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Case Report

Histomorphology of trabeculated urinary bladder – A cadaveric report

Sarah Ralte [a,*], *Vandana Raphael* [b], *Asima Bhattacharyya* [a]

[a] *Department of Anatomy, North Eastern Indira Gandhi Regional Institute of Health & Medical Sciences (NEIGRIHMS), Shillong 793018, Meghalaya, India*

[b] *Department of Pathology, North Eastern Indira Gandhi Regional Institute of Health & Medical Sciences (NEIGRIHMS), Shillong 793018, Meghalaya, India*

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ABSTRACT

Introduction: Trabeculation of urinary bladder is a common complication of benign prostatic hyperplasia, a predominant cause of bladder outlet obstruction in elderly males. The musculature of the urinary bladder, known as the detrusor urinae muscle, is hypertrophied so as to overcome the bladder outlet obstruction giving rise to trabeculated appearance of the urinary bladder.

Methods: In the present case study, trabeculation of the urinary bladder was observed in one of the cadavers, an elderly male aged 79 years, during routine dissection of pelvis by the students in the department of anatomy. Small pieces of the bladder were excised from its walls. Seven μm thick paraffin sections were stained with Masson's trichrome stain and examined under light microscope.

Results: The urinary bladder was big in size weighing 240 g. Well developed large trabeculae were seen criss-crossing the walls of the bladder on its inner surface. Smooth muscle hypertrophy and abundant deposition of collagen fibres between smooth muscle fibres were the main histological features. Stained sections of the median lobe of prostate gland showed large prostatic follicles with epithelial hyperplasia and inflamed fibrous stroma suggestive of benign prostatic hyperplasia.

Discussion: Benign prostatic hyperplasia commonly affects elderly men above 50 years of age presenting with symptoms of bladder outlet obstruction. In the present case study, we report the histological findings of the trabeculated urinary bladder observed in an elderly male cadaver during routine dissection.

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* *Corresponding author*. Tel.: +91 9774289885 (mobile).
E-mail address: sarahzoremi@gmail.com (S. Ralte).

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1. Introduction

The response of the urinary bladder to infravesical obstruction varies with the duration, severity and nature of the obstruction.[1] A variety of symptoms are associated with bladder outlet obstruction in men with benign prostatic hyperplasia. Trabeculation of the urinary bladder is one of the common complications associated with benign prostatic hyperplasia. The detrusor urinae muscle, the muscular component of the urinary bladder wall, is made of smooth muscle fibres arranged in spiral, longitudinal, and circular bundles. This muscle layer is hypertrophied so as to overcome the bladder outlet obstruction, resulting in a trabeculated appearance of the urinary bladder. The symptoms experienced, and the functional effects observed in males with bladder outlet obstruction, reflect the various changes occurring both within, and the surrounding detrusor smooth muscle cells.[2]

Previous studies have reported hypertrophic and/or hyperplastic changes in detrusor smooth muscle and increase in the extracellular connective tissue fibres, predominantly collagen fibres, and to a lesser extent elastic fibres, within the detrusor muscle component in patients with infravesical obstruction of urinary bladder.[3,4] Experimental studies conducted on animal models with induced bladder outlet obstruction have also demonstrated detrusor muscle hypertrophy along with a marked increase in the collagen fibres of extracellular matrix.[5–7] Congenital or acquired infravesical obstruction can result in a fibrotic vesical wall, characterized by trabeculation, increase in extracellular matrix proteins, low volumetric capacity under high pressure and low vesical compliance of the urinary bladder.[8–11]

In the present case study, we report the histomorphological findings of the trabeculated urinary bladder observed during routine dissection in an elderly male cadaver.

2. Case report

A case of trabeculated urinary bladder was found in one of the cadavers; an elderly male aged 79 years, during routine dissection of pelvis by the students, in the department of anatomy, NEIGRIHMS. The urinary bladder was big in size as compared to a normal bladder, filling up the entire pelvis and protruding into the lower abdominal cavity. The dry weight of the urinary bladder measured 240 g.

An incision was given along the lateral borders of the urinary bladder. On cutting open the urinary bladder, well developed large trabeculae were seen criss-crossing the walls of the bladder on its inner surface (Fig. 1). Enlargement of median lobe of prostate gland could be seen protruding through the mucosa of the bladder (Fig. 1). Small pieces of the bladder were excised from its walls. Sections were also taken from the median lobe of prostate gland. Seven μm thick paraffin sections were stained with Masson's trichrome stain and examined under light microscope.

2.1. *Light microscopy observations*

In Masson's trichrome stain, we observed there was hypertrophy of individual smooth muscle cells along with overall

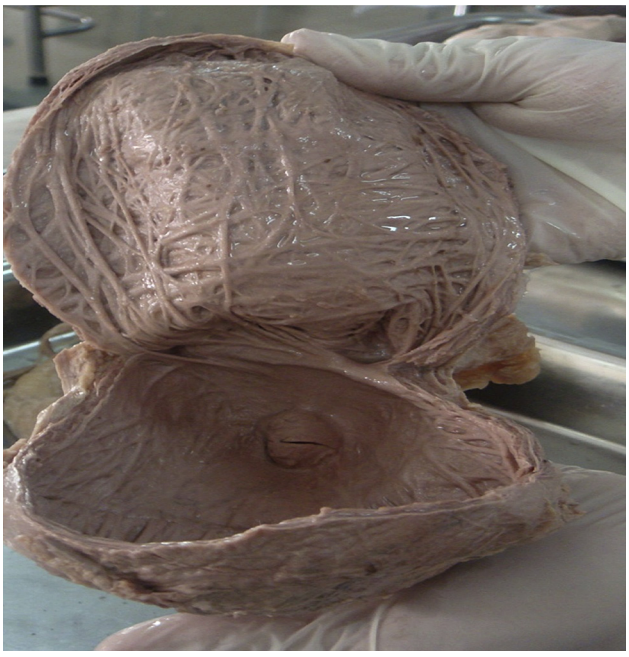

Fig. 1 – Interior of urinary bladder showing smooth muscle trabeculations.

increase in the size of muscle fascicles (Fig. 2) which would account for the size and increased weight of the urinary bladder. