

Morphogenesis and Morphology of Stromal Structures in Glandular Hyperplasia of the Prostate Gland

Khamraev O. A.¹, Israilov R. I.², Kosimhojiev M. I.¹

¹Andijan State Medical Institute, Andijan, Uzbekistan

²Republican Center of Pathological Anatomy, Tashkent, Uzbekistan

Abstract The main of this study The aim was to clarify the morphogenesis and pathomorphological changes of stroma tissue structures in prostate glandular hyperplasia. It has been confirmed that the disease of prostatic hyperplasia consists of 5 morphologically stages. In the first stage of the disease, it is observed that 2 or 3 glandular acini are hyperplastic separately, forming a single unicentric micro-nodule, and the connective tissue between them is roughened and increased. Starting from the second period of the disease, lympho-plasmacytic cells first increase in the stroma, then after the proliferation of histiocytic cells, the inflammatory infiltrate densely surrounds the gland cells and forms a membrane. Exacerbation of the inflammatory process in the stroma of the gland is acidification of the tissue secretion in terms of biochemical composition, on the other hand, it is the development of an autoimmune process against the cells of the gland cells and stroma tissue structures.

Keywords Prostate, Hyperplasia, Stroma, Morphogenesis, Morphology

1. Introduction

When discussing morphological changes in the disease of safe hyperplasia of the prostate, one must first call the processes correctly and with specific terms. An increase in the volume of organelles, tissue, cell or intracellular, is called hypertrophy in General Pathology. The numerical reproduction of the organ and tissue component is called hyperplasia. In the study of the skirts of the morphogenesis of hyperplastic processes in prostate-safe hyperplasia, it will be necessary to take into account the presence of parenchyma consisting of glandular structures in the tissue of this member and fibromuscular stroma between them [1,2,3]. Anatomically, the size of the organ increases from the appearance of nodular structures in the structure of the gland, and the development of this process can consist of several periods. At the beginning of the process, nodes appear from 2-3 densely packed glands. Around these glands, the myofibrillary stroma develops into coarsened connective tissue, giving rise to a distinctive fibrosis curtain. Later in the development of the process, the glandular ayinar structures hyperplasia and increase dramatically, and microscopically the appearance of numerous nodules coincides with period II. Around these glandular nodes, stroma tissue structures proliferate, creating connective tissue curtains. The outbreak of the pathological process produces new additional specific

proliferative centers around the glandular nodes and is a morphological appearance of period III. An active increase in asinar foci leads to the dimming of secretions from them and the formation of retentive cysts in asinuses, and this is the IV-period of the process. The epithelium covering the asinuses that have become cysts is the V-Day of the process if it atrophy and becomes smaller in shape. Prostate safe hyperplasia paralleling developmental morphogenetic periods, stroma structures also proliferate, proliferate, and outperform parenchyma in volume when it comes to the last period, and this is known as fibrosed adenosis [4,5,6,7,8]. Asynar yachies are fragmented as a result of proliferation and proliferation of fibromuscular structures in the gland stroma.

The main goal of this study was considered to clarify the morphogenesis and pathomorphological changes of stroma tissue structures in glandular hyperplasia of the prostate.

2. Material and Methods

As an object of examination, the men's prostate in the safe hyperplasia of the prostate, which was examined in the direction of biopsy diagnostics of the Republican Center for pathological anatomy, the prostate fragments obtained in the type surgery were hardened for 48 hours in 10% solution of formalin for histological examination purposes. After 4 hours of washing in running water, the concentrate was dehydrated in increasing alcohols and paraffin was poured and bricks were prepared. 4-5 µm histological incisions

were made from paraffin bricks and painted in the hematoxylin-eosin and Van-Gison methods. The preparations were examined under a light microscope and the desired areas were photographed.

3. Results and Discussion

In the i-period of safe glandular hyperplasia of the prostate, it is found that the 2nd or 3rd glandular asinuses hyperplasia separately, giving rise to a single unisentric micro node. This gland begins to proliferate, proliferating fibrocytic and myocytic cells, as well as fibrous structures, containing stroma between and around the yachts. The specificity of proliferation of these tissue structures is that fibrocytes become fibroblasts, with increased proliferative activity causing their nuclei to both hyperchromasize and increase in size. Fibroblasts have produced large amounts of fibrous structures and are found to have produced tufts of varying sizes from joining with each other, and they wrap the glandular yachts densely. Affected by the proliferation process of fibroblasts, smooth muscle cells between them also proliferate and proliferate (Figure 1), and are found to join fibrocytic tissue Tufts to form combinatorial fibromuscular tissue and wrap the glandular sacs. In the conclusion, it can be shown that from a violation of hormone metabolism, initially, the glandular structures of the prostate parenchyma undergo hyperplasia, and next, from the fact that hormones also affect the stroma tissue structures, the fibrocytic and myositar cells in them proliferate, fibromuscular tissue structures with a coarse structure are formed from the production of fibrous structures.

