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Research Article

Features of compensatory and adaptive reactions of the vascular bed of the bladder of elderly and senile men with prostatic hyperplasia

Igor S. Shormanov, Sergey V. Kulikov, Andrey S. Solovyov
Yaroslavl State Medical University, Yaroslavl, Russia

BACKGROUND: Bladder outlet obstruction caused by prostatic hyperplasia eventually leads to detrusor working hypertrophy, and its development is impossible without synchronous remodeling of its vascular bed. Taking into account the age-associated nature of the disease, bladder transformation occurs following involutive structural changes of both the organ and its vascular system due to natural aging. To date, morphological features of compensatory reactions of the vascular bed, taking into account background age-related changes in the bladder wall, is not fully disclosed.

AIM: To investigate the structural transformations of the bladder and its vascular bed in elderly and senile persons with prostatic hyperplasia and to determine the significance of vascular restructuring in providing compensatory detrusor hypertrophy.

MATERIALS AND METHODS: Autopsy material from 35 men aged 60–80 years who died from diseases not related to urological and cardiovascular pathology was examined, as well as those from 25 men of the same age who had prostatic hyperplasia without signs of bladder decompensation. The control group included 15 men aged 20–30 years who died as a result of injuries. A complex of histological, morphometric and immunohistochemical methods was used

RESULTS: In patients with prostatic hyperplasia, there is a violation of the histoarchitectonics of the bladder, which is expressed in the development of focal detrusor hypertrophy, layering on the existing involutive changes, which are characterized by the atrophy of muscle fibers, sclerosis of the intermuscular stroma, defragmentation of the elastic frame, and neurodegenerative processes. A similar state of the detrusor is caused by sclerosis and hyalinosis of small arteries and arterioles, hypertrophy and hyperelastosis of large arteries associated with arterial hypertension, combined with atherosclerosis of extraorganic arteries. Moreover, a complex is formed in the vascular basin of the bladder that can regulate blood transport in conditions of increasing chronic ischemia and provide a normal blood supply to the preserved parts of the detrusor that can undergo hypertrophy with an increase in functional impairment. These structures include intimate muscles, muscular elastic sphincters, polypoid cushions of the conti arteries, muscle "couplings," muscle rollers, and vein valves.

CONCLUSIONS: Structural changes in the bladder are characterized by a combination of focal detrusor atrophy resulting from angiosclerosis and development of hypertrophy of preserved areas with an increase in functional load. The hypertrophic potential of the detrusor is attributed to vascular regulatory structures.

Keywords: prostatic hyperplasia; bladder outlet obstruction; bladder; blood vessels; compensation; age-related involution.

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ECO•VECTOR

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Научная статья

Особенности компенсаторно-адаптационных реакций сосудистого русла мочевого пузыря мужчин пожилого и старческого возраста в условиях гиперплазии предстательной железы

И.С. Шорманов, С.В. Куликов, А.С. Соловьев
Ярославский государственный медицинский университет, Ярославль, Россия

Актуальность. Инфравезикальная обструкция, обусловленная гиперплазией предстательной железы, со временем приводит к рабочей гипертрофии детрузора, развитие которой невозможно без синхронного ремоделирования его сосудистого русла. Учитывая возраст-ассоциированную природу заболевания, трансформация мочевого пузыря происходит на фоне инволютивных структурных изменений как самого органа, так и его сосудистой системы, развивающихся в процессе естественного старения. На сегодняшний день морфологические особенности компенсаторных реакций сосудистого русла с учетом фоновых возрастных изменений в стенке мочевого пузыря остаются до конца не раскрытыми.

Цель — изучение структурных преобразований мочевого пузыря и его сосудистого русла у лиц пожилого и старческого возраста при гиперплазии предстательной железы, а также определение значения сосудистой перестройки в обеспечении компенсаторной гипертрофии детрузора.

Материалы и методы. Исследован аутопсийный материал от 35 мужчин 60–80 лет, умерших от заболеваний, не относящихся к урологической и сердечно-сосудистой патологии, а также от 25 мужчин того же возраста, имевших гиперплазию предстательной железы без признаков декомпенсации мочевого пузыря. Контрольную серию материала составили 15 мужчин в возрасте 20–30 лет, погибшие в результате травм. Использован комплекс гистологических, морфометрических и иммуногистохимических методик.

