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MORPHOLOGICAL FEATURES OF HEART DEFECTS, INTERVENTRICULAR BARRIER DEFECTS AND ATRIAL BARRIER DEFECTS IN THE TERRITORY OF BUKHARA REGION

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БУХОРО ВИЛОЯТИ ҲУДУДИДА ЮРАК ТУҒМА НУҚСОНИ ҚОРИНЧАЛАРАРО ТЎСИҚ ВА БЎЛМАЧАЛАРАРО ТЎСИҚ НУҚСОНЛАРИНИНГ МОРФОЛОГИК ХОС ЖИҲАТЛАРИ

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МОРФОЛОГИЧЕСКИЕ ОСОБЕННОСТИ ПОРОКОВ СЕРДЦА, ДЕФЕКТОВ МЕЖЖЕЛУДОЧКОГО БАРЬЕРА И ДЕФЕКТОВ МЕЖПРЕДСЕРДНОГО БАРЬЕРА НА ТЕРРИТОРИИ БУХАРСКОЙ ОБЛАСТИ

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Abstract: *The work identifies the morphological aspects of myocardial hypertrophy, chaotic anastomoses of the small capillary network and dystrophic changes in cardiomyocytes due to defects in the tissues of the interventricular and interlobular barriers. This is characterized by a chaotic arrangement of sparse fibrous structures between myocardial bundles in places of changes in angioarchitecture and the detection of cells rich in fatty inclusions due to the functional enhancement of the high impulse conductivity of atypical cardiomyocytes.*

Key words: *morphology, congenital heart disease, interventricular barrier defect, atrial barrier defect, myocardium, hypertrophy, sclerosis.*

Аннотация: *Ушбу тадқиқот ишимизда қоринчалараро тўсиқ ва бўлмачалараро тўсиқ тўқималаридаги нуқсонга боғлиқ миокарднинг гипертрофияси морфологик жиҳатлари, майда капиллярлар тўрининг ўзаро хаотик анастамози ва кардиомиоцитларнинг дистрофик ўзгаришлари аниқланди. Бу эса, шу соҳадаги ангиоархитектоникани ўзгаришга учраган соҳалардаги миокард тутамлари оралиғида сийрак толали тузилмаларнинг хаотик жойлашиши ва атипик кардиомиоцитларнинг юқори импульс ўтказувчанлигининг функционал кучайиши оқибатида ёғли киритмаларга бой бўлган ҳужайраларнинг аниқланиши билан характерланади.*

Калит сўзлар: *морфология, юрак туғма нуқсони, қоринчалараро тўсиқ нуқсони, бўлмачалараро тўсиқ нуқсони, миокард, гипертрофия, склероз.*

Резюме: *В настоящей работе выявлены морфологические аспекты гипертрофии миокарда, хаотических анастомозов мелкой капиллярной сети и дистрофических изменений кардиомиоцитов вследствие дефектов тканей межжелудочкового и междолькового барьеров. Для этого характерно хаотичное расположение разреженных фиброзных структур между пучками миокарда в местах изменений ангиоархитектоники и обнаружение клеток, богатых жировыми включениями, за счет функционального усиления высокой импульсной проводимости атипичных кардиомиоцитов.*

Ключевые слова: *морфология, врожденный порок сердца, дефект межжелудочкового барьера, дефект межпредсердного барьера, миокард, гипертрофия, склероз.*

Relevance of the topic: Heart is one of the most complicated congenital defects. Depending on the localization of the interventricular defect and the diameter of the hole, the morphological adaptation of the heart defect develops differently. In most newborns, in cases where the diameter of the hole is larger than 0.5 cm in the upper defect of the interventricular barrier, the development of blue pus and the rapid development of venous damping within the small blood circulation continue. This is manifested in the form of the occurrence of a secondary infectious factor in both lung tissues and the appearance of complicated pneumonias, and in the period of complete diagnosis, pneumonia develops as the main disease in infants, and is manifested in the form of an indication against the practice of correcting a congenital heart defect. In 20% of babies born with congenital heart defects in the world, the death rate of babies due to this type of pathology is 40-72%.

Among the congenital heart defects in Uzbekistan, interventricular barrier defect and interlobular barrier defect make up 50.8% of the total number of defects, 11.2% of which are fatal.

The urgency of the problem is to study the prevalence and morphological characteristics of babies with interventricular barrier defects and interventricular barrier defects in the Bukhara region of the Republic of Uzbekistan. An average of 26-35 cases of congenital heart defects diagnosed per year in Bukhara region are ventricular septal defects and septal septal defects.

The purpose of the research is to study and analyze the incidence of congenital heart defects, anatomo-histological and morphometric changes in Bukhara region.

Material and methods: the clinical anamnestic data of heart tissue and disease history taken from autopsy of 65 babies who died of congenital heart defect in Bukhara Region Pathological Anatomy Expert Bureau are analyzed. Sections taken from heart tissue by the morphological method are frozen in 10% buffered formalin for 72 hours. Then after rinsing in wastewater for 1 hour, it is dehydrated in ascending alcohols (70,80,90,100%). The slices are then frozen in paraffin and poured into cassettes. Using a microtome, 5-7 μm thick sections are taken, deparaffinized in xylene, and stained with hematoxylin and eosin. The obtained results are viewed under a light microscope, micrographs are taken and morphometrically analyzed.

Research results and their discussion: The regional distribution of congenital heart defects is different in the territory of Uzbekistan, and the morphological aspects of interventricular septal defect and interlobular septal defect (combined 30-48%) were studied in the Bukhara region. Changes in histioarchitectonic topography of myocardium and membranous tissue, which is one of the specific aspects of congenital heart defect or combined heart defect, were studied in Bukhara region.

According to the analysis of data on the morphological changes of the right ventricle of the heart, the defect of the interventricular barrier is characterized by the formation of sparse fibrous connective tissue between the bundles of cardiomyocytes of the right ventricle, and the focal appearance of cells with fatty and hyaline droplet dystrophy in the myocytes of this area. From the morphofunctional point of view, this indicator is characterized by branched areas between hypertrophied bundles of cardiomyocytes and interstitial tumors and lymphocytes in poorly formed focus. while the angioarchitectonics of the coronary vessels in this area does not change, a layer rich in sparse fibers with perivascular sclerosis is determined around the vessel wall. At the same time, focal histiocytosis and active foci of fibroblasts are detected in the interval. Group hypertrophy of the right ventricular myocardium, cardiomyocytes was mostly detected in the area of the anterior wall of the ventricle. Including branched hypertrophy of cardiomyocytes, large hyperchromic, transversely distributed, clearly delineated cells are detected in the 200X field of view. The number of large cardiomyocytes was 220-255 in the 200x field of view. Compared to the control group, it was found to be 2.25 times (up to 100-125), the size increased by 2.5 times. Vessels between cardiomyocytes: capillaries and

small-caliber blood vessels 1.75 times less than the control group. These changes mean that compensatory mechanisms in the right heart area are clearly developed. At the same time, it was found that in the cytoplasm of atypical cardiomyocytes (pacemaker cells) located along the perimeter of the right ventricle, a large number of pale pink inclusions (glycogen) were found. It was found that the lymphatic vessels located in the interval of cardiomyocytes have a cavernous appearance of different widths (Fig. 1).

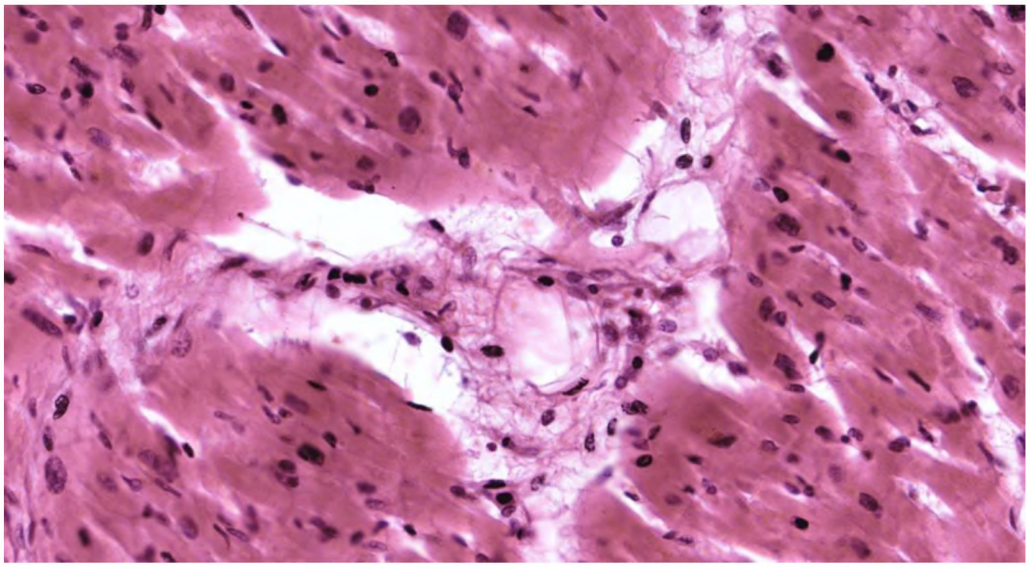

Fig. 1. **Myocardium obtained from ventricular septal defect, right ventricular dorsal horn and lateral surface horn. Cavernous dilated lymphatic vessels (1), tufted appearance of large hyperchromic cardiomyocytes (2), uneven interstitial swellings are detected in the interval (3). Paint G.E. The size is 20x10.**

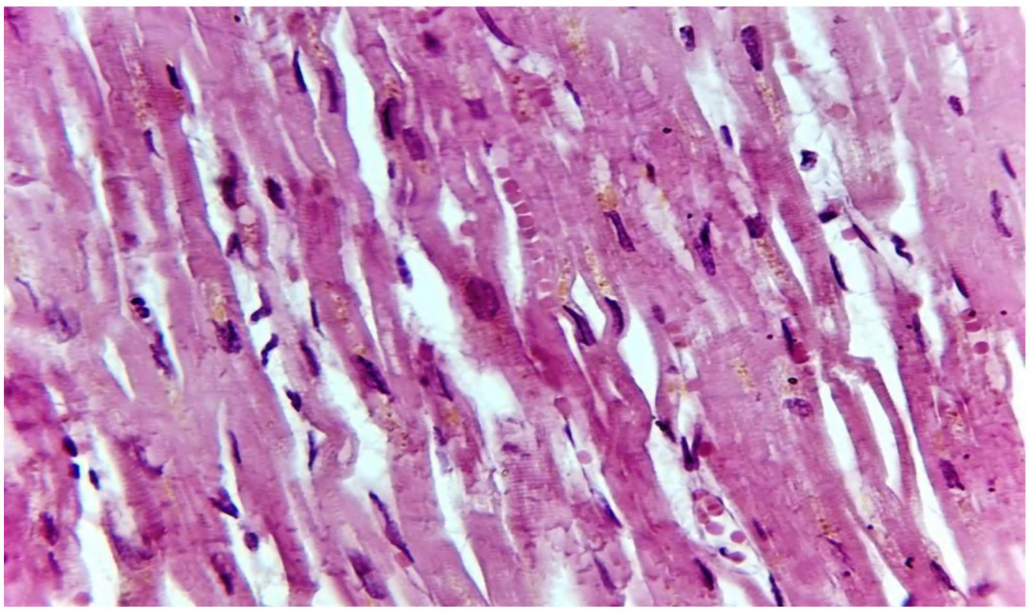

Fig. 2. **Myocardium obtained from ventricular septal defect, right ventricular dorsal horn and lateral surface horn. Sludge phenomenon in most capillaries (1), tufted appearance of large hyperchromic cardiomyocytes (2), uneven interstitial swellings in the interval are determined (3). Paint G.E. The size is 40x10.**

Due to hypertrophic changes in most cardiomyocytes of the right ventricular myocardium with defect of the interventricular barrier, some changes in the histioarchitectonics of most capillaries are observed. This, in turn, violates the laws of hemodynamics, causes the blood circulation in the capillaries to be partially disrupted and the permeability of the capillary wall to increase. As a result, focal plasmorrhagia causes interstitial edema, discontinuities or dilated foci between the interstitial discs that submerge cardiomyocytes (Fig. 2), derailing synchronous contractions. At the same time, it creates the phenomenon of sludge in the expanded capillaries. This, depending on the duration of the process, leads to the activation of fibroblasts in these areas and the increase of sparse fibrous structures.

As a result, it leads to atrophic change of bundles of cardiomyocytes that have not undergone hypertrophy and have low functional activity. As a result, heart contractions are clinically morphologically characterized by conditions for the development of arrhythmic contractions. It is characterized by the creation of conditions for the development of necrobiotic processes in cardiomyocytes with a focus due to the formation of microticin by sludged erythrocytes in the capillaries (Fig. 3).

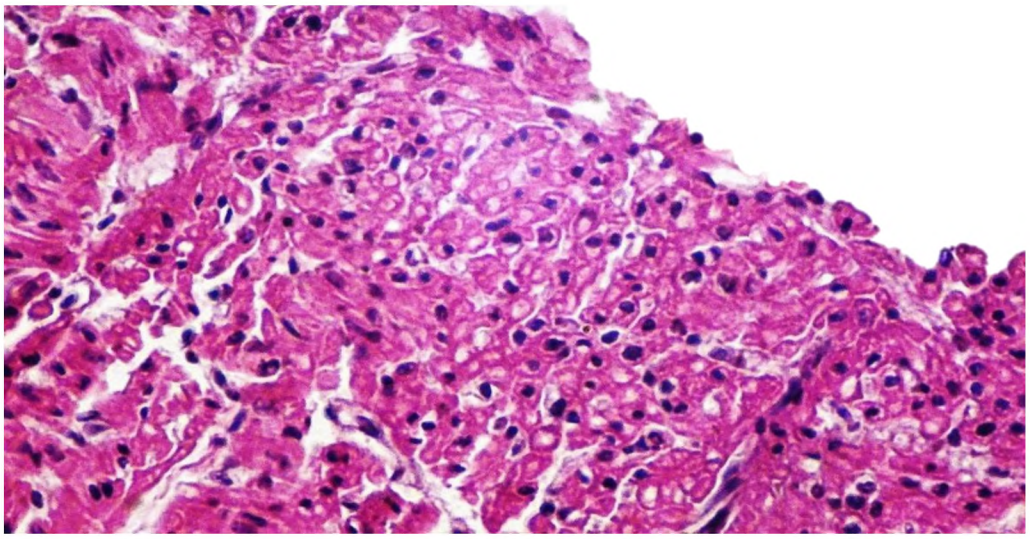

Fig. 3. **Interventricular septal defect. In most cardiomyocytes, fatty dystrophy has medium and small droplet-like foci (1), smooth interstitial edema in the subendocardial area (2), cytoplasm of morphofunctionally active cardiomyocytes is dark pink in color (3). Paint G.E. The size is 40x10.**

In the cytoplasm of cardiomyocytes in the right ventricular papillary muscles, the development of focal dystrophies in the form of various droplets continues, leading to sharp hypertrophy of morphofunctionally active cardiomyocytes, and macroscopically, the appearance of rough fibrous surfaces on the papillary muscle surfaces. As a result, atrophic changes in cardiomyocytes continue with the development of endocardial thickening and fibroelastosis foci on the cardiac endocardial surfaces (Fig. 3).

In particular, the branches of the right ventricle close to the lower left ventricle develop with the appearance of various granular basophilic inclusions in the endocardium, foci of fibroelastosis and the cytoplasm of Purkin cells. This, in turn, continues with the development of interstitial edema around the foci of fibroelastosis, scarring processes that penetrate the myocardium (Fig.4). Macroscopically, it continues with the appearance of foci characterized by the detection of uneven non-smooth surfaces on the surface of the lower branch of the right ventricle and facing the area of the interventricular septum.