Later in the development of the glandular hyperplasia process of the prostate, the glandular asinar structures hyperplasia and increase dramatically, and microscopically the appearance of numerous nodules is period II. From this period, it is observed that a chronic inflammatory infiltrate appears in the structure of the stroma structures between and around the hyperplasiated glandular structures. The cause of the development of the inflammatory process can be a change and sourness of the biochemical composition of substances synthesized in hyperplasic gland yachts. It is also the entry of a bacterial infection into the asinuses of the gland, by ascending from their excretory tubes. During this period of hyperplasia, the structure of stroma structures initially produced inflammatory infiltrate from the proliferation of lympho-plasmocytic cells, while next it is found that the histocytic cells of the stroma also proliferated by proliferation (Figure 2). This, lympho-histocytic cell inflammatory infiltrate is observed surrounding the glandular yachts directly adjacent to them. As a result, it is found that the glandular yachts expanded and deformed from the discrete accumulation they produced, and that some evolved into retentative cysts.

Among the few glandular yachts that have hyperplasia during the III period of glandular hyperplasia of the prostate, there is an increase in chronic inflammatory infiltrate and

connective tissue growth (figure 3). As a result, the glandular yachts are compressed, atrophy, and morphologically develop into glands of various large and small shapes. The cause of the exacerbation of the inflammatory process is sourness in terms of the biochemical composition of the secretion, which is synthesized in hyperplasiated gland cells. Gland cells and stroma, on the other hand, are the development of the autoimmune process in relation to tissue structures. In this case, the fact that low-specific fibroblasts in the stroma are actively divided and increased, differentiated fibroblasts begin to actively produce fibrous structures, glycosaminglicans and an intermediate. On the second hand, there is the emergence of relatively large histiocytes from monocytes (Figure 4), phagocytosis of foreign bodies by them, cooperation with humoral immune cells, synthesis of lysozyme and interferon and synthesis of a factor that harms polynuclear leukocytes. This type of cell proliferation and biochemical alteration of the intermediate cause causes connective tissue structures to dysplasia. As a result, anomalous collagen is synthesized, they undergo degradation, collagen fiber composition and structure are disrupted, elastic fibers are formed and added with collagen, the destruction of fibrous structures is caused by the development of an autoimmune process.

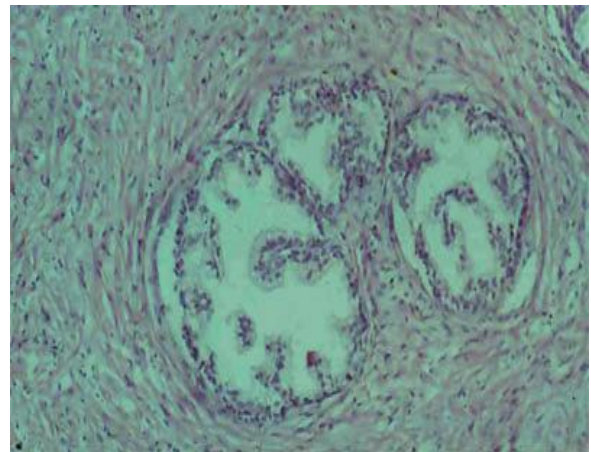


Figure 1. Hyperplasia of individual glandular yachts in the prostate and surrounded by Stroma tissue. Paint: G-E. Size: 10x40

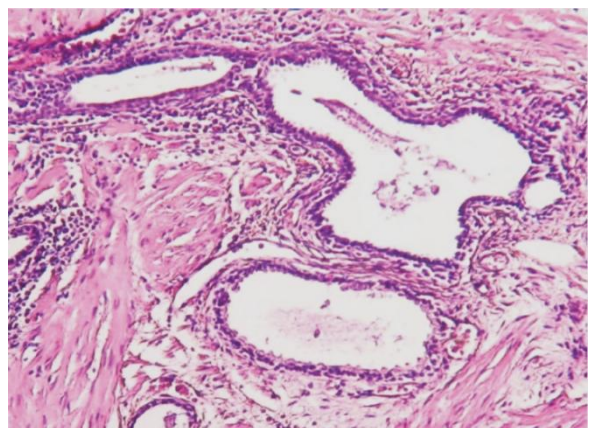


Figure 2. The appearance of inflammatory infiltrate in the hyperplasia and stroma of the asinus of the prostate gland. Paint: G-E. Size: 10x40

