortical substance, is poorly visible. Under the influence of hypoxia, weak pronounced dystrophic changes in the cells of the medllary substance are noted. Thus, the «slots» of chromaffin cells, the main component of the brain substance, and the «protrusions» of the supporting cells surrounding them, were poorly deformed, the size of the ganglious cells are decreased. The cytoplasm of cells is lightly edematos, foamy, the nuclei are dark, the nucleoli are not noticeable, but the cell membrane is clearly visible. In some adrenocytes, nuclei with karyopyknosis are found. In the cytoplasm of the adrenocyte of the medullary substance, fat droplets are not detected, the cytoplasm is weakly stained with PAS. Histochemically, glycogen in the adrenocorticocytes of the adrenal gland is not detected with the PAS reaction.

In the intracellular connective tissue of the adrenal gland, poor edema, areas of loosening, signs of mucoid swelling are noticeable. Fibrous structures of connective tissue, especially collagen fibers, are relatively swollen but don't disintegrate. The noticeable edema in the interstitial area leads to the interstitial separation of tissue elements, tropocollagen microfibrils, resulting in the manifestation of the phenomenon of metachromasia in tissues stained with hematoxylin-eosin. The reticular fibers of the capsule trabeculae are poorly defined, and the plexus formed around the cells of the parenchyma of the gland is not noticeable.

Thickening of the walls and deformation of some parts of capillaries of the microcirculatory bed of the gland, weak dystrophic changes such as the fullness of veins, interstitial edema in paravasal areas can be seen with a light microscope. In histological preparations, endothelial cells of large vessels were swollen and subjected to desquamation, and their walls became brittle. In the microscopic examination, the walls of the capillaries of the medullary substance are edematous, dispersed, poorly stained with PAS. The effect of hypoxia is a consequence of increased vascular permeability, dilatation of the capillaries – in particular, with the formation of signs of focal plasmorrhagia in the fascicular zone and perivascular areas of the medullary cubstance. Hemolysis of erythrocytes is observed in the detachment zone. The cells of the detachment zone, based on the hemorrhages, are clear and the capillaries are emptied. As a result of the influence of hypoxia, vascular permeability increases, the lumen of capillaries dilates – in particular, signs of focal plasmorrhagia appear in the perivascular areas of the fascicular zone and medullary substance (fig.4).

Microscopically, there is an increase in the relative weight of the adrenal glands, although the boundaries of the cortex are clearly visible, the thickness has decreased significantly, especially in the fascicular zone. The thickness of the reticular zone of the cortex and medulla does not differ from that of the control group.

On the 5th day of the acute hypoxia model, acute pathomorphological changes, diffuse edema, and disruption of tissue metabolism are noted in the histotopography of the thyroid and adrenal gland cells due to a decreased oxygen supply of tissues. As a result of hypoxia, most tissues, cells, organelles of the glands completely lost their structural properties, suffered acute dystrophy and destruction. Also in the statistical indicators of the experiment, sharp changes characteristic of the effect of hypoxia are noted. It appears that these pathological changes are deeper in the adrenal gland cells than in the thyroid gland.

In the macroscopic examination, the color of the thyroid and adrenal glands were dimmed, the volume of which grew sharply, and the consistency became soft and brittle. The growth of its volume led to the release of connective tissue fibers, roughening and deformation of the capsule cover-

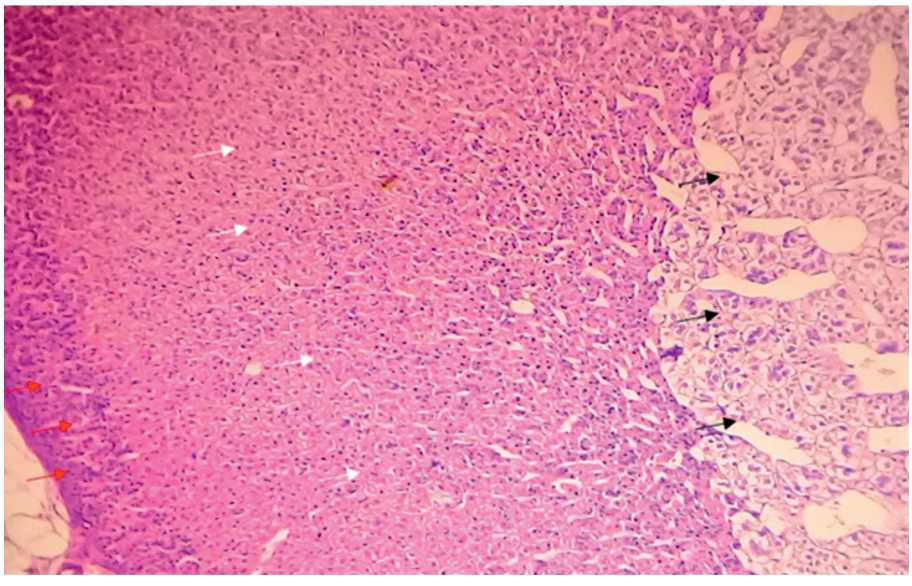

Figure 4.
2nd day of acute hypoxia model. Pathohystological structure of the adrenal gland. Stain: Hematoxilin-eosin: x10

ing the gland. Microscopically, the appearance of grayish-pink in the transverse section of the gland tissue reflects its damage. The release of connective tissue fibers is manifested by the breakdown of trabeculae directed from the capsule into the gland. The central and peripheral parts of the thyroid gland parenchyma and the adrenal gland parenchyma are visually swollen.

Microscopically, the border between the central and peripheral parts of the thyroid gland is not observed, the follicles are sharply hypertrophied. Among the follicles, noticeable signs of interstitial edema are noted, which leads to their separation and it is clear that there are no connections between them. It is observed that the follicles of the central part of the gland swell and pass from the prismatic form into the round. In the cavity of some follicles of the central part of the gland, ruptured thyrocytes, resorbed vacuoles in a colloid are found along the periphery of the follicles. Desquamated thyrocytes are absorbed by colloid.

The structure of the follicles located outside the center differs from the follicles located in the central part, the epithelial cells that cover the thyrocytes are acute edematous, colloid fills the lumen of the follicle. Along the peripheral part of the gland, an increase in the size of the follicles is noted; large-sized follicles are more common. In the cytoplasm, there is a complete breakdown of chromatin in some cells, as well as nuclei with karyopyknosis and karyolysis.

Noticeable destruction of the epithelial cells of the thyrocytes, loss of the nucleus in the group of many cells, pallorization of some cells is detected. The nuclei are observed on one edge of the cell, in a swollen, pale, flattened form. The absence of glycogen grains in the cytoplasm is associated with the use of glycogen by thyrocytes since the restoration of intracellular energy sources is more often compensated by glycogen (fig.5).

In the biopsy materials taken from the thyroid gland, the fibrous carcass of the stroma is deeply deformed, has lost its normal staining properties, acute alterations, signs of total edema in the interstitial area are detected. Thus, the fibrous structures of the connective tissue are swollen, fragmented, especially collagen fibers are fragmented and dispersed, in some places they are destroyed. Fibroblastic structures are not detected. Most cells are on the verge of necrobiosis. In the microscopic examination, small-sized foci of inflammation consisting of lymphocytic leukocyte elements appear in the peripheral parts of the stroma of the gland.

In the histological preparations, acute destructive changes in the microcirculatory bed, productive endovasculitis of the vascular walls, non-selection of contours, plethored capillaries and loss of the border of pericapillary cells in some areas are also observed. Acute plasmorrhagia, localized diapedez hemorrhages are noticeable. As a result of the increase in vascular permeability, the lumen of the vessels was emptied and the walls became brittle, some parts lost their continuity. A weak PAS reaction mainly in peripheral capillaries is noted.

In the case of microscopic examination of experimental animals with a hypoxia model, the boundaries between the cortex and medullar substances of the adrenal gland, as well as the zones of the cortical substance, the cytoplasm and nuclei of the adrenocytes are not noticeable, acute hypertrophy of the cells is noted. Histotopographically as a result of hypoxia, the inability to visualization the border of cells of the parenchyma of the gland is explained by acute edema of the tissues. In this connection, deep dystrophic and destructive changes in the cells of the gland are noted. Thus, «glomeruli»

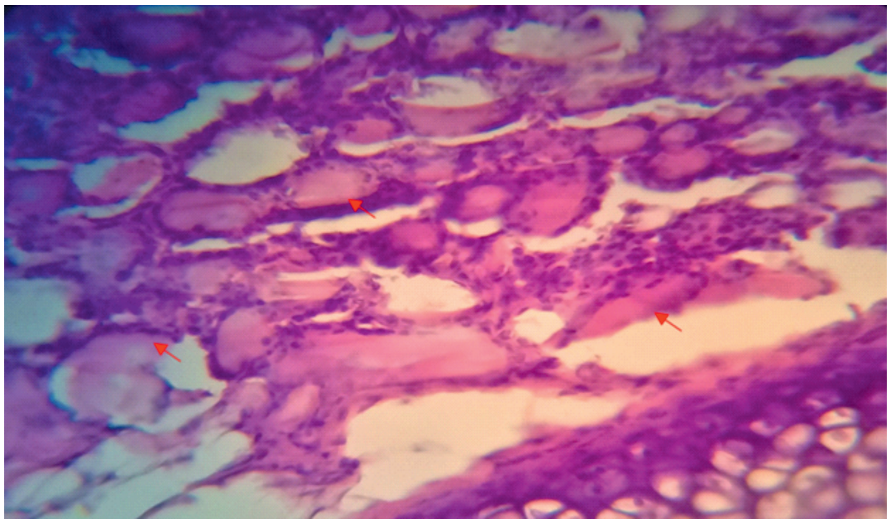

Figure 5. 5th day of acute hypoxia model. Pathohystological structure of the thyroid gland. Stain: Hematoxilin-eosin:x20

of the glomerular zone, «fascicles» of the fascicular zone, «epithelial trabecuale» of the reticular zone, chromaffin, ganglious and supporting cells of the medullar substance have lost their forms, subjected to acute structural changes. Therefore, in the histological preparations, there is no border between the zones, it is impossible to distinguish them from each other. In the fascicular zone, «light» and «dark» cells change each other, and in the glomerular and reticular zones, «dark» cells prevail. The radial direction of spongiocytes was violated, the size of their cells connection with perivascular edema was repeatedly increased, and the size of adrenocytes of glomerular and reticular zones was relatively increased.

Adrenocorticocytes of the adrenal cortex are large, cytoplasm eosinophilic, acute edematous and pale. The nucleus of the cells of the cortical substance is located adjacent to the wall of the cytoplasm and is not stained with hematoxylin-eosin. An unequal amount of microvesicular (small granular) lipid droplets are noted in the cytoplasm of the fascicular and reticular zones (fig.6).

The connective tissue covering between the fascicular zone and the medullary substance is disintegrating, and the boundary is not visible. There are also acute dystrophic changes in the cells of the medullary substance of the gland, noticeable signs of interstitial edema between the cells are observed, cytoplasm is acute edematous, the nucleus is sharply shrunk and is visualized in the periphery of the cytoplasm. The detection of a large amount of fat droplets in the cytoplasm is explained by denaturation of cytoplasmic proteins. Due to hypoxia, the complete dystrophy of some cells of the medullary substance causes the destruction of the nucleus, the development of necrobiosis and necrotic processes.

Some fibers of the fibrous components of the connective tissue of the adrenal gland are swollen, in particular, the fibrous structure of the brain substance is brittle and subjected to mucoid swelling. The reticular fibers, which go from the connective tissue trabecules to the depth of the organ, are noticeable due to interstitial edema. In some areas of stroma, signs of destruction – mucoid swelling and edema not only in fibrous structures but also in main substance are observed.

Microangioarchitectonics of the cortical substance of the gland is characterized by an unequal plethora of different zones of the organ. Thus, when the plethora of the glomerular zone is weak and the capillaries are empty compared to the 2nd day of the experiment, sinusoid capillaries in the fascicular and reticular zones are sharply enlarged, blood-filled, and as a result, the distance between the cell columns is reduced. Enhanced plethora of the medulla and signs of plasmorrhagia are more pronounced. Sinusoid capillaries are maximally enlarged, their walls are conjoined, the walls of some capillaries are scattered, and the points of diapedesis in the stroma are visualized. In the histological samples, a vascular network formed by various loops appears between the medullary substance and the reticular zone.

Microscopically, the thickness of the adrenal cortex is less than the control group, and the adrenal medulla is closer to the control group. Unlike the first day of the experiment, the glomerular and reticular zones are thicker. Interstitial edema leads to the incompletion of cells.

In conclusion, it should be noted that powerful short-term hypoxic stimulants, besides the visible changes in the cells of the thyroid and adrenal glands, also change their histofunctional state. These dystrophic changes are more pronounced in the adrenal gland than in the thyroid gland. This is explained because the adrenal gland is more sensitive to stress factors, and the thyroid gland is earlier to hypoxia. Thus, the effect of hypoxia leads to an earlier change in the interaction of glomerular, fascicular and

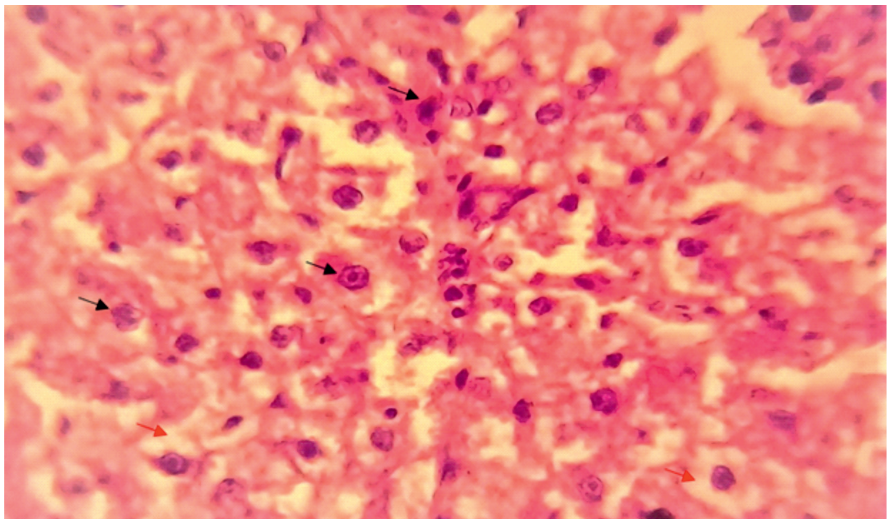

Figure 6.
5th day of acute hypoxia model. Pathohystological structure of the adrenal gland. Stain: Hematoxilin-eosin: x20

reticular zones of the cortical substance of the adrenal gland, an increase in the activity of the medullary substance. Increased adrenocortical activity during the initial effect of hypoxia provokes the synthesis and secretion of adrenocorticotropic hormone.

Thus, the early stage of hypoxia is characterized by the appearance of acute pathomorphological changes in the cells of both glands. Under the influence of acute hypoxia, in the central part of the thyroid gland there are separate prismatic follicles, liquid colloid, and in the periphery there are flattened follicles, condensed colloid, and in the adrenal gland there is an earlier change in the interaction of the glomerular, fascicular and reticular zones, increased activity of the medullary substance. Despite the fact that there is a dilatation of the capillaries in both glands, focal diapedesis, parenchymatous and interstitial edema, accompanied by an increase in the density of the vessels, edema in the thyroid gland does not cover large areas and is of focal nature. This is explained by more damage to the cells of the adrenal gland.

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МРНТИ 76.29.35

THE PULMONARY BULLOUS EMPHYSEMA. LITERATURE REVIEW

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Keywords
bullous emphysema, pulmonary bullae, surgical reduction of lung volume, valvular bronchoblocation

Abstract

At the moment, there are some differences in the papers about bullous emphysema of the lungs. Some authors prefer to divide this pathology into a separate nosological unit, despite the general tendency to consider bullous emphysema as one of the manifestations of emphysema of the lungs, which in most cases is a clinical form of COPD. But the dilemma, in turn, does not solve the problem of qualitative diagnosis and treatment of this pathology. Currently, there is a small amount of works related to epidemiology and structured data on the treatment of bullous emphysema, although this pathology has a significant influence on the population. This review article will show the etiology, pathogenesis, classification, signs and symptoms, diagnosis and treatment of bullous emphysema.

Өкпенің буллезді эмфиземасы. Әдебиет шолуы

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Н.В. Выровщиков, Н.В. Бокова, Ш.И. Ахмедов, К.Е. Гвоздырев, О.Р. Джафаров, С.М. Джусупов *– «хирургия» мамандығы бойынша 7 курс интерндері, ҚРМУ хирургия кафедрасы анестезиология және реаниматология курсымен бірге.*

Түйін сөздер
өкпенің буллезді эмфиземасы, өкпе бұлласылары, өкпе мөлшерінің хирургиялық редукциясы, клапанды бронхоблокация.