Результаты и обсуждение. У пациентов с гиперплазией предстательной железы происходит нарушение гистоархитектоники мочевого пузыря, что выражается в развитии очаговой гипертрофии детрузора, наслаивающейся на имеющиеся инволютивные изменения, которые характеризуются атрофией мышечных волокон, склерозом межмышечной стромы, дефрагментацией эластического каркаса и нейродегенеративными процессами. Подобное состояние детрузора связано с нарушением ангиоархитектоники в виде склероза и гиалиноза мелких артерий и артериол, гипертрофии и гиперэластоза крупных артерий, возникающих при артериальной гипертензии в сочетании с атеросклерозом внеорганных артерий. При этом в сосудистом бассейне мочевого пузыря формируется комплекс образований, регулирующих транспорт крови в условиях нарастающей хронической ишемии, обеспечивающих нормальное кровоснабжение сохраненных участков детрузора, способных подвергаться гипертрофии при повышении функциональной нагрузки. К таким структурам относятся интимальная мускулатура, мышечно-эластические сфинктеры и полиповидные подушки Конти артерий, а также мышечные «муфты», мышечные валики и клапаны вен.

Заключение. Структурные изменения мочевого пузыря характеризуются сочетанием очаговой атрофии детрузора, возникающей в результате ангиосклероза, и развитием гипертрофии сохраненных участков при повышении на него функциональной нагрузки. Гипертрофические потенции детрузора обеспечиваются согласованной работой целого комплекса сосудистых регуляторных структур.

Ключевые слова: гиперплазия предстательной железы; инфравезикальная обструкция; мочевой пузырь; сосуды; компенсация; возрастная инволюция.

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ЭКО•ВЕКТОР

BACKGROUND

Prostatic hyperplasia (PH) is one of the most common diseases in older and senile men [1]. Over time, its natural evolution results in the formation of compensatory detrusor hypertrophy [2, 3]. The development of work hypertrophy in PH allows the prevention for some time the development of urinary stasis and ascending infection of the upper urinary tract [4]. Compensatory changes in the muscular layer of the bladder are associated with preexisting involutive changes that are pathogenetically related to natural aging [5–7]. However, the morphogenetic aspects of the detrusor compensatory rearrangement and the nature and severity of age-related and senile changes remain largely undiscovered [8, 9]. Specifically, no comprehensive information is available on the effect of structural remodeling of the vascular bed of the bladder in the presence of infravesical obstruction on the hypertrophic potentials of detrusor smooth muscles [10–12]. The data obtained would have not only important scientific and fundamental but also practical value, contributing to the implementation of new approaches to the treatment of patients with PH.

This study aimed to analyze the structural transformations of the bladder and its vascular bed in older and senile patients with PH and determine the significance of vascular restructuring in ensuring compensatory detrusor hypertrophy.

MATERIALS AND METHODS

In this study, segments of the bladder wall were collected from 35 men aged 60–80 years, who died from diseases not related to urological and cardiovascular pathology. Series 2 of fragments of this organ was obtained at autopsy in 25 men of the same age with Prostatic hyperplasia (PH) without clinical and morphological signs of bladder decompensation. All tissue samples were obtained in the Department of Morbid Anatomy of the Yaroslavl Regional Clinical Hospital. The control group consisted of materials collected in the Thanatological Department of the Yaroslavl Regional Office of the Chief Medical Examiner from 15 men aged 20–30 years, who died by violence. In all of the above group, from various sections of the bladder (anterior, posterior, side walls, and bottom), segments of the wall were cut out for the entire thickness, from the mucous membrane to the adventitial membrane, including paravesical tissue. The sectional material was fixed in 10% neutral formalin and embedded in paraffin. Histological sections, not more than 5 μm thick, were stained with hematoxylin and eosin, according to Masson for collagen fibers using brilliant green and Hart's fuchselin for elastic fibers. An immunohistochemical study was performed on paraffin sections using murine monoclonal antibodies CD34, S100 (Cell Marque, USA), and α-SMA (DAKO, USA). The preparations were evaluated semiquantitatively in 10 fields of view (×200).