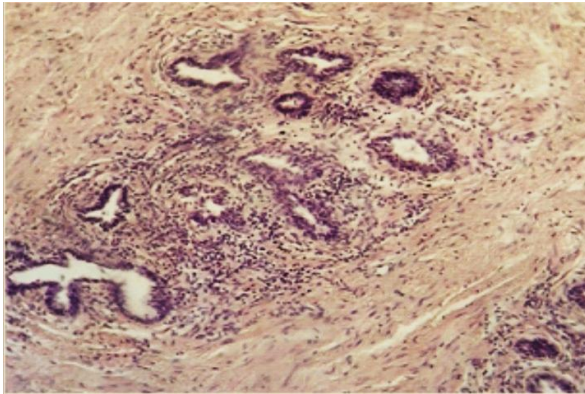


Figure 3. Increased inflammatory process in stroma in glandular hyperplasia of the prostate. Paint: G-E. Size: 10x40.

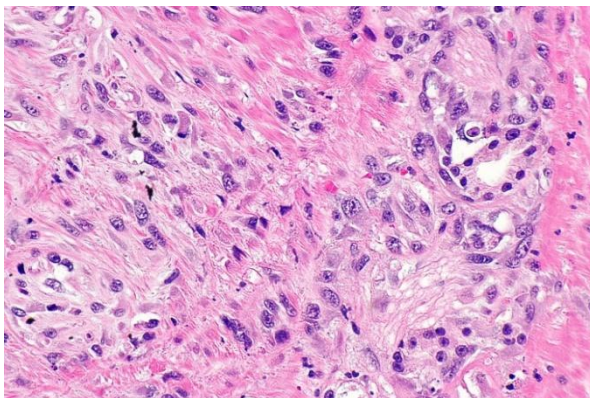


Figure 4. Activation of prostate stroma cells and fibrous structures. Paint: G-E. Size: 10x40

Active in glandular hyperplasia of the prostate, the secrete produced in proliferated glandular yachts is dimmed as a result of difficulty exiting through the ducts, dilating the ASINs and turning them into cysts. The epithelium lining the interior of cyst-reduced asinuses is found to have metaplasia and dysplasia to varying degrees. It is often observed that epithelial cells atrophy, diminish their shape, and become tiny prismatic epithelium. In other areas, one-layer prismatic epithelium is found to be metaplasialized to multi-row epithelium. In the structure of the stroma between asinuses, which has become a glandular-cystic structure, it is observed that a coarse-fibrous fibromatosis stroma appears, mainly from the growth and reproduction of connective tissue structures.

In glandular hyperplasia of the prostate, we used a special dye Kreyberg method for the purpose of identifying mucopolysaccharides, sour glycosaminglicans, keratin and Musin, mucoid, which are contained in asinus structures and stroma tissue structures. In doing so, it was found that mucopolysaccharides in interstitial tissue were blue pherose when treated with alcian Moss, sour glycosaminglicans were darker brown, and musins and mucoid substances contained in mucus in the lining of the gland asinuses were havo-colored. Therefore, during the IV - and V-periods of glandular hyperplasia of the prostate, an increase in the amount of sour mucopolysaccharides is observed in the

intermediate tissue, the synthesis of Musin and mucoid-rich mucus in the space of the glandular asinuses.

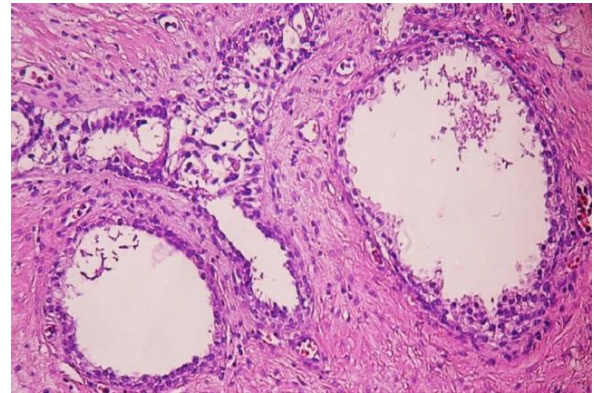


Figure 5. The transformation of glandular asinuses into cysts, the growth of coarse fibrous fibromatous tissue in the intermediate tissue. Paint: G-E. Size: 10x40