Аңдатпа

Қазіргі уақытта өкпенің буллезді эмфиземасына арналған жұмыстарда кейбір айырмашылықтар бар. Көп жағдайда СОӨА клиникалық нысаны болып табылатын буллезді эмфиземаны өкпе эмфиземасы көріністерінің бірі ретінде қарастырудың жалпы үрдісіне қарамастан, кейбір авторлар бұл патологияны жеке нозологиялық бірлікке бөлуді жақтайды. Бірақ бұл мәселе, өз кезегінде, осы патологияны сапалы диагностикалау мен емдеу мәселесін шешпейді. Қазіргі уақытта эпидемиология мәселелеріне және буллезді эмфиземаны емдеу туралы құрылымдалған деректерге арналған аздаған еңбектер бар, дегенмен, кейбір деректер бойынша, бұл патология популяция арасында үлкен маңызға ие. Бұл шолу мақалада өкпенің буллезді эмфиземасының этиологиясы, патогенезі, жіктелуі, клиникалық көрінісі, диагностикасы және емдеу мәселелері қарастырылады.

Буллёзная эмфизема лёгких. Обзор литературы

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Ключевые слова
буллёзная эмфизема лёгких, лёгочные буллы, хирургическая редукция объёма легких, клапанная бронхоблокация

Аннотация

На текущий момент имеется некоторые различия в работах, посвященных буллёзной эмфиземе лёгких. Некоторые авторы предпочитают выделять данную патологию в отдельную нозологическую единицу, несмотря на общую тенденцию рассматривать буллёзную эмфизему как одно из проявлений эмфиземы лёгких, в большинстве случаев являющейся клинической формой ХОБЛ. Но этот вопрос, в свою очередь, не решает проблему качественной диагностики и лечения данной патологии. На текущий момент имеется малое количество работ, посвященных вопросам эпидемиологии и структурированных данных о лечении буллёзной эмфиземы, хотя по некоторым данным, эта патология несёт весомое значение среди популяции. В этой обзорной статье будут рассмотрены вопросы этиологии, патогенеза, классификации, клинической картины, диагностики и лечения буллёзной эмфиземы лёгких.

Introduction

Bullous emphysema (BE) of the lungs is considered as a variant of emphysema of the lungs and is characterized by the destruction, stretching or merging of the air spaces of the adjacent alveolar walls with the formation of cavities larger than 1 cm which are called bullae. The bullae is a limited thin-walled space consisting of compressed or stretched sections of the lung parenchyma, filled with air [1,2]. Bullae can have primary and secondary origin and occur in the context of different clinical situations, which implies some differences in terminology: bullous lung disease (BBL) is characterized by the formation and development of bullae in a relatively normal, unchanged pulmonary parenchyma of one or both lungs; BE is a consequence of chronic obstructive pulmonary disease (COPD) in which bullae are formed as a result of existing pathological emphysematous changes; in the vanishing lung syndrome normal parenchymal lung tissue is gradually replaced by numerous bullae, which is reflected in the dynamics of x-ray images; bullae can also occur in the last stages of pulmonary fibrosis in sarcoidosis or complicated pneumoconiosis [3,4,5].

There is may be a confusion between the definitions of BBL and BE, as some pathologists believe that BBL is a consequence of panacinar emphysema. This statement is not valid, since panacinar emphysema most often develops in the lower lobes, while BBL tends to develop in the upper lobes and there are a sufficient number of differences in pathophysiology between these diseases [6,7].

There are some differences between such pathomorphological structures as bull, bleb and cyst. The bleb is an accumulation of air between two layers of the visceral pleura – the outer and inner elastic layer, its origin is associated with pathological penetration of air from the pulmonary parenchyma into the visceral pleura [9]. Cysts are the cavities lined with a layer of epithelial cells; the x-ray picture of lung cysts can be very similar to bulls [10].

There is not so much epidemiological data on BE in the literature, but in general, it is known that EB affects more than 5% of the world population with a total prevalence of about 12% in adults over 30 years old in the structure of lung pathology. In the United States, BE is on the 3rd place among diseases that lead to death, and kills more than 120,000 people per year [4].

Etiology

At the moment, there is no well-defined etiological factor leading to the development of bullae in lung tissue. According to available data, pulmonary bullae are formed in the presence of the following factors and diseases:

- Bullae development is associated with long-term smoking. The formation of giant bullae is associated with marijuana smoking in some patients, but this claim can be challenged due to the simultaneous smoking of tobacco by the same patients [11].
- Cases of association of BBL with HIV infection are described, including in patients with drug dependence, in particular those who use injections of methadone, methylphenidate or talc-containing substances. When injected with methylphenidate, a similar pattern of pulmonary bullae development is observed, as in patients with alpha-1 antitrypsin deficiency [12,13].
- Bullae also develop in patients with such diseases as: Marfan syndrome, Ehlers-Danlos syndrome type IV, Aspergen syndrome, alpha-1 antitrypsin deficiency, 1-antichymotrypsin deficiency, polyangiitis with granulomatosis, sarcoidosis [14,15].
- Also, bullae can be formed as a result of emphysema of distal acinuses, as a result of long-term chronic inflammation and destructive changes in the terminal bronchioles and respiratory bronchioles of the first order; cases of bullae formation associated with inhalation of crushed glass fiber are also described [6,15].

Classification

The following will be a classification based on the etiology and pathomorphological characteristics of bullae:

Anatomically pulmonary bullae are classified into three main types:

- Type I bullae are characterized by the presence of a narrow neck that connects the bullae

Primary bullae	Secondary bullae	Lung fibrosis	Hereditary disorders
Vanising lung syndrome; Single giant bullae; Bullous lung disease.	Emphysema: - paraseptal; - panacinar (panlobular); - centriacinar (centrilobular).	Sarcoidosis; Idiopathic pulmonary fibrosis; Conglomerate silicosis; Cirrhotictuberculosis.	Alpha-1 antitrypsin deficiency; Ehlers-Danlos Syndrome; Salla disease; Marfan syndrome; Fabry Disease; Cutis laxa.

Table 1.
Classification of pulmonary bulls [6,8].

with the pulmonary parenchyma. This type of bullae can be caused by volumetric hyperventilation or formed at the site of damaged lung tissue. The walls of type I bullae are thin, the contents are represented by an empty cavity. Type I bullae are usually located in the area of the apices of the lungs and along the edge of the tongue of the lower lobe of the left lung, as well as in the area of the middle lobe. They very often develop in association with paraseptal emphysema [6,17].

– Type II bullae develop from subpleural parenchyma and are characterized by the presence of a neck of panacinar emphysematous pulmonary tissue. The bullae cavity can be filled with emphysematous pulmonary tissue, in which small vessels are preserved. Unlike type I bullae, the wall of these bullae is formed by a pleura covered with intact mesothelial cells. Type II bullae most often develop in the area of the upper lobes, the anterior surface of the middle lobes and in the area of the lower lobes of the lungs adjacent to the diaphragm [8,17].
– Type III bullae consists of a moderately hyperventilated portion of the lung connected to the rest of the lung by a broad base extending deep into the parenchyma. It is believed that this type of bullae occurs in atrophic forms of pulmonary emphysema [6,18].

A group of Russian researchers proposed to subdivide bullous emphysema into homogeneous (total lesion of lung tissue) and heterogeneous (with predominant lesion of segments) forms. It is also proposed to introduce a combined form of emphysema of the lungs, which implies the presence of signs of both bullous and diffuse (without bull) emphysema [19].

Pathogenesis

According to the present knowledge, given the lackof specific studies on BE, the genes involved in the molecular pathogenesis of BE are probably the same involved in the development of DE. However, a 4-bpdeletion in the Birt-Hogg-Dube gene (FLCN) hasrecently been shown to be strongly associated with dominantly inherited spontaneous pneumothorax, “(SP)”the main complication of subpleural bullae, in a large Finnish pedigree with a tendency to SP development [20].

A number of case reports showed an association between BE and lung cancer arising from scarred and contracted areas close to a bulla wall, although specific studies at the molecular level have not been conducted on this field [21].

Bullae develop after retraction and collapse of surrounding lung away from a region of weakness. The mean pressure inside the lung bullae is negative and shows a constant parallelism with the pleural pressure. The atelectasis of the surrounding areas, observed at times, is due to the elastic retraction of normal parenchyma and not to the compression by the bulla [21,22].BE complicating DE substantially contributes to the functional deterioration and causes significant con-founding effects on the functional assessment [20]. These findings can be explained by the contribution of lung bullae to the airways obstruction, because of their complete loss of elastic recoil. The static elastic recoil pressure of the emphysematous lung is, the refore, further decreased. Even if the bullae remain in free communication with the airway, they do not significantly participate in the ventilation [22]. Further more, the chest wall mechanics is altered because of the lossof linkage with the nonbullous lung tissue, leading toincreased chest wall work and worsening of hyperin-flationand sensation of dyspnea [21,23]. The confound ingfunctional effect of bullae depends on BE extent: relatively milder obstruction can be observed with severe BE, whereas moderate BE causes modest deterioration of diffusing capacity [23].

Clinical manifestations

One of the main clinical symptoms of bullous emphysema is shortness of breath, which can be asymptomatic in many patients [24]. The development of shortness of breath may be an indication for surgery, since Bulls can occupy more than 30% of hemithorax. Shortness of breath at the onset of the disease appears only with significant physical exertion, and initially patients often do not notice it. Shortness of breath in patients with bullous emphysema is dangerous because it does not manifest for many years, and progressing, it turns into a condition that threatens the patient’s life. Tolerance to physical activity is reduced, since even at rest, the compensatory capabilities are at the limit [26]. Dyspnea usually has an expiratory character. Patients have a short, “sharp”, “grabbing” breath and an elongated, sometimes step-shaped exhalation. They exhale with closed lips, puffing out their cheeks (“puffing”). At the same time, pressure in the bronchial tree rises, which reduces the expiratory collapse of small non-cartilaginous bronchi (due to a violation of the elastic properties of the lung tissue and an increase in intrathoracic pressure) and contributes to an increase in ventilation. As the disease progresses, patients may complain of shortness of breath with tension or wheezing [24,25].Patients often have a productive (wet) cough, often called a “smoker’s cough,” more pronounced usually in the morning. Cough is not a specific complaint of patients with bullous emphysema and is most often due to the presence of chronic bronchitis [25].

Body weight with bullous emphysema is reduced, which is associated with the intense work of the respiratory muscles, aimed at overcoming the high resistance of the terminal airway. Patients with pulmonary emphysema in the initial stages of the disease take a forced position on the abdomen with their head down and shoulder girdle, which brings them relief. In this situation, the patient achieves an increase in abdominal pressure, lifting up the diaphragm and improving its function. However, in severe emphysema with marked changes in the chest and fatigue of the respiratory muscles, a horizontal position causes intense diaphragm work, so patients are even forced to sleep in a sitting position [25,26]. Patients with bullous emphysema often occupy a sitting position with their torso slightly inclined forward, resting their hands on their knees or the edge of the bed, which allows the shoulder girdle to be fixed and additional muscle to be included in the act of breathing [27].

The color of the skin with emphysema is more pink than cyanotic. Slight cyanosis is caused by prolonged preservation of the gas composition of the blood, only in advanced cases does cyanosis appear, which is caused by the development of hypercapnia [25,27]. There is a swelling of the cervical veins during exhalation due to increased intrathoracic pressure.

Examination of the chest reveals a barrel-shaped chest. Kyphosis is sometimes observed. With percussion, a boxed sound is determined. There is an increase in the standing height of the tops, a shift of the lower borders of the lungs down and a sharp restriction of the mobility of the lower pulmonary margin. A decrease in cardiac and hepatic dullness due to increased airiness and an increase in lung tissue volume is characteristic [26,27]. Often there is a loud heart sound P2, which is a sign of pulmonary hypertension. In addition, a symptom of "drumsticks" may be present. Edema can be a sign of a decrease in the power of the right ventricle and pulmonary artery [24].

Diagnostics

Methods of laboratory diagnostics include studies of hematocrit, ABGs and PFTs, alpha-1-antitrypsin. Methods of instrumental diagnosis of bullous emphysema are based on chest radiography in two projections, CT, spirometry.

Radiography. In the early stages of the pathological process, emphysema cannot be detected by traditional radiographic examination. Direct radiographic signs of emphysema are:

- thin-walled air cavities (usually large);
- extensive areas of pulmonary fields devoid of pulmonary pattern, usually in combination with the displacement or rupture of visible pulmonary vessels [28,29].

Both these signs characterize bullous emphysema, in which large air cavities occur in the lung tissue. Intra-lobular emphysema can be detected by radiation examination only with the help of CT [30].

CT. Emphysema is a constant pathological increase in the air-containing spaces distal to the terminal bronchioles, accompanied by the destruction of their walls, in the absence of obvious fibrosis. Emphysema is usually classified into three main types depending on the predominant localization of destruction zones: centrilobular, panlobular and paraseptal. In the early stages of development, these forms of emphysema can be confidently distinguished in IPT. In the final stage of the disease, it is difficult or impossible to distinguish them not only with CT, but also with morphological examination.

Centrilobular emphysema is one of the most common forms and is usually the result of Smoking. It mainly affects the terminal bronchioles located in the Central part of the secondary pulmonary lobule. The changes are most pronounced in the upper lobes of the lungs, especially in the apical and posterior segments. In IPT and morphological examination, centrilobular emphysema in the initial stage of development is characterized by the presence of zones of reduced density, having a rounded shape and small dimensions (usually 2-5 mm). The walls of such air cavities are actually lung tissue [30,31].

Panlobular emphysema in typical cases is associated with a deficiency of A1-antitrypsin, but can also be observed in smokers, the elderly, with obliterating bronchiolitis. Panlobular emphysema is characterized by uniform destruction of pulmonary tissue within the secondary pulmonary lobule. The walls of such air cavities become invariable connective tissue partitions between the lobules. In areas of reduced density, preserved vessels may be visible. The most pronounced changes are usually observed in the lower lobes of the lungs. A common process leads to the formation of large areas of reduced density without visible walls and impoverishment of the vascular pattern. Bullae and cysts are usually absent.Such changes can be difficult to recognize in CT [30,32].

Paraseptal emphysema is characterized by involvement in the pathological process of the distal part of the secondary pulmonary lobule. Air cavities most often have subpleural localization. This form may be independent or be detected in combination with centrilobular emphysema. As a rule, paraseptal emphysema is asymptomatic functionally, but may be accompanied by the development of spontaneous pneumothorax. Often in such patients, bulls of different sizes and shapes are detected. Bull is

defined as an air cavity with clear smooth thin walls with a diameter of more than 1 cm. Most bull subpleural localization regardless of size are manifestations of paraseptal emphysema.

Bullous emphysema is not an independent morphological concept, although it usually develops in connection with centrilobular and para-septal emphysema. Nevertheless, multiple large and giant bulls with visible walls are often described as a separate clinicorentgenological syndrome - "disappearing lung syndrome", "primary bullous lung disease", etc. Giant, increasing in size bulls can be found not only in middle-aged and elderly smokers with long Smoking experience, but also in relatively young people. VRCT is much better than radiography, reflects the prevalence of bullous changes and manifestations of paraseptal and centrilobular emphysema [31,32]

Spirometry. Pulmonary function testing is the standard for assessing emphysema. The ratio of forced expiratory volume per second (FEV1)/forced vital capacity (FVC) is usually <0.7. FVC decreases due to loss of elastic recoil of the lung. There will be an increase in total lung capacity, residual volume and functional residual capacity, as well as a decrease in vital capacity. A decrease in lung diffusion capacity for carbon monoxide (DLCO) will often be present due to the destruction of the lung interstitium. The basis of diagnostic search in patients with pulmonary bullous emphysema is radiation and endoscopic methods. Thoracoscopy should be a mandatory study in patients with BEL complicated by pneumothorax [28,31].

From laboratory methods, it may be useful to test for alpha-1-antitrypsin deficiency. A complete blood test may show increased hemoglobin and hematocrit due to reactive erythrocytosis from chronic hypoxia. Blood chemistry panels will show increased serum bicarbonate because of metabolic compensation for respiratory acidosis [29,33].

Treatment options, complications and prognosis

– *Conservative treatment*

To treat such a terrible manifestation of COPD, conservative treatment is used in a combination of drugs: beta-2 adrenomimetics with glucocorticosteroids in the form of inhalations. For example:

Table 2.
Pharmacotherapy of the clinical manifestations of bullous emphysema. This combination allows to reduce the pace of development of RB in patients with COPD [38].

Formoterol/Budesonide (B)	4.5/160, 9/320
Salmeterol/Fluticasone (B)	50/100, 250, 500
Vilanterol/Fluticasone furoate (B)	25/100 (22/92)

– *Surgical treatment*

For a long time until the middle of the last century there were no methods of surgical treatment of this pathology. Surgery was first proposed by O. Brantigan and E. Mueller in the mid-50s. At that time, the experience of specialists in performing operations in the chest cavity and anesthetic management of patients was insufficient, as a result, every fifth patient with resection of 20-30% of the lung. died. For a long time there was no significant progress in this direction, until in 1995 J. D. Cooper published a methodology and low mortality of patients in the postoperative period [37,41].

The essence of the lung volume reduction surgery (LVRS) is to remove the altered pulmonary parenchyma, which is dominated by the bulls, which compress the alveoli of the neighboring departments of the lung, as a result, gas exchange is reduced not only in the altered segment, but also in the healthy department. In addition to straightening the healthy parts of the lung, the load on the diaphragm decreases, the lung volume increases, which reduces the residual volumes and increases the volume of inhalation and exhalation [34,37,41].

At the moment, LVRS is performed both by open and thoracoscopic method. For open resection with bilateral lesions perform median sternotomy, and with unilateral-lateral thoracotomy [34,41].

Indications for LVRS [34,41]:

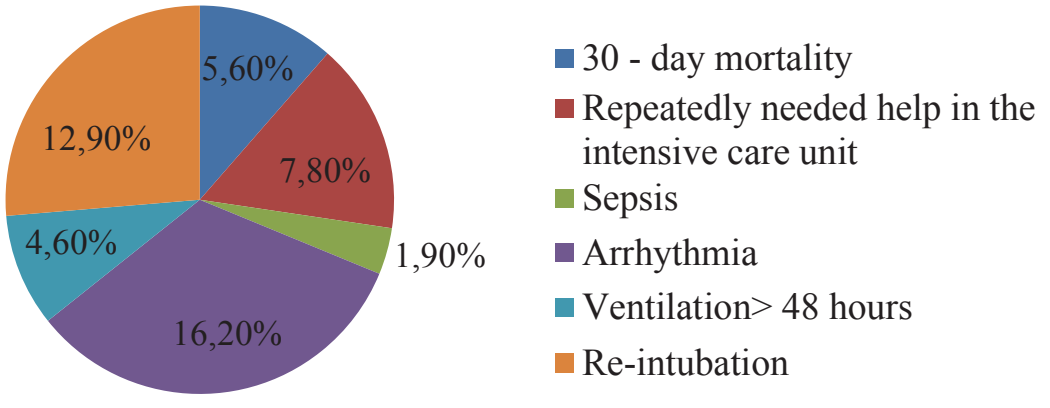
– FEV1 20-40% of the proper;
– residual volume >200% of due;
– total lung capacity >125% of due;
– upper lobe localization of emphysema;
– heterogeneity of emphysema (presence of unchanged lung tissue according to CT);
– the body mass index is 17-32 kg/m2;
– low physical endurance;
– motivation of the patient;
– patient's readiness for rehabilitation measures after surgery.

Contraindications to LVRS [34,41]:

– age >75 years;
– continuation of Smoking;
– bronchiectasis, acute respiratory infections;
– prior surgery on the chest;
– lower lobe localization of emphysema;
– "disappearing" lungs according to CT;

Results	The society of thoracic surgeons U.S.	National study on the treatment of emphysema USA
	%	%
30 - daymortality	5,6%	2,2%
Repeatedly needed help in the intensive care unit	7,8%	11,7%
Sepsis	1,9%	2,5%
Arrhythmia	16,2%	18,6%
Ventilation> 48 hours	4,6%	13,6%
Re-intubation	12,9%	21,8%

Table 3.
Complications from LVRS in the United States in 2014. [40]



Complications from LVRS. [40]

– partial voltage C02 in arterial blood (C02) >55 mmHg. st.;
– mean pulmonary artery pressure >35 mmHg. st.;
– left ventricular ejection fraction <40%;
– continuous intake of systemic corticosteroids (>10 mg/day of prednisone).

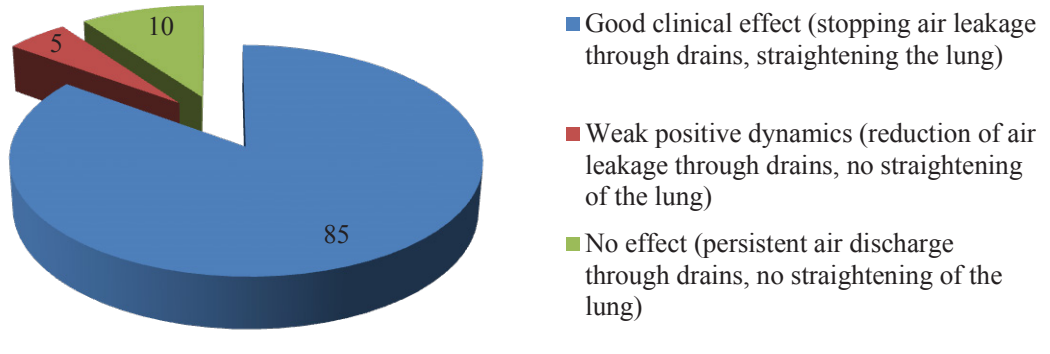
As surgical techniques over the past 20 years, many techniques have been put into practice, including steam ablation of the bull, the use of bronchoscopic valvular bronchoblocation. Steam ablation is currently not widely used [35].

A valve bronchomalacia carries more interest for the provision of care to patients. Due to the wide prevalence of COPD and sufficient diagnosis of bull in the initial stages, this method is of considerable interest [36].

This method is carried out even under local anesthesia of the oropharynx and nasal mucosa. To do this, pre - 30 minutes enter atropine 0.5 mg, in order to reduce the secretion of mucus by the bronchi. Then anesthetize the oropharynx with a Lidocaine solution of 2 ml, spraying it with a nebulizer. A valve is placed in the lumen of the bronchus. This valve works unidirectionally, does not let in air during inhalation, but releases air on exhalation. According to Lee E. G., the use of valves contributes to the disappearance of bulls up to 5 cm in size a month after the procedure, which was confirmed on CT of the chest cavity [39].

This technique is safer for patients, the duration of the procedure is small, the cost of the procedure, the preparation of the patient, the postoperative period is much lower than in LVR. The patient on the next day after x-ray control is able to go home [36,41].

The technique of valvular bronchoblocation is cheap compared to foreign analogues or the use of adhesive compositions to seal the lung tissue during surgery and does not require intravenous an-



Results of endobronchial valve installation. [41]

esthesia to install or remove the blocker. The phenomena of endobronchitis, arising from prolonged bronchial occlusion, are stopped independently without the formation of its stenosis [36,41].

The data obtained indicate the need for greater use of this method in patients with pulmonary bullous emphysema in the practice of thoracic departments [8].

Conclusion

Given the available epidemiological data, bullous emphysema is a relatively common lung pathology. It is determined that the development of bullae in the lung tissue, in general, can be associated with emphysema of the lungs (most often), as well as have a primary genesis. The pathophysiology of bullous emphysema is closely associated with chronic inflammation of the distal portions of the bronchial tree, as well as hereditary diseases associated with impaired synthesis of structural components of the lung parenchyma and some fermentopathy, which leads to destruction of walls of alveoli or change their properties, further contributing to the permanent increase in the size of the newly formed air spaces. Clinical manifestations of bullous emphysema are nonspecific, and are usually accompanied by respiratory failure syndrome and asthenic syndrome. The most specific of the instrumental methods of research is CT, however, the results of CT do not always make it possible to establish the exact type of bullous emphysema. Some variants of surgical treatment were described in the work. Each method is effective and justified depending on the severity of the pathological process. The technique of endobronchial bronchioblocation allows to achieve good results in the treatment of bull in the amount of two or three units, including large size. This method does not require anesthetic administration of the patient, does not require inpatient medical care, which significantly reduces the total cost of treatment of the patient. Endobronchial bronchomalacia may be provided in an outpatient setting with the appropriate equipment. With multiple lesions of the pulmonary tissue bulls, endobronchial bronchioblocation is noticeably difficult, in consequence of which it is recommended to carry out LVRS, preferably thoracoscopically. LVRS will relieve the pressure on the unchanged segments of the lung, which, in turn, will contribute to the straightening of these segments. This method will require inpatient care, high-quality anesthetic support during surgery, long-term postoperative management.

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МРНТИ 76.29.30

VARIANTS FOR THE DEVELOPMENT OF CEREBRAL ARTERIES: POSTERIOR TRIFURCATION OF THE INTERNAL CAROTID ARTERY

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Abstract

Vascular diseases of the brain are one of the main problems of modern medicine. The quantity and quality of neurosurgical operations is growing. We conducted a search for literature covering various variants of the structure of vascular blood supply to the brain; we were interested in trifurcation of the internal carotid artery, as the most common development option among non-classical ones. We tried to illuminate the frequency of occurrence of posterior ICA trifurcation, its functional significance, and the effect on the development of circulatory disturbances in the brain and vascular anomalies.

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Keywords
trifurcation, internal carotid artery, structural features of blood vessels, brain

Ми артериясының даму нұсқалары: ішкі ұйқы артериясының артқы трифурациясы

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Аңдатпа

Қазіргі медицинаның басты мәселелерінің бірі мидың қан тамырлары аурулары болып табылады. Нейрохирургиялық операциялардың саны мен сапасы өсуде. Біз миды тамырлы қанмен қамтамасыз ету құрылымының әр түрлі нұсқаларын қамтитын әдебиеттерді іздедік, классикалық емес түрлердің ішіндегі ең кең таралған нұсқасы ретінде ішкі каротид артериясының трифурациясы қызықтырды. Біз ИКА-ның артқы трифурациясының пайда болу жилігін, оның функционалды маңыздылығын және церебральды қанмен қамтамасыз етудің бұзылыстары мен тамырлы ауытқулардың дамуына әсер етуді атап көрсетуге тырыстық.

АВТОРЛАР ТУРАЛЫ

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Түйін сөздер
трифуркация, ішкі ұйқы артериясы, қан тамырларының құрылымық ерекшеліктері, ми

Варианты развития артерий головного мозга: задняя трифуркация внутренней сонной артерии

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Аннотация

Сосудистые заболевания головного мозга являются одной из основных проблем современной медицины. Растет количество и качество нейрохирургических операций. Мы провели поиск литературы, освещающей различные варианты строения сосудистого кровоснабжения головного мозга, нас заинтересовала трифуркация внутренней сонной артерии, как наиболее часто встречающийся вариант развития среди неклассических. Мы постарались осветить частоту встречаемости задней трифуркации ВСА, ее функциональное значение, влияние на развитие нарушений кровоснабжения мозга, сосудистых аномалий.

ОБ АВТОРАХ

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Ключевые слова
трифуркация, внутренняя сонная артерия, особенности строения сосудов, головной мозг

The problem of vascular diseases of the brain remains one of the leading in modern world medicine. There is an increase in neurosurgical operations performed, for which knowledge of morphometry and anatomical variability is important [1]. The influence of the structural variants of the arterial blood supply to the brain affects the state of hemodynamics in the brain. Unequal blood circulation in certain variants of the development of the arterial circle can lead to the formation of vascular aneurysms, with subsequent rupture, and the development of hemorrhagic stroke, or can to a decrease or full cessation of blood flow through the supply vessels with the development of ischemic stroke [2]. The arterial circle of the cerebrum is the most important, constantly acting anastomosis between the systems of the internal carotid arteries and the vertebrobasilar system. However, in the available literature there is no clear conception which variations of the Willis circle are normal variants that do not worsen the compensatory capabilities of collateral blood flow, and which predispose to a disturbance in the blood supply to the brain [3]. Anomalies in this network can lead to serious clinical conditions, such as stroke, leading to poor blood supply to the brain [4]. Congenital vascular abnormalities of the brain: aplasia or hypoplasia of blood vessels, vessels with atypical morphology, etc. are the result of an early defection of their development [5].

The anatomical anastomosis provided by the circle of Wellis is of great importance if one of the main vessels of the brain undergoes occlusion. It was found that in more than 50% of people with a healthy brain and 80% with dysfunctional changes, at least one artery is present in the arterial circle of the brain that is absent or underdeveloped [4,6]. “Non-classical” variants of the structure of the arterial circle of the cerebrum (Willis circle) are found according to various authors from 25 to 75% of cases [7]. F. Toda and D. Gacheshiladze give the following figures: 14-25% of cases, 7-16% [8]. In studies of J. Sidorov and his colleagues, 24% posterior ICA trifurcation was also more often detected, which conform to 13 patients [9]. According to A. Gorbunov as a result of his research of 322 people, the “classic variant” of the Willis circle was found in 51.6% and in 48.4% one of the non-classical variants, of which 5.1% revealed posterior trifurcation [10]. In his work, Gorbunov singled out partial posterior ICA trifurcation – equality in diameter of the posterior connecting artery and posterior cerebral artery on one side and full trifurcation – superiority in diameter of the PcCA over the proximal segment of the PCA on one side [10]. In opinion of scientists, such options show its full-fledged function, which plays a large role in the regulation of blood flow in the brain. Variants of the non-classical structure of the Willis circle can be the cause of aneurysms in both its anterior and posterior departments. [7]. The most dangerous are the anterior and posterior trifurcations of the carotid artery, which affect the spreading of blood in the brain, until the occurrence of occlusive changes requiring good collateral circulation. The internal carotid artery on the side of its posterior trifurcation delivers about 50% of the blood to the brain, the opposite internal carotid artery - 40% of the blood, and the basilar artery - only 10% of the blood [11,12]. In the posterior section, the most common are: posterior trifurcation of the internal carotid artery (the posterior cerebral artery departs from the internal carotid artery), aplasia of one or both posterior connecting arteries [13]. Kotsenko’s studies have shown that patients with ICA pathology, such as complete posterior trifurcation, PCA hypoplasia, s-shaped pathological tortuosity, and loop formation are more often observed cognitive impairment (12.6%), less often moderate (7%) and lungs (8.5%). With a combination of anomalies of the intracerebral arteries and precerebral arteries (complete posterior trifurcation of the left ICA and hypoplasia of the left PCA), moderate violations of intellectual-mnestic functions more often developed (33.3%)[14].

In the posterior trifurcation of the ICA, the anterior, middle, and posterior arteries of the brain depart from it, in this case, the posterior cerebral artery departs by means of the posterior connecting artery. The proximal segment of the posterior cerebral artery adjacent to the basilar artery (P1) is usually hypoplastic, however, in rare cases, the diameter of P1 is equal to the diameter of the enlarged posterior connecting artery. Unilateral posterior trifurcation of the internal carotid artery is detected in 22% of cases, and on the right - 2 times more often than on the left, bilateral - less often (4% of cases). Posterior trifurcation of both internal carotid arteries, in which there are large posterior connective arteries, is found to be normal in the first 4 half of the intrauterine period, which is of great functional importance [7]. Also, the presence of posterior trifurcation is important in the treatment of aneurysms of the mouth of the posterior connective and anterior villous arteries located on the supraclinoid segment. Due to the frequent cases of trifurcation and the importance of the anterior villous artery, determine the need to maintain their patency in the treatment of these aneurysms [15].

Consequence and conclusions:

According to various data, the structure of the arterial circle of the cerebrum (Willis circle) is found in about half of the cases, the other half consists of various non-classical structures [7,11,12]. Also, some authors suggest a dependence of the structure of the arterial circle with the frequency of occurrence of cerebral circulatory disorders [16,4]. In

Trushel's studies, only people with non-classical variants of the structure of the Willis circle were found to have cerebrovascular disorders [3]. Knowing the structural features of the various variants of the Willis circle also allows you to choose the right tactics in making medical procedures, such as the treatment of aneurysms of the intracranial ICA, for example, paraclinoid aneurysms, which make up 20-33% of all cerebral aneurysms [15]. The posterior ICA trifurcations are of particular interest since, according to Trushel's data, they are found more often than others, in Gorbunov's work, trifurcation was also more common than others, and in combination with other phenomena of the structure of cerebral vessels [3,7,10,16]. In studies Kostrov O.Yu. and his colleagues, it was shown that among the patients with arterial aneurysms mainly women prevail. Moreover, they were 4 times more likely to have an open type of structure of the Willis circle. Thus, the features of angioarchitectonics of cerebral vessels, of course, predispose to the development of cerebrovascular disorders [13]. Anomalies and variants of the cerebral arteries are biologically less reliable and lead to the development of diseases. It is no accidentally that they are often associated with aneurysms [5].

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FEATURES OF IMMUNOHISTOCHEMICAL CHANGES IN PROXIMAL AND DISTAL RENAL TUBULES IN PATIENTS DIED FROM SEPSIS

МРНТИ 76.29.50

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Abstract

The aim of the investigation was to study immunohistochemical changes in the kidneys in patients who died as a result of sepsis.

Materials and methods. *Sectional materials were studied in 5 patients who died from sepsis: 4 women (80%) and 1 man (20%). Histological structural changes in renal tissue were investigated by histochemical and immunohistochemical methods. The autopsy was performed at an early stage (1-6 hours after death). The control group has included 10 kidneys of healthy people who died, as a result, an accident. Sections were made from paraffin blocks. Sections 6 ө thick were cut from paraffin blocks and Anty-E. Coli LPS antibody (2D7/1) 1/100 by the immunocytochemical method was used.*

Results. *Pronounced changes were observed in the proximal, distal tubules of the kidneys and Henle loop cells in histochemical and immunochemical studies. The following 1 (20%) patients had 3 (+ + +) staining in the proximal tubules, 2 (40%) 2 (++) in the proximal and distal tubules, 1 (20%) 1 (+) weak staining. Staining was not observed in 1 (20%) patient.*

Conclusion. *These changes were manifested primarily as a result of the accumulation of lymphocytes in the area of inflammation. Thus, under the influence of endotoxins LPS (lipopolysaccharide) with bacterial intoxication, irreversible changes in epithelial cells and sensitive tubules are not rarely observed, accompanied by fragmentation of the microvilli of the brush border on the apical part of the epithelial cells of the proximal tubules.*

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Keywords
sepsis, LPS endotoxin, Anty-E.Coli

Сепсистен қайтыс болған емделушілерде проксимальды және дистальды бүйрек арналарындағы иммуногистохимиялық өзгерістердің ерекшеліктері

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Аңдатпа

Зерттеудің мақсаты: *сепсистен қайтыс болған емделушілерде бүйректердег иммуногистохимиялық өзгерістерді зерттеу болды.*

Материалдар мен әдістер. *Секциялық материалдар сепсистен қайтыс болған 5 емделушілерде зерттелген: 4 әйелде (80%) және 1 ер адамда (20%). Бүйрек тініндегі гистологиялық құрылымдық өзгерістер гистохимиялық және иммуногистохимиялық әдістермен зерттелді.Патологоанатомиялық сою ерте кезеңде жүргізілді (қайтыс болғаннан кейін 1-6 сағаттан кейін). Бақылау тобы жазатайым оқиға салдарынан қайтыс болған дені сау адамдардың 10 бүйрегі болды. Парафин блоктарынан кесіктер жасалды. Парафин блоктарынан қалыңдығы 6 мкм кесілген және Anty-E қолданылған. Coli LPS antibody (2d7 / 1) 1/100, иммуноцитохимиялық әдістің көмегімен.*

Нәтижелері. *Гистохимиялық және иммунохимиялық зерттеу кезінде бүйректің проксимальді, дистальді арналарында және Генле ілмектерінің жасушаларында айқын өзгерістер байқалды. Келесі 1 (20%) пациентте проксимальды каналдарда 3 (+ +) бояу, проксимальды және дистальды каналдарда 2 (40%)2(++) бояу, 1 (20%) 1 (+) әлсіз бояу байқалды. 1 (20%) пациентте бояу байқалмады.*

Қорытынды. *Бұл өзгерістер, ең алдымен, қабыну аймағында лимфоциттер жасушаларының жинақталуы нәтижесінде пайда болды. Осылайша, ЛПС(липополисахарид) эндоксиндерінің әсерімен бактериялық уыттану кезінде эпителиалды жасушаларда және сезімтал каналдарда қайтымсыз өзгерістер жиі байқалады, проксимальды каналдардың эпителиалды жасушаларының апикальды бөлігінде щеткалы жиектің микроторсиналарының фрагменттауымен қатар жүреді.*

АВТОРЛАР ТУРАЛЫ

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Түйін сөздер
сепсис, эндотоксин ЛПС, Anty-e. Coli

Особенности иммуногистохимических изменений в проксимальных и дистальных почечных канальцах у пациентов, умерших от сепсиса

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Аннотация

Целью исследования было изучение иммуногистохимических изменений в почках у пациентов, умерших от сепсиса.

Материалы и методы. *Секционные материалы изучены у 5-и пациентов, умерших от сепсиса: у 4-ех женщин (80%) и 1-ого мужчины (20%). Гистологические структурные изменения в почечной ткани были исследованы гистохимическим и иммуногистохимическим методами.Патологоанатомическое вскрытие проводилось на ранней стадии (через 1-6 часов после смерти). Контрольная группа имела 10 почек здоровых людей, которые погибли в результате несчастного случая. Из парафиновых блоков были изготовлены срезы. Из парафиновых блоков вырезали срезы толщиной 6 мкм и использовали Anty-E. Coli LPS antibody (2D7/1) 1/100, с помощью иммуноцитохимического метода.*

Результаты. *Выраженные изменения наблюдались в проксимальных, дистальных канальцах почек и клеток петель Генле при гистохимическом и иммунохимическом исследовании. У следующих 1 (20%) пациента наблюдалось 3 (+++) окрашивания в проксимальных канальцах, у 2-х (40%)2(++) в проксимальных и дистальныхканальцах, у 1 (20%) 1 (+)слабое окрашивание. Окрашивание не наблюдалось у 1 (20%) пациента.*

Заключение. *Эти изменения проявились, прежде всего, в результате накопления клеток лимфоцитов в области воспаления. Таким образом,под воздействием эндоксинов ЛПС(липополисахарид) при бактериальной интоксикации нередко наблюдаются необратимые изменения в эпителиальных клетках и чувствительных канальцах, сопровождающиеся фрагментацией микроворсинок щеточной каймы на апикальной части эпителиальных клеток проксимальных канальцев.*

Ключевые слова
сепсис, эндотоксин ЛПС, Anty-E.Coli

Introduction

Sepsis is the Greek word for «rot». Sepsis is usually understood as blood poisoning, which is an inflammatory reaction of the body, including the development of multiple organ failure syndrome. In other words, sepsis is a systemic inflammatory response syndrome associated with a suspected or confirmed infection. Sepsis is not only a disease, but also a heterogeneous syndrome, which is the result of the interaction of the body and a pathogenic factor, manifested by biochemical reactions and stages of inflammation [1,2]. The clinical manifestations of sepsis depend on many factors, including pathogenic infections, clinical interventions, and the genetic and general health status of the body.

Sepsis is a process that is accompanied by hemodynamic and metabolic changes and leads to damage and death of tissues and organs [3,4]. Acute kidney injury (AKI), accompanied by sepsis, is a common disease. Toxic necrosis of renal tubule cells is different from typical ischemic damage to the kidneys. Therefore, the mechanisms underlying renal dysfunction in patients with sepsis are less clear [5,6].

AKI occurs in approximately 50% of patients with acute sepsis and the simultaneous occurrence of sepsis, almost doubles the risk of death from sepsis. In the modern world there is no targeted treatment of AKI accompanied by sepsis, and recent clinical trials have not established the optimal form of therapy for these patients [7,8]. However, in 50% of all cases of severe sepsis and septic shock, it is impossible to detect bacterial colonies in the blood [9,10].

An important pathogenetic factor that provides structural and functional changes in many organs and systems during sepsis is lipopolysaccharide (LPS). However, recent studies show that any critical condition is associated with an imbalance in the antioxidant system [11,12]. The appearance of “oxidative stress” in the body is associated with excessive formation of free radicals due to impaired oxygen metabolism during hypoxia caused by impaired microcirculation. However, the bacterial factor also plays an important role in the imbalance of the oxidation-antioxidant system during the complication of sepsis. Thus, the massive influx of microbial lipopolysaccharides into the bloodstream is accompanied by the activation of macrophages and neutrophils, which provide the formation of oxygen free radicals and the oxidation of lipid peroxide [13,14]. Since septicopyemia is accompanied by functional disorders of many organs, including the kidneys, we believe that studying the morphological features of

the kidneys and their ducts during sepsis can play an important role in the prevention and treatment of renal complications [15].

The aim of the study was to examine immunohistochemical changes in the kidneys in patients, died from sepsis.

Materials and methods

Sectional materials were studied in 5 patients: in 4 women (80%) and 1 man (20%) who died in 2016-2018 as a result of sepsis. The control group had 10 kidneys of healthy people who died as a result of an accident. The pathological study took place at an early stage - 3-6 hours after death. Tissues from the cortical and brain layers of the kidneys were studied by histochemical and immunohistochemical methods.

To prepare the paraffin block, sectional preparations were treated in 10% formalin buffer solution. At the next stage, the samples were dehydrated in 50˚, 70˚, 80˚, and 96˚ solutions of ethanol for 30 minutes; subsequently, they were processed stepwise, first in a xylene-paraffin solution in a concentration of 1:1 (in a mixture of xylene and paraffin in a ratio of 1:1), then in a liquid form of paraffin 2. Each stage proceeds in a thermostat at a temperature of 62˚C for 30 minutes. Later they were blocked in special forms and were prepared paraffin blocks. From each block obtained, two sections 5–6 em thick were prepared for samples of hematoxylin-eosin, histochemical and immunohistochemical staining for microscopic examination.

The obtained sections were processed in a thermostat at 65˚C for 1 hour, then in a toluene solution for 10 minutes to determine E. Coli Serotype 111: B4-Echericha LPS Coli. Then, for 5 minutes, the sections were processed in ethyl alcohol solutions 96˚, 80˚ , 75˚, 40˚. After this, the sections were washed 3 times with phosphate-buffered saline (PBS). Then diluted with 1/100 antigen retrieval Anty-E saline. Coli LPS antibody (2D7/1) was added to the samples and incubated for 1 hour. In positive samples with Anty-E. Coli LPS (2D7/1), the cell membrane is stained. The results are evaluated as positive (+) and negative (-).

The criteria for kidney damage during sepsis are as follows:

1) Interstitial edema;
2) Inflammatory cell infiltration;
3) Expansion in a Bowman capsule;
4) Damage to the proximal and distal tubules.

Each criterion was rated as 0-3 (0 - absence, 1 - minor damage, 2 - moderate damage, 3 - severe damage). The maximum score was 9.

In a bacteriological examination of blood and exudate isolated from the abdominal cavity, E. Coli was found in 3 cases, and Pseudomonas aeruginosa - in one case.

Results and discussion

During the macroscopic examination of the control group, the renal capsule was observed in a smooth condition and pink color. During microscopic examination of the kidneys, proximal and distal tubules retained their normal histoarchitectonic structure (arrow in Fig. 1).

Microscopic examination of the kidneys in patients who died as result of sepsis with hematoxylin-eosin staining showed marked changes in the tubules and interstitial tissue. As a result of sepsis, under the influence of endotoxin, interstitial edema, dystrophic and necrotic changes in the tubules are observed. In some proximal tubules, the disappearance of the ultra microvillis of the brush border is observed. The microvillis of the brush border, located in the apical part of the epithelial cells as a result of its lysis, were destroyed.

In the control group during immunohistochemical studies, as a result of the determination of anti-E.Coli LPS in the proximal and distal tubules and interstitial tissue, staining was not observed (Fig. 3, marked by black arrows).

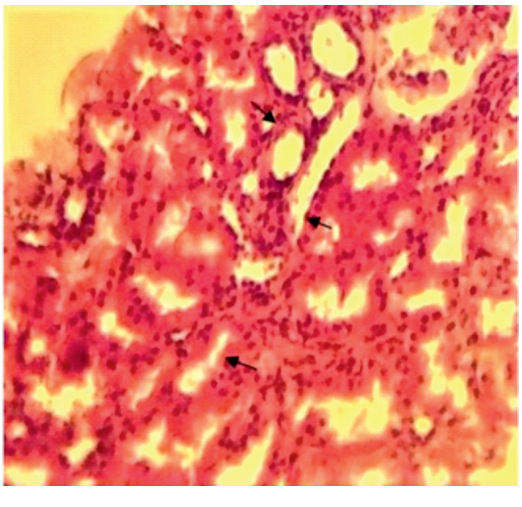

Figure 1.
Normal microscopic structure of renal tissue. Dye: Hematoxylin-eosin. Scale x40.

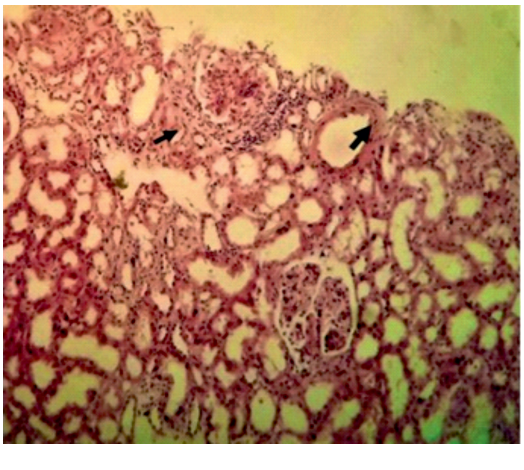

Figure 2.
Microscopic structure of the renal tissue of the study group. Dye: Hematoxylin-eosin. Scale: x40

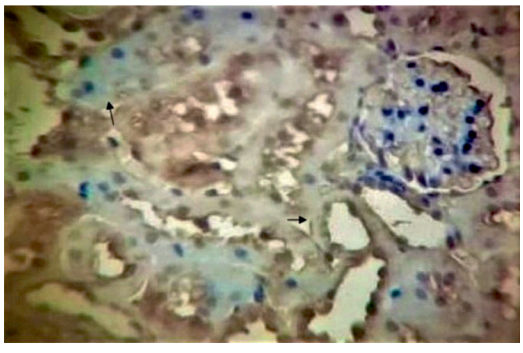

Figure 3.
Control group - staining. IHC. Anti-E. Coli LPS antibody. Scale x60.

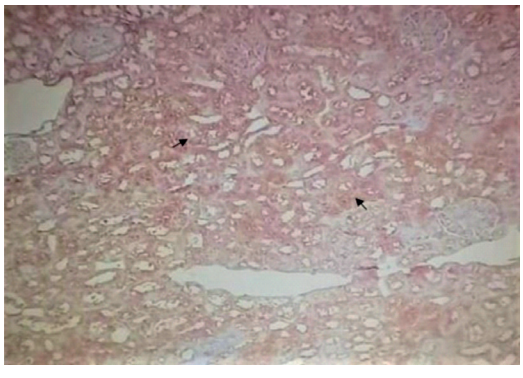

Figure 4.
The study group (endotoxin damage. IHC. Anti-E. Coli LPS antibody. Scale x20.

In the study of renal tissue preparations, weak (+), moderate (++) and pronounced (+++) staining areas were noted.

Marked changes were observed in the proximal, distal tubules of the kidneys and cells of the Henle loops during histochemical and immunochemical studies. In the following 1 (20%) patient - 3 (+++) staining was observed in the proximal tubules, in 2 (40%) - 2 (++) in the proximal and distal tubules, in 1 (20%) - 1 (+) weak staining. No staining was observed in 1 (20%) patient.

This staining was more pronounced in the proximal and distal tubules (Fig. 4, black arrow).

Thus, the results of the study showed that necrosis of the cells of the proximal and distal tubules and pronounced destructive changes in the basement membrane were observed in places where bacteria accumulated. Histochemical and immunohistochemical changes were observed in the proximal, distal tubules and Henle loop cells with septic damage. These changes are manifested primarily by the accumulation of lymphocyte cells in the area of inflammation and the destruction of the membranes of the renal tubules.

Thus, our study allowed us to obtain some morphological criteria for renal failure, which may develop as a result of endotoxin exposure in septic damage. In bacterial intoxication, irreversible changes in the epithelial tubular cells are often observed, which are sensitive to the effects of LPS endotoxins (lipopolysaccharides) and are accompanied by fragmentation of the brush border microvillis of the apical part of the proximal tubule epithelial cells.

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THE CASE OF A COMPLICATED AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE

УДК: 616-002.191:166-085-07 + 616.61-003.4

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Abstract

Autosomal dominant polycystic kidney disease is the most common inherited kidney disease found in adults. The urgency of the problem is due to the peculiarity of the development and growth of cysts, the inability of existing methods of treatment to prevent an increase in the size of cysts and kidneys, and progressive impaired renal function. This article describes the general characteristics of ADPP, presents an analysis of the clinical case of a complicated course of ADPP.

Keywords
duodenal ulcer, bleeding, treatment

Аутосомды-доминантты поликистоздық бүйрек ауруының асқынған жағдайы

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Аңдатпа

Аутосомды-доминантты поликистоздық бүйрек ауруы – ересектерде жиі байқалатын тұқым қуалайтын бүйрек ауруы болып табылады. Мәселенің өзектілігі – АДПБА кезіндегі кисталардың өсу және даму ерекшеліктері, қолданыстағы емдеу әдістерінің бүйрек және киста өлшемдерінің ұлғаюының алдын алуға қабілетсіз болуы. Осы мақалада АДПКА-ның жалпы сипаттамасы, АДПБА асқынған ағымының клиникалық жағдайының талдауы берілген.

Түйін сөздер
аутосомды-доминантты поликистоздық бүйрек ауруы, бүйрек поликистозы, асқынулар

Случай осложненной аутосомно-доминантной поликистозной болезни почек

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Ключевые слова
аутосомно-доминантная поликистозная болезнь почек, поликистоз почек, осложнения

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Аннотация

Аутосомно-доминантная поликистозная болезнь почек - наиболее распространенное наследственное заболевание почек, выявляющийся у взрослых. Актуальность проблемы обусловлена особенностью развития и роста кист, неспособностью существующих методов лечения предотвратить увеличение размеров кист и почек, прогрессирующее нарушение функции почек. В настоящей статье описана общая характеристика АДПБП, представлен анализ клинического случая осложненного течения АДПБП.

Introduction

The urgency of the problem of autosomal dominant polycystic kidney disease (ADPBP) is the development and growth of cysts, the inability of existing treatment methods to prevent an increase in the size of cysts and kidneys, and progressive impairment of renal function.

ADPBP is the most common hereditary kidney disease, with a prevalence rate in the general population of 1: 1000–2500, often detected in adulthood (30–40 years) [1, 2].

ADPBP is caused by mutations in the PKD1 (chromosome 16p13.3) and PKD2 (chromosome 4q21) mutations (abbreviation for Polycystic Kidney Disease). These genes encode proteins called polycystins 1 and 2 (PC1 and PC2). PKD1 mutation is responsible for 78% of cases of ADPD; PKD2 mutation is found in 15% of patients [3]. Also, the PKD 3 gene (11q12.3) was recently discovered, which is responsible for the interaction of PC1 and PC2. This mutation contributes to 0.3% of cases [4, 5].