A morphometric study of the vascular bed of the bladder was performed according to a previously developed technique [13, 14]. Accordingly, all intraorgan arteries of the control group and PH group, depending on the outer diameter, were divided into four groups, namely, large (⩾125 μm), medium (51–124 μm), small arteries (21–50 μm), and arterioles (⩽20 μm). Venous collectors, depending on the caliber, were divided into large (⩾190 μm), medium (110–189 μm), small (51–109 μm) veins, and venules (⩽50 μm). In arteries of various calibers, the tunica media thickness was measured, and in veins, the wall thickness was measured in microns. In large arteries, the thickness of the tunica intima (intima) was measured in microns. In the mucous membrane of the bladder, the thickness of the urothelium basement membrane was determined in microns. In addition, a stereometric study was performed to determine the specific area of the detrusor structural components (muscle fibers, intermuscular stroma, and vessels), using a special ocular insert with 100 equidistant points applied to it. The digital material was processed with Student's *t*-test using the Statistica program. The distribution of the digital data corresponded to the normal one. Data were considered significant if the error probability did not exceed 5% ($p < 0.05$).

RESULTS

The transitional epithelium of the urinary bladder mucosa in older and senile men, regardless of the presence or absence of PH, was hyperplastic because of the proliferation of basal and parabasal cells with the formation of acanthotic strands submerging in the connective tissue of the lamina propria, often forming von Brunn's nests. In some areas of the mucous membrane, the epithelium became thinner or underwent alternative changes, such as hydropic degeneration manifested by the formation of light small vacuoles in the cell cytoplasm, necrobiosis, and severe desquamation. The basal membrane of the epithelium was thickened by two times ($p = 0.001$) and had a slightly eosinophilic, uniform, and homogeneous appearance, which indicated the development of hyalinosis. In the lamina propria and submucosa, the proliferation of coarse fibrous connective tissue was detected, as well as a plethora of veins, stromal edema, and pronounced mononuclear inflammatory infiltration represented by lymphocytes, plasma cells, macrophages, and fibroblasts, indicating the development of chronic cystitis. In both groups of samples from older and senile men, extraorganic arteries belonging to vessels of the muscular-elastic type, located in the paravesical tissue or adventitia, had uneven wall thickening and formation

of fibrous plaques, often narrowing the lumen of the vessels up to 25%, compared with controls. In large- and medium-sized muscle-type intraorganic arteries, wall thickness increased because of the hypertrophy of the smooth myocytes of the tunica media (media) with splitting of the internal elastic membrane into several laminae (Fig. 1*a*), indicating the development of hyperelastosis. A pronounced expression of α-smooth muscle actin (α-SMA) was detected in the muscular membrane of the arteries of the indicated caliber compared with similar vessels of the control group. In addition to hypertrophy of the media and hyperelastosis, as markers of arterial hypertension, in the tunica intima (intima) of large arteries, a layer of oblique longitudinal smooth muscles was clearly visible, located along the entire perimeter of the vessel, which was often completely replaced by coarse fibrous connective tissue, narrowing the vessel lumen.