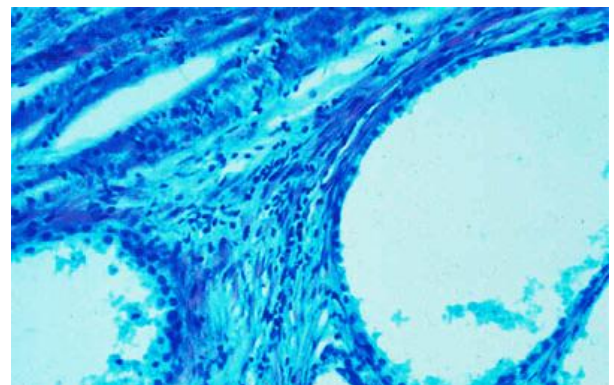


Figure 6. An increase in sour mucopolysaccharides in the gland stroma, the appearance of Musin and mucoid in the cavity of ksita. Paint: Kreyberg method. Size: 10x40

4. Conclusions

It has been confirmed that prostate glandular hyperplasia consists of 5 periods that develop morphologically one after another.

In the I-period of the disease, it is observed that the 2nd or 3rd glandular asinuses hyperplasia separately, giving rise to one unisentric micro node, the connective tissue between which becomes rough and increases.

Since the II-period of the disease, stroma initially increases lympho-plasmocytic cells, then after the addition of proliferation of histiocytic cells, inflammatory infiltrate envelops the glandular cells densely, creating a veil.

The advance of the inflammatory process in the gland stroma is the sourness of the tissue secretion in terms of its biochemical composition, but on the other hand it is the development of the autoimmune process in relation to the cells of the glandular cells and the tissue structures of the stroma.

In glandular hyperplasia of the prostate, the low-specific fibroblasts contained in the activation of the stroma increase actively, differentiated fibroblasts produce fibrous structures,

glycosaminoglycans and intermediate, on the other hand, monocytes become large histiocytes, phagocytosis of foreign bodies, co-operationalization with humoral immune cells, lysozyme and interferon synthesis, and synthesis of polynuclear leukocyte deacetylation factor synthesis. As a result, anomalous collagen is synthesized, they undergo degradation, collagen fiber composition and structure are disrupted, elastic fibers are formed and added with collagen, the destruction of fibrous structures is caused by the development of an autoimmune process.

REFERENCES

-
- [1] Filipenko P.S., Malooka Y.S. The role of connective tissue dysplasia in the formation of mitral valve prolapse. *Wedge. Med.*, M, 2006; 84(12): 13-19. (in Russian)
 - [2] Klemenov A.V. Undifferentiated connective tissue dysplasia. M., 2005. 136 p. (in Russian)
 - [3] Zemtsovsky E.V. Dysplastic phenotypes. Dysplastic heart. St. Petersburg: publishing house "Olga", 2007. 80 p. (in Russian)
 - [4] Makolkin V.I., Podzolkov V.I., Rodionov A.V., Sheyanov M.V., Samoylenko V.V., Napalkov D.A. Variety of clinical symptoms of connective tissue dysplasia. *Ter. Archive* 2004; 76(11): 77-80. (in Russian)
 - [5] Kleimenov A.V. Extracardial manifestations of undifferentiated connective tissue dysplasia. *Wedge. Med.*, M, 2003; 81(10): 4-7. (in Russian)
 - [6] Kleimenov A.V., Martynov V.L., Torgushina I.S. Insufficiency of the bauginium flap as a visceral manifestation of undifferentiated connective tissue dysplasia. *Ter. Archive* 2003; 75(4): 44-46. (in Russian)
 - [7] Zweers M.C., Dean W.B., van Kuppevelt T.H., Bristow J., Schalkwijk J. Elastic fiber abnormalities in hypermobility type Ehlers–Danlos syndrome patients with tenascin–X mutations. *Clin Genet.* 2005; 67(4): 330–334.
 - [8] Cabral W.A., Chang W., Barnes A.M., Weis M., Scott M.A., Leikin S., Makareeva E., Kuznetsova N.V., Rosenbaum K.N., Tiffit C.J., Bulas D.I., Kozma C., Smith P.A., Eyre D.R., Marini J.C. Prolyl 3–hydroxylase 1 deficiency causes a recessive metabolic bone disorder resembling lethal/severe osteogenesis imperfecta. *Nat Genet.* 2007; 39(3): 359–65 Epub 2007 Fe.