The PKD 1 mutation is associated with a more severe course of the disease, faster progression to the terminal stage of chronic kidney disease, and early death. So, in patients with PKD 1, by the age of 50, chronic renal failure develops in 35% of cases, by the age of 60 - in 70%, and by the age of 70 - exceeds 95% [6]. Today there is no way to completely cure ADPP.

Cysts can be localized in the medulla, in the cortical layers of the kidney; in the pelvis of the kidney, in the perinefral region. Kidney cyst in ADPBP is an extended segment of the nephron or collecting tube. The wall of the cyst is lined with one layer of altered tubular epithelium (cubic, flattened) or a thin layer of connective tissue, it is a partition between them. The size of the cysts depends on the amount of content and can vary within various limits: from small (<2 mm in diameter, containing not > 3 ml of fluid) to giant. Areas of cystic tissue are combined with areas of healthy renal transplant, the amount of which, as the first grows, rapidly decreases. Cyst fluid communicates with tubular contents, blood vessels of the kidneys, and contents of the renal pelvis [7].

ADPBP is characterized by the development and growth of multiple cysts in the parenchyma of both kidneys. Progressive loss of kidney function occurs over several decades and often leads to the terminal stage of chronic kidney disease. Hypertension is a common manifestation of ADPD and is found in most people with enlarged kidneys or a reduced glomerular filtration rate.

Non-genetic factors also influence the clinical manifestations and the rate of progression of ADPD. This is evidenced, for example, by the fact that cystic lesion of the liver is more severe in women, especially in those who took combined oral con-

traceptives, replacing estrogen therapy, and who have a history of multiple pregnancies. It is believed that in men with ADPD, the rate of increase in cyst size is higher than in women, and the terminal stage of chronic renal failure with ADPD occurs earlier in men, which indicates the role of sex hormones that can change the course of the disease [8].

Complications of the disease can be macrohematuria, infection and suppuration of cysts, nephrolithiasis, which are manifested by acute or chronic pain in the lumbar region [9].

Polycystic liver disease (PLD) is one of the most common extrarenal manifestations (in over 83% of cases) of autosomal dominant polycystic kidney disease. Although in many patients PLD is asymptomatic, nevertheless, in some cases, patients develop hepatomegaly, which is accompanied by abdominal pain, a feeling of fullness, shortness of breath, and a decrease in the quality of life. ADPBP can also be accompanied with pancreatic and / or spleen cysts, intestinal diverticulosis (65%), heart valve pathology (24%), ovarian cyst lesion (40%), intracranial vascular aneurysms (10%), inguinal hernia (15%) [10].

Treatment

For ADPB symptomatic therapy is performed. When detecting arterial hypertension, an exception is currently being made to renal pathology. It is necessary to exclude stay in adverse weather conditions, heavy physical exertion. It is important to follow a diet in the early stages with a moderate restriction of animal proteins, as kidney failure progresses, more strict adherence to a low-protein diet [11].

Antihypertensive therapy should include inhibitors of angiotensin-converting enzyme, calcium antagonists (lerkamen), and beta-blockers (concor, betalok-ZOK, moxonidine) [12].

In patients with severe polyuria for the prevention of dehydration, electrolyte loss, it is advisable to use thiazides to distally reduce the release of electrolytes and water. Therapy of urinary tract infection should be carried out taking into account the results of bacteriological examination of urine. Periodically carry out herbal medicine. In patients with frequent relapses of macrohematuria, a coagulogram study is necessary.

With the development of anemia - erythropoietin preparations, intravenous administration of iron are useful. With the development of terminal chronic renal failure to perform programmed hemodialysis. It must be borne in mind that the introduction of heparin during hemodialysis sessions can cause the appearance of macrohematuria, hemorrhages in the cysts, and especially severe consequences can be observed with hemorrhages in the brain cysts. A radical treatment for ADPD is kidney transplantation [11].

The purpose of this article was to demonstrate the clinical, laboratory, and instrumental features of a case of complicated ADPP, as well as the tactics of treating a patient.

Clinical case

Patient O., a woman born in 1975, 44 years old, was admitted to the clinic on December 7, 2019, with complaints of pain in the lumbar region on both sides, more on the right, an fever to 38.0°C for 2 days, weakness, malaise.

Patient considers ill for three days before admission. According to her own treatment did not accept. In connection with the deterioration of the condition and the growth of the above complaints, she called an ambulance team and was taken to the admission department of the GBSNP in Almaty, examined by the urologist. Laboratory and instrumental research methods were prescribed. Spasmolytic and analgesic therapy was carried out, after which the pain syndrome was partially stopped. Then the patient was hospitalized in the Department of Urology for further examination and determination of treatment tactics.

From the anamnesis, polycystic kidney and liver disease was detected about 10 years ago. Inspected annually by the therapist at the place of residence. The last time she was examined was in November 2018, outpatient treatment was recommended. Kidney transplantation not suggested.

The patient's condition is serious, due to pain and intoxication syndromes. External pain scale - 2 points. The patient's consciousness is clear, adequate.

On palpation, the abdomen is soft, painless. The lumbar region is symmetrical, with superficial palpation in the projection of the right kidney pain is noted. With deep palpation: the kidneys are mobile, pain on the right. Symptom striking (+) on both sides, more on the right. Urination is frequent, painless.

Laboratory methods: The full blood count determined: leukocytosis, moderate erythropenia and anemia, thrombocytopenia. In dynamics, the number of leukocytes decreased (table 1).

In the biochemical analysis of blood, an increase in nitrogenous toxins was revealed, which indicated a decrease of renal function; moderate increase glucose in blood (table 2).

Coagulological blood tests were within normal limits.

In the general analysis of urine, signs of renal failure, inflammation (proteinuria, hematuria, leukocyturia) were revealed (table 3).

When analyzing urine according to Nechiporenko, a large number of leukocytes in the urine was detected - 20,250 Leu / ul (normal indicators 2000-4000 in 1).

Table 1.
Dynamic indicators of a full blood count.

Components, Elements	Standard values	Date, Results				Values of SI Units
		07.12.2019	08.12.2019	13.12.2019	18.12.2019	
White blood cells (WBC)	4,0-9,0	17,2	11.70	14.30	10.40	10^9/l
Red blood cells (RBC)	4,3-5,5	4,09	3.47	2.54	2.51	10№l/l
Hemoglobin (HGB)	120-140	118	101.00	74.00	72.00	g/l
Hematocrit (HCT)	0,39-0,49	38,25	32.40	23.80	23.70	%
Platelets (PLT)	150–400	198	205.00	466.00	819.00	10^9/l

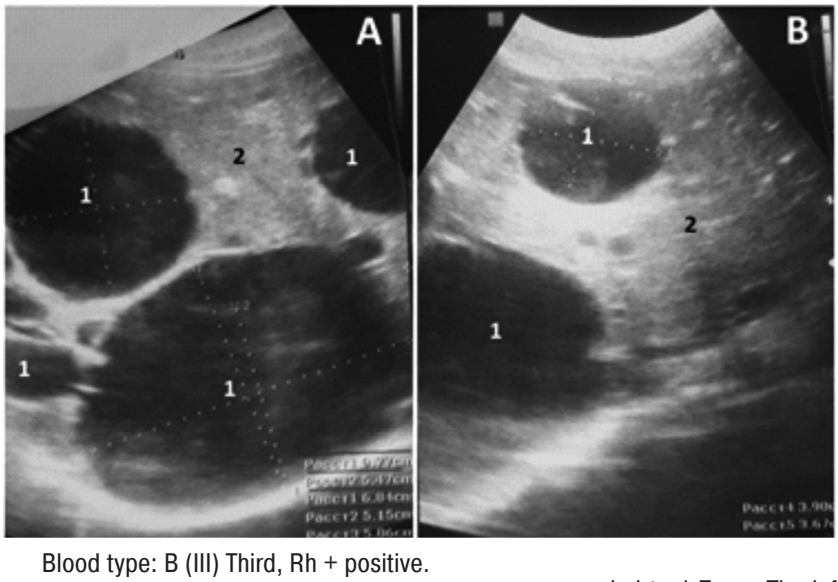
Table 2.
Modified components of a biochemical blood test.

Components, Elements	Results	Standard values	Values of SI Units
Urea	12.49	2,7 - 8,3	mmol/L
Creatinine	200.05	men (44 - 115), women (45 - 97)	umol/L
Glucose	7.17	3,50 - 6,00	mmol/L

Table 3.
Modified components of the urine test.

Components, Elements	Results	Standard values	Values of SI Units
Color	Light yellow		
Transparency	muddy		
Number	50,0		ml
Protein (PRO)	2+	Negative	‰
pH	6,0	5-7	
Specific gravity (S.G.)	1.010	1,005-1,025	
Blood (BLD)	2+	Negative	mg/L
Ketones (KET)	+-	Negative	mmol/L
Leukocyte (LEU)	500	to 25	Leu/uL

Figure 1.
Sonograms of the right (A) and left (B) lobes of the liver during longitudinal scanning: 1 - multiple cysts, 2 - liver parenchyma.



Blood type: B (III) Third, Rh + positive.

Instrumental methods of research: Ultrasound examination of the liver. The size of the liver is increased, the oblique vertical size of the right lobe is 20.5 cm, the oblique vertical size of the left lobe is 13.9 cm. The echostructure is even, the echo density is increased, the intrahepatic ducts are not dilated. In the structure of the liver, multiple anechogenic thin-walled cystic formations are determined, with sizes from 0.7 cm to 9.2 cm, homogeneous liquid contents. Conclusion: Polycystic liver disease. Diffuse changes in the liver parenchyma (figure 1).

Ultrasound examination of the kidneys. The location of the kidneys is typical. The right kidney is 15.3 x 7.8 cm in size, the parenchyma is thinned. In the structure of multiple anechogenic masses in diameter from 0.5 cm to 5.6 cm. Pyelocaliceal system – major calyxes expanded to 1.7 cm. The left kidney is 16.5x8.2 cm in size, the parenchyma is thinned. In the structure, anechogenic masses are in diameter from 0.5 cm to 6.3 cm. The pyelocaliceal system is not expanded. Conclusion: Polycystic kidney disease. Calicoectasia on the right. Acute pyelonephritis on the right (Figure 2).

Magnetic resonance imaging of the abdominal cavity and retroperitoneal space. On a series

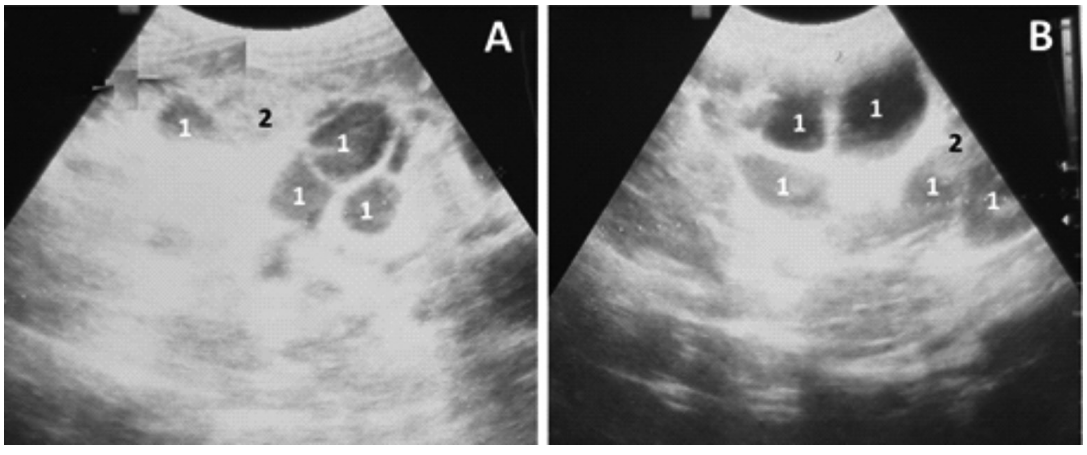


Figure 2.
Sonograms of the right (A) and left (B) kidneys during longitudinal transluminal scanning: 1 - multiple cysts, 2 - renal parenchyma.

of MRI tomograms, the kidneys are relatively the same, significantly increased to 20.0x10.0x1010 cm. The usual structure of the kidneys is not traced against the background of many cystic lesions diffusely occupying the entire volume of the renal parenchyma, so that pyelocaliceal system is not traced on both sides. There is also a heterogeneous composition of the contents of the cysts, some of which have sedimented hemorrhagic contents, some are filled with a liquid with a high protein content, which intensively limits diffusion. A larger number of cysts with a changed composition of contents is located in the right kidney. Paranephric fiber is not changed. Adrenal glands without features.

The liver is sharply enlarged (maximum craniocaudal size - 24.0 cm, maximum transverse size - 24.6 cm). In the structure of the liver, multiple cystic lesions ranging in size from 0.5 to 10.0 cm in diameter, filled with a homogeneous fluid without impurities. The hepatic ducts are not dilated. The portal vein is not dilated, up to 0.9 cm in caliber (Figure 3, 4).

Conclusion of MRI: Multicystic lesions of the liver and kidneys, hemorrhages and inflammatory changes in several cystic formations of the right kidney. Inflammatory changes in single cysts of the left kidney. Severe hepatomegaly and bilateral nephromegaly.

MR picture of complicated polycystic kidney disease with diffuse extrarenal liver damage.

Chest x-ray. On the survey x-ray of the chest organs, pulmonary fields without fresh focal and infiltrative shadows. Pulmonary pattern reinforced,

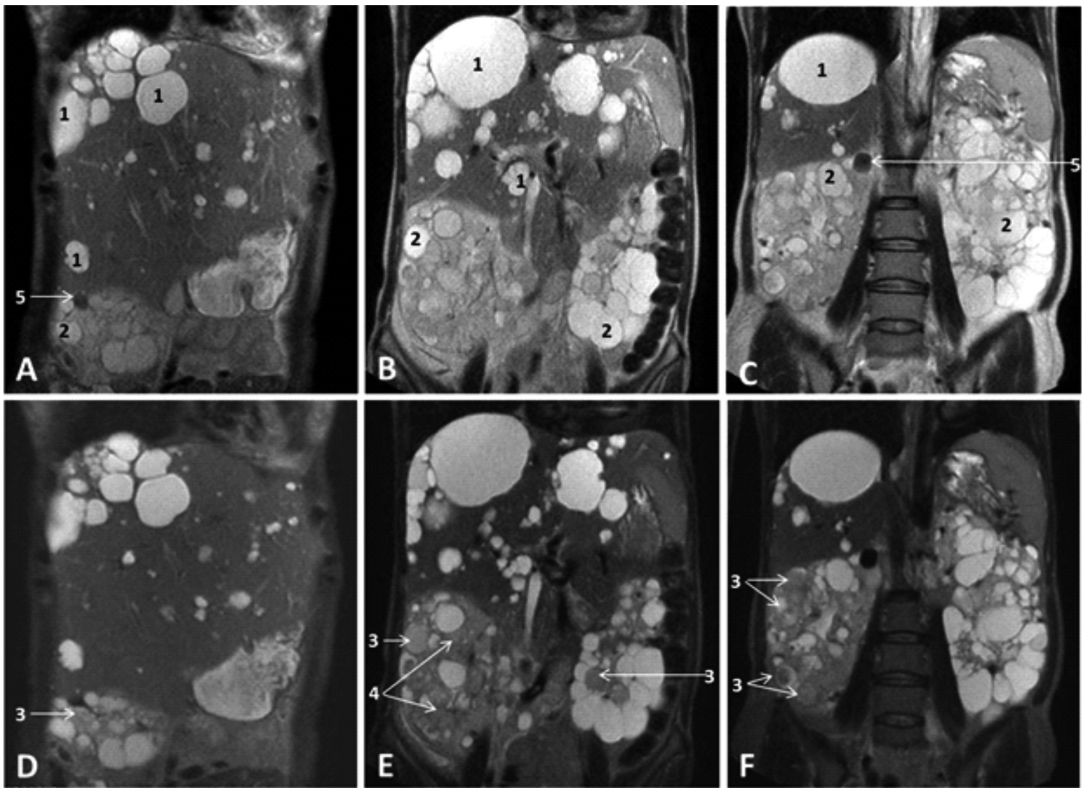


Figure 3.
MRI - tomograms in the coronal view. A, B, C - in T2; D, E, F - Diffusion Weighted Imaging (DWI): 1 - multiple cysts in the liver, 2 - multiple cysts in the kidneys, 3 - cysts with purulent contents, 4 - kidney parenchyma with inflammatory changes, 5 - single cysts with hemorrhagic contents.