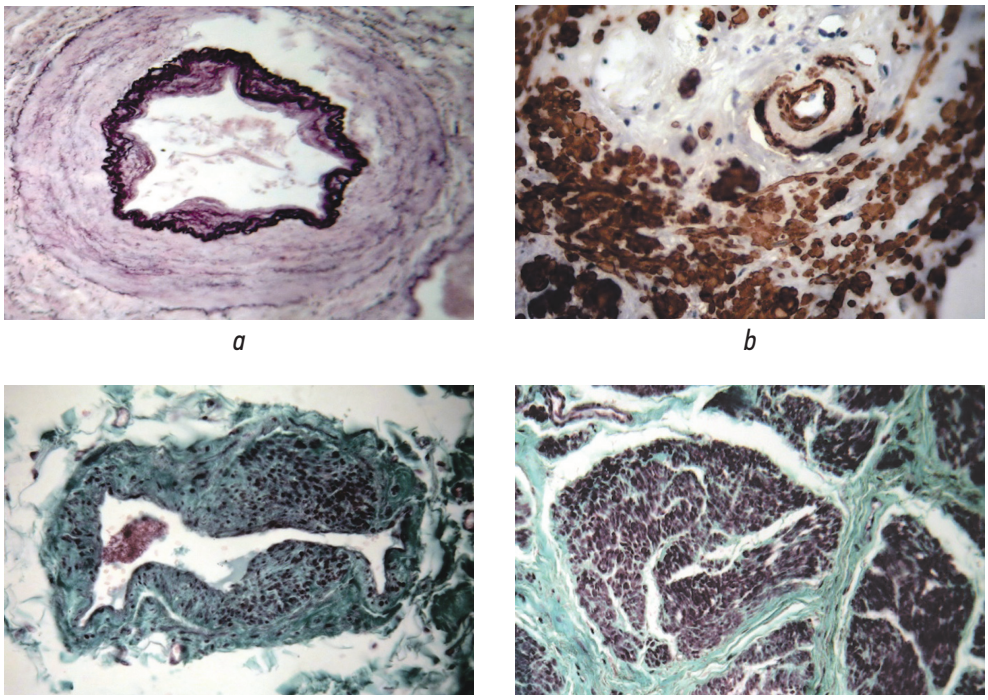
According to morphometric data, the intimal thickness caused by sclerosis increased by five times ($p = 0.001$) compared with the control. Small arteries and arterioles in both preparations were subjected to hyalinosis; as a result, the wall was replaced by an eosinophilic homogeneous structureless mass with pronounced narrowing of the lumen, loss of all layers, and significant decrease in α-SMA expression (Fig. 1*b*).

Pronounced structural changes were also detected in the venous system of the bladder in older and senile men. Thus, in large extraorganic veins located in the paravesical tissue and adventitia, significant uneven thickening of the walls was determined to be caused by hyperplasia and hypertrophy of smooth myocytes, which made them similar to large arteries (Fig. 1*c*). However, compared with the arterial branches, they lacked well-formed layers, a relatively strong media, and an internal elastic membrane. Coarse fibrous connective tissue proliferated in the walls of some veins.

A morphometric study enabled us to objectify histological changes in the arteries, manifested by hypertonicity and lumen narrowing. Specifically, the thickness of the media of large intraorgan arteries in older and senile men with PH increased by 5.2 times ($p = 0.001$), medium arteries by 2 times ($p = 0.001$), small arteries increased by 1.4 times ($p = 0.05$), and arterioles by 1.8 times ($p = 0.05$) compared with the control. The wall thickness increased by 4.6 times in large veins, 2 times in medium veins ($p = 0.001$), 1.8 times in small ones ($p = 0.001$), and 1.6 times in venules ($p = 0.05$) compared with the control. Vascular wall thickening was due to hypertrophy of smooth muscles.

In older and senile men, individual muscle bundles of the detrusor were subjected to thinning, coarse fibrous connective tissue proliferated between them (Fig. 1*b, d*), and the elastic fibers that enveloped the myocytes were defragmented. A stereometric study showed that the specific area of the detrusor muscle fibers decreased by 2 times ($p = 0.001$) and that of the stroma increased by 1.5 times ($p = 0.05$) compared with the control; however, the area occupied by the vessels did not change significantly. By contrast, in the PH group, changes were detected, which were caused by the increased functional load on the detrusor with the development of hypertrophy. Specifically, against the areas of atrophy and sclerosis of the detrusor smooth muscles, areas with powerful muscle fibers collected in large bundles, increased counts of cells and nuclei, and hyperchromatosis were determined. In hypertrophied smooth muscles, a well-developed elastic framework was detected in the form of fibers braiding both bundles and each individual fiber, and the α-SMA expression (Fig. 1*b*) was higher than not only to the group without PH but also with the control group. In addition, in the intermuscular stroma of the hypertrophied detrusor, the amount of capillaries increased, and a high expression of CD34 was detected. This was indicated in the data obtained using stereometry. Thus, the specific area of detrusor muscle fibers increased by 1.3 times ($p = 0.05$), that of vessels increased by 1.2 times ($p = 0.05$), and that of the stroma decreased by 1.1 times ($p = 0.05$) compared with that of the control group. The area occupied by the vessels did not change significantly. In the bladder wall, the expression of S100 in nerve fibers and their endings (Fig. 2*a*) was weak compared with that in the control group. In our opinion, weak staining intensity indicates degenerative changes in the neural conductors of the detrusor.

The main morphological difference in the vascular reorganization of the bladder in the PH group compared with the non-PH and control groups was the presence of specific myogenic regulatory structures in the arteries and veins. In the arterial bed, in contrast to the control and non-PH groups, intimal muscle bundles, muscular-elastic sphincters, and Conti's polypoid cushions appeared, which are important in ensuring blood transport to the most functioning, hypertrophied areas of the detrusor. Thus, the intimal muscles, found mainly in medium and small arteries and arterioles, consisted of smooth myocytes located in the inner coat of the vessel in the form of bundles of rounded or irregular shape protruding into its lumen (Fig. 2*b*). In the places of their formation, the internal elastic membrane split into two leaflets, which covered them from above and below. As described above intimal muscles were also found in the closing arteries of persons without PH, where the circular oblique longitudinal layer was located along the entire circumference of the vessel, often subject to sclerosis. Muscle-elastic sphincters are located in the initial sections of the lateral branches of the middle arteries. They formed petals from bundles of smooth myocytes, and in the area of their location, the inner elastic membrane was split into separate petals. Conti's polypoid cushions looked like rounded

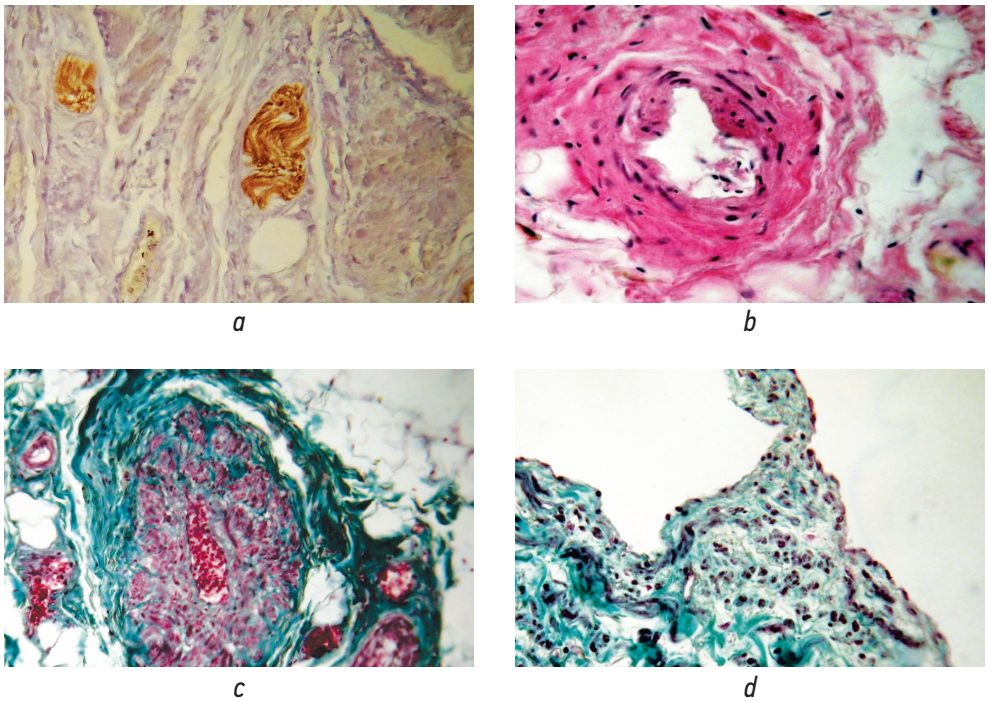


a *b*

c *d*

Fig. 1. Structural changes in the bladder wall of elderly and senile men with prostatic hyperplasia: *a*, fuchselin staining according to Hart (magnification ×200); *b*, indirect immunoperixidase method with antibodies to α-SMA (magnification ×200); *c*, *d*, Masson stain using brilliant green (magnification ×200 — *c*, ×160 — *d*)

Рис. 1. Структурные изменения стенки мочевого пузыря у мужчин пожилого и старческого возраста при гиперплазии предстательной железы: *a* — окраска фукселином по Харту (увел. ×200); *b* — непрямой иммунопероксидазный метод с антителами к α-SMA (увел. ×200); *c*, *d* — окраска по Массону с использованием бриллиантового зеленого (увел. ×200 — *c*, увел. ×160 — *d*)



a *b*

c *d*

Fig. 2. Structural changes in the bladder wall of elderly and senile men with prostatic hyperplasia: *a*, indirect immunoperixidase method with antibodies to S100; *b*, hematoxylin and eosin stain; *c*, *d*, Masson stain using brilliant green. Magnification ×200