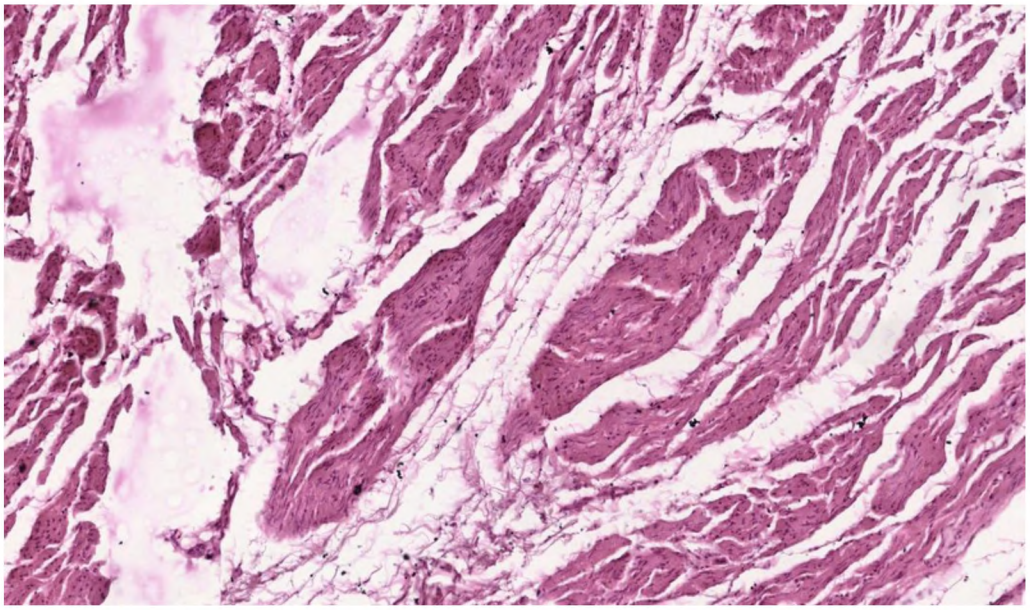

Fig. 4. **Interventricular septal defect. The focus of mysclerosis (1), interstitial swellings (2), destructive changes in fibrous structures are determined (3). Paint G.E. The size is 40x10.**

Sudden changes do not develop in the left ventricle, on the contrary, most of the cardiomyocytes are kept uniform in size, in the network of capillaries, they are characterized by a relatively low development of interstitial tumors. Most endocardially located cardiomyocytes have the same tuft structure and histioarchitectonics, and fatty dystrophy cardiomyocytes are almost undetectable (Fig.5), which means an orderly arrangement of cardiomyocytes of the same morphofunctional size.

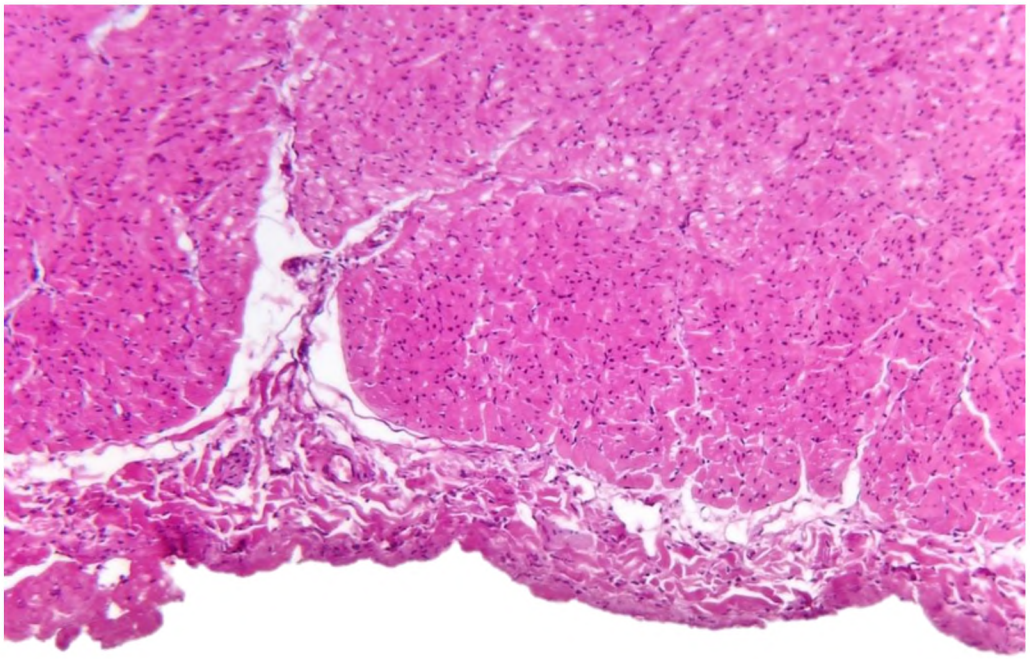

Fig. 5. **Interventricular septal defect. Right ventricular subvalvular area. Focus of fibroelastosis (1), intermediate swellings (2), destructive changes in fibrous structures are determined (3). Hydropic dystrophy of Purkin cells (4). Paint G.E. The size is 40x10.**