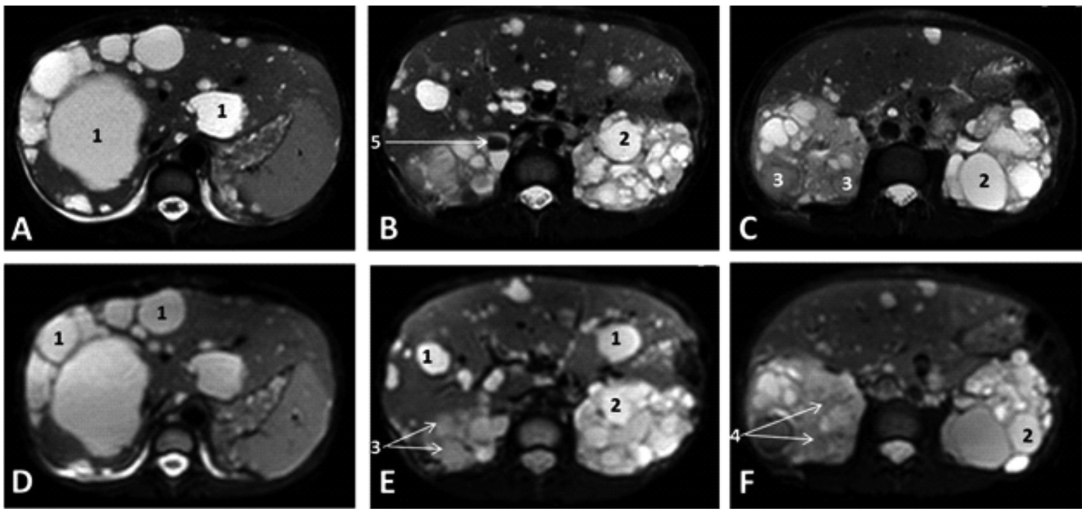


Figure 4.
MRI - tomograms in axial view. A, B, C - in T2 WI; D, E, F - Diffusion Weighted Imaging (DWI): 1 - multiple cysts in the liver, 2 - multiple cysts in the kidneys, 3 - cysts with purulent contents, 4 - renal parenchyma with inflammatory changes, 5 - cysts with hemorrhagic contents.

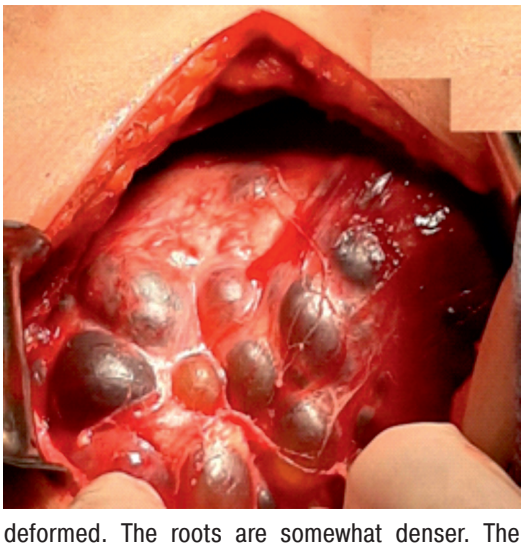


Figure 5.
Intraoperative image of a polycystic right kidney with purulent-necrotic contents of cysts.

deformed. The roots are somewhat denser. The diaphragm is smooth, the sinuses are free. The shadow of the heart corresponds to age. Conclusion: Chronic bronchitis.

Based on the foregoing, a diagnosis was made: Polycystic kidney and liver, type adult. Acute tubulo-interstitial nephritis. Suppurative cysts of the right kidney. Chronic bilateral pyelonephritis, exacerbation on the right. Chronic renal failure of the I degree.

Complex treatment was prescribed: Mode II, diet table No. 7. Antibacterial, detoxification, analgesic therapy. Preparing for the operation.

On December 10, 2019, an operation was performed: Lumbotomy on the right. Autopsy and excision of suppurative cysts of the right kidney. Lumbostomy.

Protocol of operation: The position of the patient on the left side. After processing the surgical field three times with a solution of povidone and once with a solution of spirit, a lumbotomic skin incision was made according to Fedorov about 10.0 cm long. Hemostasis. The wound was opened in layers (subcutaneous tissue, aponeurosis, muscles) to the peritoneum. The peritoneum is pushed back. Hemostasis. Paranephric fiber was opened, the latter is gibbed, there is vitreous edema. With technical difficulties, the right kidney is highlighted. The right kidney is increased in size to 16.0 x 8.0 cm. During the audit, the structure of the right kidney is represented by multiple thin-walled cystic formations, sizes from 0.5 cm to 6.0 cm, with turbid fluid contents in many of them, as well as the presence of a hemorrhagic component in some cysts (Figure 5).

Excision and drainage of inflamed cysts was performed, purulent hemorrhagic contents were obtained. A small amount of biological material was taken for bacteriological studies in order to identify an infectious agent and sensitivity to antibiotics. Pieces of tissue were taken from several cyst walls for histological examination. Conducted hemostasis. The wound cavity and kidney are actively washed with solutions of chlorhexidine, furatsillin. A lumbostomy is established. The wound cavity was washed with furatsillin solution, drained. Control for hemostasis is dry. The wound is sutured in layers. Stitches are applied to the skin. Spirit. Aseptic dressing.

Postoperative diagnosis: Polycystic kidney disease, type adult. Suppurative cysts of the right kidney. Chronic bilateral pyelonephritis, exacerbation on the right. Chronic renal failure of the I degree.

Conclusion of bacteriological research: Enterobacter Aerogenes. This is an optional anaerobic, gram-negative, rod-shaped bacterium of the Enterobacteriaceae family, which is a conditionally pathogenic microflora of the gastrointestinal tract. It is resistant to ampicillin, sensitive to meropenem, cefuroxime, cefotaxime, amikacin, ceftazidime.

The histological conclusion: fibrous adipose tissue with extensive fields of hemorrhage, purulent inflammation.

After complex therapy, body temperature decreased from 38.0°C to normal values, with episodes of febrile temperature (37.1-37.5°C) in the following days after surgery.

On the tenth day after the operation (20/12/2019), the general condition of the patient is relatively satisfactory, stable in dynamics. Consciousness is clear. Complaints of a periodic increase in body temperature to 37.5°C. Urination independent, urine light. Swathe is clean, removed. The wound is calm, with no signs of inflammation, the seams lie evenly. Toilet wounds. Aseptic swathe. Continues treatment.

After complex treatment (antibiotic therapy, surgery, symptomatic therapy), the patient was discharged in satisfactory condition on day 16 from the hospital under the supervision of a urologist in the clinic at the place of residence.

Conclusion

Thus, in this clinical case, the importance of understanding and timely revealing the complications of ADPKD, the features of clinical, laboratory and instrumental research methods, as well as the tactics of treating a patient with purulent-necrotic inflammation of the kidney cysts, is shown. It is necessary to remember the possibility of suppuration of cysts not only of the kidneys, but also of other organs, which can cause the development of a purulent-necrotic process of cystic transformed kidneys, which will further aggravate the morphological and functional state of the kidneys. A brief review of ADPKD will inform practitioners, especially young professionals, about the etiology, clinical, and instrumental manifestations of the disease and the complication of ADPKD.

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УДК 616.24.-002.5:579.252.55

EXPERIENCE OF SURGICAL TREATMENTS OF XDR-TB PATIENTS WITH NEW AND REPURPOSED PREPARATIONS CHEMOTHERAPY

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Keywords

Organization of surgical care, thoracic surgery, combined Echinococcus, multidisciplinary approach

Abstract

Performed analysis of experience sing surgical methods of treatment of 22 patients with M/XDR-TB with chemotherapy by new and repurposed preparations. Clinical surgical efficiency was achieved in 20 (91.0%) patients with mortality in 2 (9.0%) patients.Of the 14 patients who completed the full course of chemotherapy, the outcomes of treatment were "Cured" in 7 (31.8%), "treatment completed" in -5 (22.7%) patients, and "died" in 2 (14.3%). Other 8 (36.4%) patients continue treatment. The outcome "Treatment success" was obtained in 12 (85.7%) patients.

Кеңейтілген дәріге төзімді туберкулезді жаңа және қайтапрофильденген дәрілермен химиотерапия фонында хирургиялық емдеу әдістерін қолдану тәжірибесі

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Түйін сөздер

Дәріге төзімді туберкулез, хирургиялық ем, жаңа және қайта профильденген дәрілермен химитерапия, резекциялық материал

Аңдатпа

Жаңа қайта профилирленген туберкулезге қарсы дәрілердің химиотерапиясының фонында туберкулездің кеңейтілген дәріге төзімді түрі бар 22 науқасқа хирургиялық емдеу әдістерін қолдану тәжірибесінің нәтижелеріне талдау жүргізілді. Науқастардың өлім-жітімі 2 (9,0%) болса, 20 (91,0%) науқаста клиникалық хирургиялық тиімділік анықталды. Химиотерапияның толық курсынан өткен 14 науқастан 7 (31,8%) «Емделді», 5 (22,7%) «емдеу аяқталды», 2 (14,3%) «қайтыс болды» деген нәтижелер алынды. Қалған 8 (36,4%) науқас ем алуды жалғастырып жатыр. 12 (85,7%) науқаста «Емдеу сәтті өтті» деген қорытынды бар.

Опыт применения хирургических методов лечения у больных ШЛУ ТБ на фоне химиотерапии новыми и перепрофилированными препаратами

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Аннотация

Проведен анализ результатов опыта применения хирургических методов лечения у 22 больных М/ШЛУ ТБ на фоне химиотерапии новыми перепрофилированными противотуберкулезными препаратами. Клиническая хирургическая эффективность достигнута у 20 (91,0%) больных при летальности у 2 (9,0%) больных.

Из 14 больных, завершивших полный курс химиотерапии исходы «Вылечен» получен у 7 (31,8%), «лечение завершено» у 5 (22,7%) больных, «умер» у 2 (14,3%). Остальные 8 (36,4%) пациентов продолжают лечение. Исход «Успех лечения» получен у 12 (85,7%) больных.

Ключевые слова

М/ШЛУ ТБ, хирургическое лечение, химиотерапия новыми и перепрофиллированными препаратами, резекционный материал, микробиологические исследования

Introduction

The Drug-resistant tuberculosis has become one of the main obstacles to control this disease [1-4] The World Health Organization reports that the number of patients who suffer from rifampicin or multidrug-resistant tuberculosis is increasing annually. In addition, the treatment success rates achieved worldwide are suboptimal, barely exceeding 50%. This proportion is reduced up to 25% patients with extensively drug-resistant tuberculosis [1,2,5].

Thus, the cure of the vast majority of patients with M / XDR-TB is relevant [6].

The modern WHO protocols for chemotherapy with new and redesigned drugs contain recommendations on the use of surgical methods for treating M / XDR-TB patients, but at the same time, there are no reliable sources in the literature on the development of indications and terms for surgical interventions at the stages of an individual treatment regimen using new a and re-profiled drugs, on the effectiveness of their usage, there are no publications on microbiological studies of resection material in abacillated patients with various tuberculosis and lung after chemotherapy with new drugs and developed, especially when destructive processes [7].

Therefore, the study of the above issues is important and relevant for modern phthisiology.

The purpose of the study: - to give a preliminary assessment of the results of surgical treatment methods on the outcomes of treatment of patients with XDR-TB with chemotherapy with new and redesigned drugs.

Materials and methods

The main goal is to achieve the study, we have performed a descriptive analysis of the results of the use of surgical methods of treatment in 22 patients with M / XDRTB on the background of chemotherapy with new and redesigned drugs.

In the analyzed group, the number of men was 13 (59.1%), women - 9 (40.9%). In most cases, in the analyzed group, there were young people from 20 to 40 years old, the proportion of which was 77.3% , one child 5 years old (4.5%,), from 41 to 50 years old, 3 (13.62%) and one patient had 51 years old (4.5%).

According to the clinical forms, the patients were distributed as follows: the vast majority of 18 (81.9%) patients had fibro-cavernous pulmonary tuberculosis of different localization, 2 (9.1%) were diagnosed with pulmonary tuberculosis, and one (4.5%) infiltrative tuberculosis complicated by em-

pyema of the pleura, in the other, empyema of the pleura.

According to the localization of the process, the majority of 15 (68.2%) patients had a right-sided lesion of the lungs with the tuberculosis process, and the remaining 7 (31.9%) patients had left-sided damage.

The prevalence of the tuberculous process in the lungs in the analyzed group of patients was different: lung damage was observed in 10 (45.4%) patients, lung lobes in 7 (31.8%) patients, segments in 3 ((13.6%), damage pleura in 2 (9.1%) patients.

The disease duration in 14 (63.6%) patients ranged from one year to 5 years, in 6 (27.3%) patients - 8-10 years, and in 2 (9.1%) - more than 16 years.

For all patients, the decision of the centralized CVCC, based on the drug resistance test, the clinical protocol recommended by WHO and the methodological recommendations of the NSCF MH RK "Practical recommendations for the use of short-term, standard and individual treatment regimens for RU and M / XDR-TB using new and redesigned anti-TB drugs" from 2018, treatment regimens with new and redesigned drugs were prescribed [8].

According to drug resistance tests, all patients had multidrug-resistant tuberculosis and corresponded to category IV: among them, 17 (77.3%) had XDR-TB, 3 (13.6%) had pre-XDR-TB, and 2 (9.1%) - MDR-TB. Among them, in 2 sick children, an individual treatment regimen was prescribed in connection with reliable contact with parents with M / XDR-TB, and in 2 patients with pulmonary tuberculomas after examining the resection material.

The prior to the use of chemotherapy in patients with chemotherapy with the use of new and redesigned drugs, bacterial excretion was noted in 20 (91.0%) patients, with the exception of 2 (9.1%) patients with tuberculomas.

The Chemotherapy treatment was controlled and was planned for 20 months.

The individual treatment regimen included drugs of at least five drugs with confirmed preserved sensitivity of the MBT from the following list: Lfx (Mfx), Cm (Am), Pto, Cs, Lzd, Cfz, Bdq, Dlm, Z, E, H (high dose), PAS, Amx / Clv, Imp / Cln.

Doses were prescribed in accordance with the weight category.

The Patients took all drugs (daily dose) in a controlled manner throughout the course of treatment. Bdq (or Dlm) was prescribed for 6 months and extended to 12 months or more by the decision of the Central Control Commission in cases where the remaining regimen was not effective enough (less than 3 effective drugs) and the tolerability of new drugs was good.

When conducting controlled chemotherapy in the IRL mode, all patients strictly observed the assessment of the condition of patients at the beginning of treatment, during and after treatment according to the treatment monitoring schedule.

The monitoring schedule for monitoring the treatment of patients of the analyzed group, as well as compliance with pharmacovigilance, in order to identify unwanted (AE) and serious adverse events (AE), was carried out by responsible specialists of the End TB project and the NSCF.

Due to the long history of the disease and repeated chemotherapy courses, all patients before the operation suffered from many concomitant diseases: chronic gastritis, duodenitis, cholecystitis, anemia, chronic hepatitis, chronic obstructive pulmonary disease, pyelonephritis, etc. The presence of concomitant diseases was not a contraindication to surgical intervention , since symptomatic therapy during preoperative preparation allowed to stabilize the course of these problems levany and did not prevent the implementation of surgical interventions regarding the main process.

During the period of chemotherapy for patients, various undesirable phenomena arose associated with the use of anti-TB drugs, which were resumed after detoxification therapy and normalization of intoxication indicators.

In general, in the analyzed group only 3 (13.6%) patients had a temporary withdrawal of drugs. In other cases, drug tolerance was satisfactory.

Against the background of chemotherapy, taking into account the clinical and radiological dynamics and bacteriological studies of sputum in patients of this group, which characterize the stabilization of the pulmonary tuberculosis process, various surgical procedures have been performed.

The main indication for surgical intervention in this category of patients was the presence of extensive destructive changes in the lungs that did not have a tendency to reverse development.

In all patients, in terms of preoperative preparation and prevention of postoperative complications, bronchological studies of the lungs were performed, which were accompanied by a biopsy and ALS sampling from the proposed resection sites for lobectomy, pulmonectomy ..

In order to prepare patients for collapse surgery, pleurectomy, decortication, thorough rehabilitation of empyema cavities with bacteriological control was carried out.

In the postoperative period, all resection material was subjected to pathomorphological and microbiological examination.

The clinical effectiveness of the use of surgical treatment methods was assessed by the occurrence of postoperative complications, and the effectiveness of the entire complex treatment of these patients, taking into account the chemotherapy, was carried out according to the outcome of treatment.

Results

The nature of surgical interventions is presented in table 1.

In the postoperative period, 2 (9.0%) patients died, of which, in the early postoperative period, one patient died from pulmonary embolism after pneumonectomy, the other patient from cardiopulmonary insufficiency A 2 months after TMPL surgery.

In other cases of postoperative complications were not observed:

- In 11 (50.0%) patients, after segmental resections and lobectomies, the lungs were completely straightened.
- In 6 (27.3%) cases after pneumonectomy, the hemitorax is completely filled with the formation of fibrothorax, without mediastinal displacement
- In 4 (18.2%) patients after TML, in the zones of collapse, a complete decline in caverns was noted.
- In one case, after pleurectomy and decortication, the lung is completely straightened and without signs of a residual cavity

Thus, the clinical effectiveness of surgical interventions in the early postoperative period was achieved in 20 (91.0%) patients.

Treatment outcomes for M / XDR-TB patients are presented in Table 2.

As shown in the table, as a result of the use of surgical treatment methods with chemotherapy with new and redesigned drugs in M / XDR-TB patients, outcomes were obtained in 14 (63.6%) patients. Of these, "Treatment success" was established in 12 (85.7%)) patients, 2 (14.3%) patients died from pulmonary embolism.

The remaining patients continue treatment.

Thus, the use of surgical methods in patients with M / XDR-TB with the use of the treatment regimen with new and redesigned drugs made it possible to achieve clinical efficacy in 20 (91.0%) cases, and the outcome "treatment success" of the entire complex treatment was established in 12 (85,7%) of 14 (63.6%) patients who have completed chemotherapy to date. Despite the small number of studies, cultural studies of resection material in abacillated patients at the time of surgery indicate the effectiveness of new and redesigned drugs in treatment regimens.

Table 1.
The nature of surgical interventions in patients with M / XDR-TB

Number of patients	Number of patients	
	absolute number	%
The Segmental resection	5	22,7
Lobectomy	6	27,3
Pneumonectomy	6	27,3
Thoracomyoplasty	3	13,7
Thoracomyoplasty using a silicone implant in combination with valvular bronchial block	1	4,5
Pleurectomy, decortication	1	4,5
Total	**22**	**100,0**

Table 2.
Outcomes of complex treatment of the analyzed group of patients with M / XDR-TB

Outcomes	Number of Patients (n-22)	%
Cured	7	31,8
Treatment completed	5	22,7
Treatment failure	-	-
Died	2	9,1
Continues treatment	8	36,4

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ДНИ КАЗАХСТАНСКОЙ МЕДИЦИНЫ В КЫРГЫЗСКОЙ РЕСПУБЛИКЕ, 13-14 НОЯБРЯ 2019 Г., БИШКЕК

13 - 14 ноября в Бишкеке стартовали Дни казахстанской медицины. Данное событие направлено на усиление сотрудничества и расширение взаимодействия между медицинскими организациями Казахстана и Кыргызстана.

Казахстанскую делегацию представляли ведущие специалисты Министерства здравоохранения РК, ведущих медицинских центров, государственных и частных организаций сферы здравоохранения, ВУЗов, производителей фармацевтической продукции, медицинской общественности. Возглавлял казахстанскую делегацию министр здравоохранения РК Елжан Биртанов.

Национальный центр хирургии представлял председатель правления, профессор Баймаханов Болатбек Бимендеевич.

Болатбек Бимендеевич на сессии высокого уровня представил доклад на тему: органная трансплантация в РК. Также подписаны меморандумы о сотрудничестве с Ассоциацией гепатологов Кыргызстана, Национальным хирургическим центром МЗ КР, а также с Центром материнства и детства КР, которые будут способствовать дальнейшему сотрудничеству, а также росту интеллектуальных и инновационных ресурсов медицинских организаций.

Во второй день проведён мастер - класс на тему: Актуальные вопросы гепатологии. Профессор Центра Ильясова Бибигуль Сапарбековна представила доклады на темы:

1. Факторы прогрессирования вирусного гепатита В и вирусного гепатита В с дельта-агентом в Казахстане.
2. Вопросы трансплантационной гепатологии. Основные результаты.
3. Актуальные вопросы гепатологии Кыргызстана.

Также проконсультированы пациенты с заболеваниями печени. Разобраны некоторые клинические случаи.

ДНИ КАЗАХСТАНСКОЙ МЕДИЦИНЫ В УЗБЕКИСТАНЕ 21-22 НОЯБРЯ 2019 Г., ТАШКЕНТ

В Республике Узбекистан, в городе Ташкент, прошли дни казахстанской медицины под эгидой «Года Республики Казахстан в Республике Узбекистан». В рамках межправительственных договоренностей организовано данное мероприятие в целях укрепления и развитие диалога между системами здравоохранения, медицинского образования республик, а также обмена опытом по новым технологиям диагностики и лечения пациентов.

Казахстанскую делегацию из числа представителей национальных центров, вузов, управлений здравоохранения возглавил Министр здравоохранения Биртанов Елжан Амантаевич.

Делегация из Национального научного центра хирургии им. А.Н. Сызганова приехала в расширенном составе. Ведущие специалисты центра: заведующий отделом интервенционной кардиологии PhD Баимбетов Адиль Кудайбергенович, заведующий отделом эндоскопии, магистр здравоохранения, Абдрашев Ерлан Байтуреевич, заведующий отделом трансплантации почки, врач высшей категории, Ибрагимов Равиль Пашаевич, главный научный сотрудник, профессор Ильясова Бибигуль Сапарбековна, заведующий отделом гинекологии, врач высшей категории Усенов Кадыржан Маратович, врач гинеколог высшей категории Чупин Александр Николаевич, руководитель стратегического блока, кандидат медицинских наук, Миржакыпов Арман Толегенович. Возглавил делегацию Председатель правления, доктор медицинских наук, профессор, главный хирург МЗ РК, Баймаханов Болатбек Бимендеевич.

В рамках проводимых дней наши специалисты презентовали свои успехи и достижения, обменивались мнениями с представителями практического здравоохранения Узбекистана.

Доклад-презентацию на сессии высокого уровня на тему: «Достижение в области хирургических технологий Национального научного центра хирургии им. А.Н. Сызганова» представил Председатель правления Баймаханов Болатбек Бимендеевич.

С крупнейшими национальными центрами Республики Узбекистан, с такими как Республиканский специализированный научно-практический медицинский центр хирургии им. Академика В. Вахидова, Республиканский специализированный научно-практический медицинский центр кардиологии, Научно-исследовательский институт вирусологии и инфекционных болезней МЗ РУ, Республиканский перинатальный центр МЗ РУ, заключены меморандумы о сотрудничестве.

Во второй день специалистами ННЦХ им А.Н. Сызганова проведены ряд мастер-классов:

Профессором Баймахановым Б.Б. проведена уникальная операция «родственная трансплантация почки с лапароскопической мануально-ассистированной нефрэктомией».

Заведующим отделом рентгенэндоваскулярной хирургии и аритмологии, PhD Баимбетовым А.К. проведены ряд операций при тяжелых формах тахикардий «Катетерная радиочастотная аблация», также прочитаны лекции на тему «Современные технологии в интервенционном лечении нарушений ритма сердца».

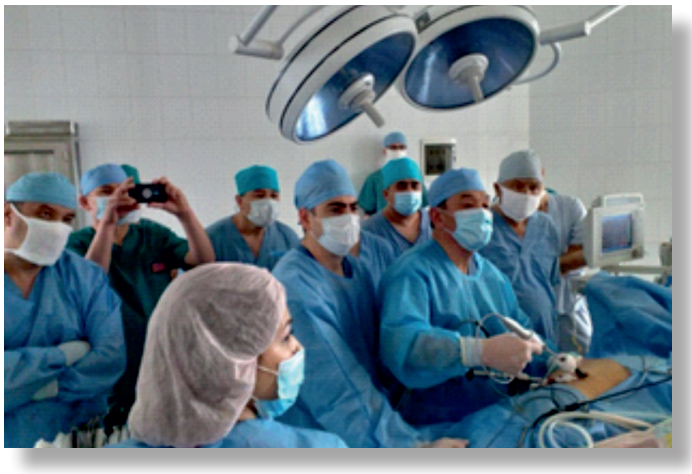
Заведующим отделом эндоскопии Абдрашевым Е.Б. эндоскопически удалены эпителиальные образования желудка трём пациентам.

Командой гинекологов проведены 10 миниинвазивных операций (гистерорезекто- и лапароскопические операции). Проведена одна операция лапароскопическая экстирпация матки, а также одна гибридная трёхуровневая реконструкция таза с установкой сетчатого протеза.

Также была презентована современная хирургическая технология ХЕОС.

В институте вирусологии профессором Ильясовой Б.С. прочитаны лекции на тему «Актуальные вопросы гепатологии». Осмотрены и обсуждены на совместном клиническом разборе ряд пациентов с патологией печени.

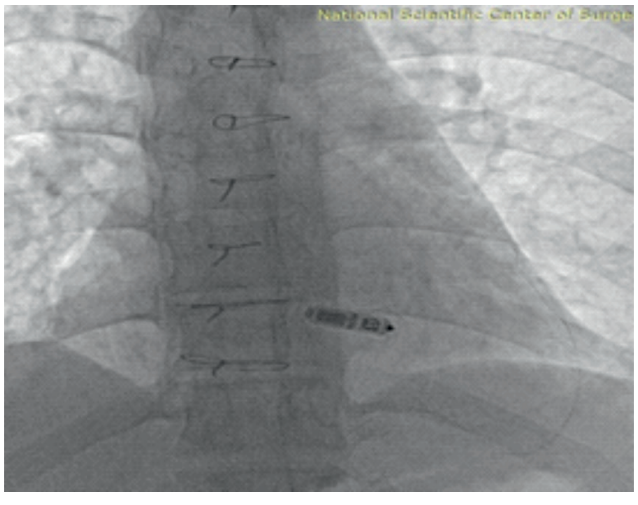
Проведённые мастер-классы вызвали огромный интерес у коллег из Узбекистана, тем самым проложили путь к дальнейшему плодотворному сотрудничеству.



МАСТЕР-КЛАСС НА ТЕМУ: «ЛУЧЕВАЯ ДИАГНОСТИКА В ПУЛЬМОНОЛОГИИ»

28 ноября 2019 г на базе Национального Научного Центра Хирургии им. А.Н. Сызганова прошел мастер-класс на тему «Лучевая диагностика в пульмонологии» с приглашением международного специалиста, а именно Сперанской Александры Анатольевны, д.м.н., профессора кафедры рентгенологии и радиационной медицины Первого Санкт-Петербургского государственного медицинского университета имени академика И. П. Павлова, г.Санкт-Петербург. Цель проведения мастер-класса – осветить вопросы современной лучевой диагностики заболеваний легких. На мастер-классе присутствовали врачи лучевой диагностики, пульмонологи и терапевты города Алматы и Алматинской области в количестве около пятидесяти слушателей. Были рассмотрены такие темы, как Лучевая диагностика фиброзирующих болезней легких, саркоидоза органов дыхания, острой интерстициальной пневмонии и бронхиолитов. После лекции была проведена дискуссия на представленные темы с разбором отдельных случаев.

МАСТЕР-КЛАСС НА ТЕМУ: «БЕСПРОВОДНОЙ КАРДИОСТИМУЛЯТОР «MICRA»



Имплантируемые электрокардиостимуляторы (ЭКС) прочно вошли в практику лечения нарушений ритма и проводимости сердца. Быстрое совершенствование этих устройств в последние годы привело к значительному расширению их использования и появлению новых возможностей электротерапии.

Обычные кардиостимуляторы имплантируются под ключицей – для этого делается разрез на коже, далее через подключичную вену к сердцу проталкиваются электроды, сам кардиостимулятор закрепляется под кожей. От операции остается шрам в подключичной области. Разработчики с каждым годом совершенствуют аппараты, уменьшая их размеры, для более безболезненной имплантации, ведь не секрет, что многие пациенты так и не способны привыкнуть к ощущению инородного устройства под кожей, у незначительного же количества людей возникает так называемая хроническая боль в области ложа аппарата, что приводит к реимплантации а порой даже к полному удалению стимулятора.

В рамках мастер-класса 23.12.2019г., в городе Алматы в стенах ННЦХ им. А.Н. Сызганова заведующим отделения интервенционной кардиологии и аритмологии Баимбетовым А.К. был успешно имплантирован четвертый в стране и первый в регионе беспроводной кардиостимулятор MicraTPS.

Участие в данном мастер классе приняли представители, а так же IT-специалисты компании Medtronic из ОАЭ, Турции и Казахстана. Слушатели в лице аритмологов, кардиологов, резидентов ННЦХ им. А.Н. Сызганова и специально приглашенный проктор заведующий отделением аритмологии ННКЦ д.м.н. Абдрахманов А.С.

Имплантация нового кардиостимулятора менее инвазивна, и в особенности подходит пациентам с проблематичным доступом через подключичную вену. Новый миниатюрный кардиостимулятор длиной 26 миллиметров и диаметром 6,7 миллиметров весит всего 1,75 граммов. Он проталкивается через бедренную вену через катетер в правый желудочек сердца. Когда достигнуто нужное место поблизости от верхушки сердца, кардиокапсула закрепляется в ткани сердца с помощью небольших крючков. Кардиокапсула объединила в себе батарейку, измеритель и водитель ритма сердца. Верхушка кардиостимулятора соприкасается со стенкой сердца, где подает необходимый электрический импульс. От имплантации остается лишь небольшой шрам на уровне бедра – от введения катетера. Безопасность и эффективность данной технологии подтверждена рядом рандомизированных контролируемых исследований. По сроку службы батареи MicraTPS ничем не уступают традиционным кардиостимуляторам, служат до 10 лет. Но в отличии от проводных стимуляторов, MicraTPS позволяет избежать всех осложнений связанных с электродами и ложем ЭКС.

Центр хирургии им.А.Н.Сызганова продолжает вносить свой вклад в научный прогресс, он один из немногих центров, который идет в ногу со временем, применяя и внедряя новейшие технологии в процесс лечения пациентов с тяжелыми формами нарушения ритма и проводимости сердца. В прошедшем мастер классе приняли участие аритмологи нашего центра, зав.отд. PhD Баимбетов А.К., Ергешов К.А., Байрамов Б.А., Бижанов К.А. и показали хорошее владение и понимание новейшей технологии имплантирования. С новыми беспроводными кардиостимуляторами MicraTPS, оперативные вмешательства займут меньше времени и станут более безопасными.

К 60-ЛЕТИЮ ПРОФЕССОРА НАРТАЙЛАКОВА МАЖИТА АХМЕТОВИЧА

Нартайлаков Мажит Ахметович родился 3 февраля 1960 года в Переволоцком районе Оренбургской области в с.Габдрафиково. Закончил среднюю школу и в 1977 году поступил, а в 1983 году закончил лечебный факультет Башкирского государственного медицинского института. В 1983-1985 гг. учился в клинической ординатуре при кафедре госпитальной хирургии БГМИ, в 1985-1987 гг. работал заведующим приемного отделения ГКБ № 6 г.Уфы. С 1987 года работает на кафедре общей хирургии Башкирского государственного медицинского университета: до 1991 года ассистентом, с 1991 г – доцентом, с 1996 г – профессором. С 1997 года, по настоящее время, является заведующим кафедрой общей хирургии БГМУ (с 2013 г – с курсом лучевой диагностики ИДПО, в 2019 г впервые среди вузов РФ открыт курс трансплантологии). С 2011 по 2015 годы работал проректором по лечебной и научной работе БГМУ.

В 1994 – 1997г.г. работал главным хирургом Министерства здравоохранения Республики Башкортостан. С 2015г. возглавляет НИИ новых медицинских технологий при клинике БГМУ. Профессор Нартайлаков М.А. является руководителем хирургической клиники Республиканской клинической больницы им. Г.Г. Куватова, а с 1995 года – руководителем Башкирского республиканского центра хирургической гепатологии.

В 2010 году, проф. Нартайлаков М.А. избран Почетным членом Ассоциации хирургов-гепатологов России и стран СНГ. Он член правления Ассоциации хирургов РБ, зам. председателя экспертной группы Минздрава РБ по аттестации специалистов хирургического профиля, член экспертной группы хирургического профиля Приволжского отделения Центральной аттестационной комиссии Минздрава РФ, зам. председателя Хирургического совета Минздрава РБ, председатель сертификационной комиссии БГМУ, член диссертационного совета Д 208.006.02 БГМУ по хирургии. Он член редколлегии журналов «Медицинский вестник Башкортостана» (г.Уфа), «Креативная хирургия и онкология» (г.Уфа), «Здравоохранение Башкортостана» (г.Уфа), электронного журнала «Вестник БГМУ» (г.Уфа).

По результатам научных исследований защищены: кандидатская (Казань, 1989) и докторская диссертации (Москва, 1995). По кафедре общей хирургии БГМУ, присвоены: ученое звание доцента (1992 г) и профессора (1997 г). Под руководством проф. Нартайлакова М.А. защищены 9 докторских и 46 кандидатских диссертаций. Является руководителем научной школы «Хирургическая гепатология» по Республике Башкортостан.

Им опубликовано 615 научных работ в журналах и сборниках трудов международного, федерального и республиканского уровней, в том числе, издано 13 монографий. Получено 28 патентов на изобретения и 51 удостоверение на рационализаторские предложения. Издано 22 методических рекомендаций, соавтор 2 справочников по хирургии.

Среди опубликованных научных работ Нартайлакова М.А. большой интерес специалистов России и стран СНГ вызвали: монография «Повторные ликворошунтирующие операции» (издана в Санкт-Петербурге в 1999 году в соавторстве с проф. Хачатрян В.А., проф. Сафиным Ш.М. и др.), мультимедийное учебное пособие «Общая хирургия» (выпущено в Уфе в 2005 году с грифом УМО по медицинскому и фармацевтическому образованию вузов России), учебное пособие «Общая хирургия» (издано в 2006 году в Ростове-на-Дону), монография «Хирургия печени и желчных путей» (издано в 2005 г в Уфе и в 2007 году в Ростове-на-Дону), признанной лучшей монографией 2005 года по итогам конкурса Ассоциации хирургов Республики Башкортостан. Огромный интерес вызвал изданный Нартайлаковым М.А. в 2011 году историко-художественный роман «Личный лекарь великого Тамерлана». В 2015 году она переиздана под названием «Лекарь великого эмира», с добавлением глав о пребывании Тамерлана в 1391 году на территориях, нынешней Республики Башкортостан, Республики Казахстан и Оренбургской области.