Рис. 2. Структурные изменения стенки мочевого пузыря у мужчин пожилого и старческого возраста при гиперплазии предстательной железы: *a* — непрямой иммунопериксидазный метод с антителами к S100; *b* — окраска гематоксилином и эозином; *c*, *d* — окраска по Массону с использованием бриллиантового зеленого. Увел. ×200

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muscular formations, resembling polyps located on a thin pedicle or wide base of the vessel wall. They are formed as a result of invagination of various layers of the vessel wall into the lumen, including protrusion of the intima only, intima and media, intima, media, and adventitia. In the myocytes of the intimal muscles, sphincters, and cushions, the expression of α-SMA was high.

In the veins and arteries of this group, special muscular, regulatory formations were determined which were not registered in the control group or non-PH group. They were represented by muscular "couplings" of extraorganic veins, muscular embankments, and valves of intraorganic veins. Venous "couplings" were powerful muscle layers with different spatial arrangements of smooth myocytes and narrow layers of collagen and elastic fibers between them, located around the entire circumference of the outer membrane of large extraorganic veins (Fig. 2*c*). These formations covered the vessel segmentally, forming intercepts, through certain segments of its length. The contraction of the smooth muscles of such muscle interception provided an impulse for the movement of venous blood to the heart, and relaxation was accompanied by the opposite effect, i.e., blood deposition. The ridges in large and medium intraorgan veins proceeded from the inner membrane in the form of flattened formations protruding into the lumen, having a wide base and consisting of smooth myocytes. The venous valves looked like long club-shaped or polypoid structures on a thin pedicle, formed as a result of invagination of the inner coat of the vessel. At the base of the protrusion-shaped valve, clusters of smooth myocytes were detected (Fig. 2*d*), and the pedicle and body contained predominantly connective tissue. Valvular structures, unlike other venous formations, were also found in the control group; however, their number increased sharply in the PH group. Functionally, the valves prevent blood regurgitation or blood deposition.

DISCUSSION

The study results revealed structural changes in the bladder of older and senile men without PH, affecting all its membranes and the vascular bed, including large extraorganic arteries. Changes in the organ itself are characterized by hydropic degeneration of the urothelium, chronic inflammation of the mucous membrane, focal atrophy of muscle fibers, sclerosis of the intermuscular stroma, and destructive changes in the elastic framework, which are associated with a vascular disorder and neuronal trophism. Dyscirculatory disorders leading to chronic ischemia of the bladder are caused by progressive atherosclerotic changes in the extraorganic arteries of the muscular-elastic type, as well as remodeling of the vascular system, which is induced by a long course of arterial hypertension. It is manifested as the development of hypertonicity, hypertrophy of the media, hyperelastosis of large and medium-sized intraorganic arteries, and hyalinosis of small arteries and arterioles, which is the result of recurrent plasmorrhagia during hypertensive crises. The closing arteries, which have a well-defined longitudinal layer of smooth muscles in the intima, contribute to the adaptation of circulatory disorders in the muscular membrane of the bladder. The development of chronic detrusor ischemia is accompanied by atrophic–sclerotic changes in some areas of smooth muscles while maintaining others, which does not contradict the principle of "functional heterogeneity in the work of structures of the same name," according to which any organ does not use all structures simultaneously to perform a function, and it includes in operation all available structural resources and reserves that undergo hypertrophy with an increase in the functional load [15–17]. Our studies have shown that PH in older and senile men leads to working compensatory detrusor hypertrophy, which is manifested as hyperplasia and hypertrophy of previously unaltered muscle fibers, and in their intermuscular stroma, the degree of capillarization increases, which is necessary to ensure an actively functioning tissue. To maintain adequate blood supply to such areas of the detrusor in the arterial bed of the bladder, a complex of structures is formed, which can regulate hemodynamics under conditions of impaired blood circulation, i.e., against persistent chronic ischemia. Such formations include bundles of intimal muscles, muscular-elastic sphincters, and Conti's polypoid cushions. An increase in the tone of the oblique intimal muscles in combination with a contraction of the smooth muscles of the media leads to shortening, twisting, and corrugation of the vessel with the formation of cushion-shaped structures protruding into its lumen. This is accompanied by a decrease or complete blockade of blood flow both along them and along the distally located lateral branches [18]. The functional role of the sphincters is reduced to blood flow regulation from the main artery to the lateral branch. A polypoid cushion causes complete blood flow cessation along the main trunk and lateral branches extending from it [19]. The appearance of these formations is based on the migration of smooth myocytes into the intima through "windows" in the internal elastic membrane, which is stimulated by hemodynamic disorders [20]. Thus, due to the active work of regulatory structures in PH against impaired hemocirculation, blood transport through the intraorganic arteries of the bladder is normalized, depending on the functional needs of the muscle layer. Such an active "switching" by these structures ensures normal blood circulation in favor of preserved, hypertrophied detrusor areas to the detriment of areas that have undergone irreversible atrophic–sclerotic processes during involution. Regulatory formations in the veins include muscle "couplings," muscular