In the cardiomyocytes of the subpericardial branches, the same changes are detected, mostly, the changes are functionally active foci of the cardiomyocytes located near the subvalvular branch: hypertrophied cardiomyocytes, uneven interstitial edema, sludge phenomenon is detected in the capillaries. The root cause of these changes is the thickened endocardium in the periva subvalvular branches, which is affected by fibroeastosis, due to advanced hydropic dystrophy in the majority of Purkin cells, which indicates that asynchronous contractions of cardiomyocytes are taking place. At the same time, it is determined that the development of sparse fibrous structures between cardiomyocytes and the occurrence of sclerotic changes around small-caliber vessels in these areas.

Thus, the characteristic features of morphological changes occurring in congenital heart defects, focal hypertrophy of cardiomyocytes, sclerotic changes around blood vessels, interstitial edema and foci of fibroelastosis are determined. The most visible changes include focal thickening of the endocardium, group atrophic changes of subendocardial cardiomyocytes, lipomatous foci in the pericardium, and medium and small droplet fatty dystrophic changes in the cardiomyocytes of the right ventricle. These changes are of the combined types of congenital heart defects. These changes have a different appearance in different areas of the region (front of the ventricle, lower and interventricular septum) according to the localization of the congenital heart defects. It was found that the main part of most cardiomyodestructive changes is in the right ventricle in the blue type of heart defects from the clinical morphological point of view of most congenital heart defects. These changes continue with the development of chronic venous congestion within the larger circulation. As a result, the heart ends with the rapid development of right ventricular failure.

Conclusion. In the combined type of interventricular barrier defect and intercompartmental barrier defect, there are changes in the bundle structure of cardiomyocytes in myocardial tissue, detection of hypertrophy and atrophic changed foci, interstitial edematous changes, and the development of poorly formed lymphocyte infiltration. sclerotic changes are detected in the perimeter of blood vessels. At the moment, the morphological adaptation of the interventricular barrier defect and the morphofunctional deficiency of decompensated cardiomyocytes directly depend on the localization of the defect and the diameter of the hole, and it was found in our scientific work that it manifests itself in the form of right myocardial failure in the pathologies of the intercompartmental barrier defect and the interventricular barrier defect, which are continued and combined with injuries of the bullet defect type.

Literature.

1. Желев В.А., Барновская С.В., Михалев Е.В., Филлипов Г.П., Серебров В.Ю., Ермоленко С.П., Попова Ю.Ю. Клинико-биохимические маркеры поражения миокарда у недоношенных новорожденных // Бюллетень сибирской медицины. – 2007. - №4. – С. 86-90.

2. Koestenberger M., Avian A., Ravekes W. Reference values of the right ventricular outflow tract (RVOT) proximal diameter in 665 healthy children and calculation of z-score values // Int J Cardiol. – 2006. – Vol. 169, №6. – P. 99-101.

3. Эргашбаева Д.А., Ташбаев О.С., Хусанова Х.А., Хакимов Ш.К., Пазилжанова М.П., Солиева М.О. Кардиоинтервалографическая оценка ранней неонатальной адаптации новорожденных, родившихся от женщин с преэклампсией // Акушерство и гинекология. – 2008. - №2. – С. 19-21.

4. Abbot N.J. RonnbackL., Hansson E.Asrocyte-endothelial interactions at the blood-brain barrier // Nature Rev. Neurosci. - 2006. - Vol. 7. - P. 41.

5. Alehan F., Ozkutlu S., Alehan D. Echocardiographic assessment of left and right ventricular diastolic functions in children with dilated cardiomyopathy // Turk J Pediatr.– Vol. 40, №3. – P. 337-346.

6. Alehan F.K., Ozkutlu S., Alehan D. Effects of respiration on left ventricular diastolic function in healthy children // Eur Heart J.– Vol. 17, №3.– P. 453-456.

7. Chao C.P., ZaleskiC.G., Patton A.C. Neonatal Hypoxic–ischemic encephalopathy: multimodality imaging findings // Radio Graphics. - 2006. -Vol. 26. - P. 159–172.