Профессор Нартайлаков М.А. является Заслуженным врачом Республики Башкортостан (1998 г) и Российской Федерации (2008г), Заслуженным деятелем науки Республики Башкортостан (2002 г), отличником здравоохранения (2002 г), член-корреспондент Российской академии естественных наук (с 2006 г).

Нартайлаков М.А. трижды награжден «Золотым скальпелем» за лучшую операцию года в Республике Башкортостан (1996, 2005 и 2013 гг), в 2010 году был награжден Благодарственным письмом Президента Республики Башкортостан. В 2012 году он награжден орденом Салавата Юлаева. Неоднократно награждался Почетными грамотами Минздрава Республики Башкортостан и Башкирского государственного медицинского университета, дипломами за лучшие операции и доклады по итогам ежегодных конкурсов Ассоциации хирургов Республики Башкортостан. Признан лучшим хирургом года Республики Башкортостан (2002 г). В 2006 году награжден знаком «За заслуги перед Чекмагушевским районом Республики Башкортостан».

Профессор Нартайлаков М.А. входил в группу разработчиков, принятой в 1995 г., Республиканской целевой программы «Развитие трансплантологии в Республике Башкортостан», благодаря которой в 1996 году сразу в двух учреждениях (Республиканская клиническая больница им. Г.Г.Куватова и Республиканская детская клиническая больница) начаты пересадки почек взрослым и детям. Оба центра функционируют по настоящее время, выполнено более 300 трупных и родственных трансплантаций почек. В 2013 году возглавил бригаду хирургов, впервые в Республике Башкортостан выполнивших трансплантацию печени. К настоящему времени выполнено 20 успешных пересадок печени.

Профессор Нартайлаков М.А. неоднократно выступал, с блестящими докладами на Международных конгрессах, по актуальным проблемам хирургии печени, поджелудочной железы, паразитарных кист печени, трансплантации печени (г.Москва, С.Петербург, Уфа, Ташкент, Алматы и др.) Лекции, мастер – классы, которые он читает и проводит привлекает не только студентов, аспирантов, но и широкий круг специалистов своей актуальностью, новизной, практической значимостью.

Нартайлаков М.А. совместно с профессором Ибадильдиным А.С. являются инициаторами заключения договора о международном сотрудничестве между Башкирским государственным медицинским университетом (г.Уфа) и Казахским Национальным медицинским университетом имени С.Д. Асфендиярова (г.Алматы), который был подписан в 2014 году.

Коллектив кафедры общей хирургии с курсами трансплантологии и лучевой диагностики ИДПО БГМУ желает юбиляру здоровья и дальнейших успехов!

Профессор Мустафин А.Х., г. Уфа.
Профессор Ибадильдин А.С.,г.Алматы.

К 60-ЛЕТИЮ СО ДНЯ РОЖДЕНИЯ ПРОФЕССОРА ЖАКИЕВА БАЗЫЛБЕКА САГИДОЛЛИЕВИЧА

Жакиев Базылбек Сагидоллиевич родился 27 ноября 2019 года. После окончания лечебного факультета Актюбинского государственного медицинского института (1982 год) и одногодичной интернатуры начал свою трудовую деятельность в качестве врача хирурга Урдинской центральной районной больнице Уральской области.

С 1986 по 1988 годы обучался в клинической ординатуре по хирургии на кафедре госпитальной хирургии АкГМИ. Во время обучения в клинической ординатуре прошел специализацию по ангиохирургии на базе сосудистого отделения Каз.НИИ К и ЭХ им А.Н.Сызганова. С 1988 – 1989 г.г. работал врачом хирургом в Актюбинской областной клинической больнице. В 1989 г. был избран по конкурсу ассистентом кафедры госпитальной хирургии Актюбинского государственного медицинского института, с 1998 г. - доцентом и с 2003 по 2004 г.г. заведующий кафедрой.. С 2004 по 2009 г. руководил кафедрой факультетской хирургии и с 2009 г заведует кафедрой хирургических болезней №2, которая с 2015 г переименована на кафедру хирургического профиля интернатуры и послевузовского обучения.

Профессор Жакиев Б.С. доброжелательный педагог, опытный лектор, читает лекции и ведет занятия свободно на двух языках, учебный материал излагает на достаточно высоком методическом уровне.

Практическую работу плодотворно совмещает с научной деятельностью. Успешно защитил диссертации в Научном Центре хирургии им. А.Н. Сызганова на соискание ученой степени кандидата медицинских наук в 1992 г. и доктора медицинских наук в 2003 г. В 1998 г утвержден в ученом звании доцента медицины, в 2008 г – профессора медицины.

Профессор Жакиев Б.С. владеет основами ораторского искусства, богатством лексики, умеет правильно использовать то богатство методов и приемов чтения лекций, которое накопили педагогика и психология.

Руководимый им коллектив кафедры помимо воспитательной и учебно-методической работы выполняет большую лечебно-профилактическую работу в пределах Западного региона РК. Он внес большой вклад в дело улучшения показателей хирургической службы, расширения диапазона хирургических вмешательств. Им внедрены в клиническую практику сложные операции на печени,поджелудочной железе,пищеводе, желчных путях и магистральных сосудах.

Профессор Жакиев Б.С., несмотря на свою высокую эрудированность и многолетний опыт практической работы, постоянно занимается над совершенствованием своего профессионального и педагогического мастерства.

В последние время активно занимается внедрением трансплантации внутренних органов (почки и печени). Неоднократно проходил стажировки в г.Минск, Республики Беларусь (2014) и г. Сеул, Южная Корея (2015). Им впервые в условиях Западного региона Казахстана выполнена трансплантация почки от трупного донора. Опубликовано 220 научных трудов, получено 12 патентов, изданы 4 методических рекомендаций, 2 учебных пособия и 4 монографии. Все предложенные им разработки внедрены в клиническую практику. Под его руководством защищены 2 докторских и 3 кандидатских диссертации.

С 2006 г является действительным членом международной организации «Ассоциации хирургов - гепатологов». С 2010 года - независимым экспертом в области качества медицинских услуг. С 2006 года внутренний аудитор системы менеджмента качества, с 2012 г возглавляет областное научное общества хирургов.

С 2003 по 2006 г.г. был руководителем государственной программы 009 «Профилактика и лечение гнойно-септических осложнений в абдоминальной хирургии» ГР 0103РК00255, который прошел международную экспертизу и оценен на 2,5 балла (высокий - 1 балл).

В 2006 г награжден «Алгыс хат» акима Актюбинской области. В 2009г был победителем конкурса «Үздік дәрігер» Актюбинской области, поэтому был награжден дипломом акима Актюбинской области. В 2006 г награжден знаком «Денсаулық сақтау ісінің үздігі». В 2014 г «Человек года Актюбинской области - 2014», в 2017 г «Человек года Уилского района Актюбинской области», также награжден медалью «Денсаулық ісіне қосқан үлесі үшін».

Профессор Жакиева Б.С. отличает дисциплинированность, трудолюбие, организаторская способность, требовательность к себе и окружающим. Пользуется заслуженным авторитетом среди коллег, сотрудников академии и медицинского центра.

Базылбек Сагидоллаевичич встречает свой юбилей в расцвете творческих сил, полных энергии и бодрости, продолжает активную хирургическую и педагогическую деятельность, всегда в готовности прийти на помощь коллегам.

Коллектив клиники хирургических болезней № 2 (госпиальной хирургии), редакция журнала «Вестник хирургии Казахстана» и ученики сердечно поздравляют юбиляра с 60-летием и желают крепкого здоровья, долгих лет жизни, семейного счастья, не иссекаемой энергии и творческого долголетия.

ТРЕБОВАНИЯ ДЛЯ АВТОРОВ ЖУРНАЛА «ВЕСТНИК ХИРУРГИИ КАЗАХСТАНА»

Уважаемые авторы!

С 1 апреля 2018 года все статьи на публикацию принимаются на государственном или русском языках с обязательным переводом всей статьи на английский язык. Статьи без версии на английском языке будут отклонены.

Также учитывая требования Консультативной Комиссией (CSAB) Scopus об интернационализации авторов и аудитории редколлегия журналов рекомендуют публиковать статьи в соавторстве с учеными дальнего и ближнего зарубежья.

В журнале публикуются научные статьи и заметки, экспресс-сообщения о результатах исследований в различных областях естественно-технических и общественных наук.

Решение о публикации принимается редакционной коллегией журнала после рецензирования, учитывая научную значимость и актуальность представленных материалов. Статьи, отклоненные редакционной коллегией, повторно не принимаются и не рассматриваются. Рукописи, оформленные не по правилам, возвращаются авторам без рассмотрения.

Рукопись направляется на отзыв члену редколлегии и одному из указанных рецензентов; в спорных случаях по усмотрению редколлегии привлекаются дополнительные рецензенты; на основании экспертных заключений редколлегия определяет дальнейшую судьбу рукописи: принятие к публикации в представленном виде, необходимость доработки или отклонение. В случае необходимости рукопись направляется авторам на доработку по замечаниям рецензентов и редакторов, после чего она повторно рецензируется, и редколлегия вновь решает вопрос о приемлемости рукописи для публикации. Переработанная рукопись должная быть возвращена в редакцию в течение месяца после получения авторами отзывов; в противном случае рукопись рассматривается как вновь поступившая. Рукопись, получившая недостаточно высокие оценки при рецензировании, отклоняется как не соответствующая уровню или профилю публикаций журнала.

Авторы несут ответственность за достоверность и значимость научных результатов и актуальность научного содержания работ. Не допускается **ПЛАГИАТ** – умышленно совершаемое физическим лицом незаконное использование чужого творческого труда, с доведением до других лиц ложных сведений о себе как о действительном авторе.

Редакция принимает на рассмотрение рукописи только на английском языке, присланные через официальный сайт журнала **www.vhk.kz.**

Материал статьи – абстракт на казахском, русском и английском языках, список литературы, рисунки, подписи к рисункам и таблицы, оформляется одним файлом; дополнительно каждый рисунок оформляется в виде отдельного файла. Если пересылаемый материал велик по объему, следует использовать программы для архивирования. Все страницы рукописи, в том числе таблицы, список литературы, рисунки и подписи к ним, следует пронумеровать.

Представленные для опубликования материалы должны удовлетворять следующим требованиям:

1. Содержать результаты оригинальных научных исследований по актуальным проблемам в области физики, математики, механики, информатики, биологии, медицины, геологии, химии, экологии, общественных и гуманитарных наук, ранее не опубликованные и не предназначенные к публикации в других изданиях. Статья сопровождается разрешением на опубликование от учреждения, в котором выполнено исследование.
2. Размер статьи 7-10 страниц (статьи обзорного характера – 15-20 стр.), включая аннотацию в начале статьи перед основным текстом, которая должна отражать цель работы, метод или методологию проведения работы, результаты работы, область применения результатов, выводы (**аннотация** не менее **20** предложений (150»300 слов) - (на английском языке) через 1 компьютерный интервал), таблицы, рисунки, список литературы (через 1 компьютерный интервал, размер шрифта – 14), напечатанных в редакторе Word, шрифтом Times New Roman, поля – верхнее и нижнее – 2 см, левое –3 см, правое –1,5 см. Количество рисунков – 5-10.

Структура должна соответствовать международной формуле IMRAD, где I – introduction (вступление), M – Methods (методы), R – Results (исследование), A – и, D – conclusion+ discussion (заключение, обсуждение результатов и выводы).

Название • Отображает суть работы • Краткое • Без аббревиатур.

Необходимо официально закрепить название организации на английском и сокращение

Резюме • Структурировано • Без аббревиатур • Передает структуру статьи – Зачем (актуальность) – Какими методами? – Что получено – Как это изменило картину знаний. Именно его читают в первую очередь, только хорошее резюме может привлечь внимание!

Вступление • Актуальность работы • Какая задача поставлена • Почему

Методы • Перечисление • Если известные - дать ссылку • Если модифицировали – указать как • Описывать так что б могли повторить • Статистика!

Результаты • Допускается не хронологическое, а логическое повествование • Основные, а не все что были сделаны •

Иллюстрируются минимально необходимыми сводными данными (исходные могут быть в дополнительных материалах)

Обсуждения • Не повторять результаты • Сопоставить полученные данные с имеющимися • Обсудить возможные причины и следствия

Функции списка литературы: • Аргументировать идею • Сопоставить с существующими аналогами • Обозначить место данного исследования • Избежать плагиата • Для журнала и ученого = признание • Часто указаны только собственные работы или очень старые (самоцитирование допускается только 10-15% от общего списка литературы) • Кочующие ошибки

Различайте • Ссылки • Список литературы • Библиография

Что могут цитировать • Книги, (монографии, главы) • Статьи научных журналов • Материалы конференций • Патенты • Диссертации • Неопубликованные данные • СМИ • Веб ресурсы (протоколы, веб странички) Источник должен быть надежным и легко доступным.

Статья начинается на английском языке. В начале, посередине страницы, идет название статьи прописными жирными буквами, название статьи должно быть коротким и емким, согласно проведенного анализа около 30-40 символов на английском языке.

Далее на следующей строчке – инициалы и фамилии авторов обычным жирным шрифтом, затем на следующей строчке – название организации(ий), в которой выполнена работа, город, страна, затем на новой строчке – адреса E-mail авторов. С красной строки идут ключевые слова **(Key words),** а на новой строчке – сама аннотация (**Abstract** – не менее **150** и более **300 слов**).

Далее, после отбивки одной строки, начинается на русском языке. В начале статьи вверху слева следует указать индекс **УДК, МРНТИ.**

Затем, посередине страницы, пишется: 1) название статьи; 2) авторы; 3) название организации; с красной строки – **Ключевые слова,** затем – **Аннотация** (оформление шрифтов, как на английском языке).

Отбиваем одну строку и начинается сама **статья.** Следом за статьей идет список **Литературы.** Ссылки на литературные источники даются цифрами в прямых скобках по мере упоминания (не менее 20).

Для каждой статьи обязателен DOI (Digital Object Identifier) - это цифровой идентификатор документа. DOI выполняет функцию гиперссылки, которая всегда помогает найти нужный документ, даже если сайт, где он находился ранее, был впоследствии изменен. Благодаря этому индексу поиск научной информации в Интернете стал проще и эффективнее. Каждое издание, журнал размещает на своих веб-страницах в интернете, как текущие, так и архивные номера, и материалы. Таким образом, в открытом доступе можно увидеть резюме, которые включают в себя название статьи, фамилию, имя, отчество автора, аннотацию и ключевые слова, место выполнения работы, а также выходные данные опубликованных статей (название журнала, год издания, том, номер, страница).

Список литературы оформляется следующим образом:

В ссылках на книги указывается ISBN (10- или 13-значный). Сокращаются названия только тех журналов, которые указаны: http://images.webofknowledge.com/WOK46/help/WOS/0-9_abrvjt.html.

Для всех ссылок на статьи, опубликованные в международных рецензируемых журналах следует указывать DOI (Digital Object Identifier). DOI указываются в PDF версии статьи и/или на основной интернет-странице статьи, также можно воспользоваться системой поиска CrossRef: http://www.crossref.org/guestquery/. Ниже приводятся примеры оформления ссылок:

Статья в международном журнале:

1. Campry TS, Anders T. (1987) SNAP receptors implicated in vesicle targeting and fusion, Environ Pollut, 43:195-207. DOI: 10.1016/0269-7491(87)90156-4 (in Eng)

Статья в русскоязычном журнале, не имеющая англоязычной версии:

2. Ivanova TV, Samoilova NF (2009) Electrochemical Energetics [Elektrohimicheskaya energetika] 9:188-189. (In Russian)

Книги:

Timrat TA (2008) Soil pollution: origins, monitoring and remediation, second edition. Springer, Germany. ISBN: 978-3-540-70777-6

Материалы конференции:

Monin S.A. (2012) Treatment techniques of oil-contaminated soil and water aquifers. Proceedings of International Conference on Water Resources and Arid Environment, Riyadh, Saudi Arabia. P.123.

Патенты:

Barin AB, Mukamedzhan NT (2000) A method for determination of 1,1-dimethylhydrazine and nitrosodimethylamine [Metodopredeleniya 1,1-dimetilgidrazina initrosodimetilamina]. Preliminary Patent of the Republic of Kazakhstan [Predvaritelnyi patent Respubliki Kazakhstan]. (In Russian)

Стандарты, ГОСТы:

RMG 61-2003. Indexes of accuracy, precision, validity of the methods of quantitative chemical analysis, methods of evaluation [GSI.Pokazatelitochnosti, pravilnosti, retsizionnosti metodik kolichestvennogo himicheskogo analiza. Metodyiotsenki]. Moscow, Russia, 2003. (In Russian)

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В конце статьи дается резюме на казахском языке. Оформляется аналогично русскому варианту. Посередине страницы пишется: 1) название статьи; 2) авторы; 3) название организации; с красной строки – **Өзекті сөздер,** после – **Аннотация.**

Последняя страница подписывается всеми авторами, ставится дата.

3. Статьи публикуются только на английском языке.

4. В случае переработки статьи по просьбе редакционной коллегии журнала датой поступления считается дата получения редакцией окончательного варианта. Если статья отклонена, редакция сохраняет за собой право не вести дискуссию по мотивам отклонения.

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