embankments, and valves. Muscular "couplings" covering the vessel segmentally and circularly and embankments that form in the tunica intima, as a result of contraction, prevent venous stasis and hypoxia and, if necessary, deposit blood when relaxing. The valvular apparatus of the veins had the same functional role, namely, prevention of venous blood regurgitation and its promotion to the heart or, conversely, its deposition [21]. Thus, vascular restructuring is an important link in providing compensatory detrusor hypertrophy in PH. Moreover, the duration and degree of effective work of the detrusor will largely depend on the extent of the efficacy of vascular compensation [22]. The development of subsequent decompensation is due to progressive background processes in the bladder.

CONCLUSION

In PH, the detrusor undergoes focal hypertrophy formed in the presence of preexisting involutive, atrophic–sclerotic, and neurodegenerative changes that have developed following dyscirculatory disorders. The working hypertrophy of the detrusor in PH is provided by the whole complex of regulatory myogenic formations in arteries and veins, which consists in the redistribution of blood transport to the detrusor areas with the highest functional activity.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work.

Competing interests. The authors declare that they have no competing interests.

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AUTHORS' INFO

Igor S. Shormanov, Dr. Sci. (Med.), Professor,
Head of the Department of Urology with Nephrology;
ORCID: https://orcid.org/0000-0002-2062-0421;
eLibrary SPIN: 7772-8420; Scopus: 6507085029;
e-mail: i-s-shormanov@yandex.ru

Sergey V. Kulikov, Dr. Sci. (Med), Associate Professor,
Head of the Department of Pathological Anatomy;
ORCID: https://orcid.org/0000-0002-3331-8555;
eLibrary SPIN: 8894-6060; e-mail: kulikov268@yandex.ru

***Andrey S. Solovyov,** Cand. Sci. (Med),
Assistant of the Department of Urology with Nephrology;
address: 5, Revolutsionnaya st., Yaroslavl, 150000, Russia;
ORCID: https://orcid.org/0000-0001-5612-3227;
eLibrary SPIN: 8198-2122; e-mail: a-s-soloviev89@yandex.ru

* Corresponding author / Автор, ответственный за переписку

ОБ АВТОРАХ

Игорь Сергеевич Шорманов, д-р мед. наук, профессор,
заведующий кафедрой урологии с нефрологией;
ORCID: https://orcid.org/0000-0002-2062-0421;
eLibrary SPIN: 7772-8420; Scopus: 6507085029;
e-mail: i-s-shormanov@yandex.ru

Сергей Владимирович Куликов, д-р мед. наук, доцент,
заведующий кафедрой патологической анатомии;
ORCID: https://orcid.org/0000-0002-3331-8555;
eLibrary SPIN: 8894-6060; e-mail: kulikov268@yandex.ru

***Андрей Сергеевич Соловьев,** канд. мед. наук,
ассистент кафедры урологии с нефрологией;
адрес: Россия, 150000, Ярославль, ул. Революционная, д. 5;
ORCID: https://orcid.org/0000-0001-5612-3227;
e-mail: a-s-soloviev89@yandex.ru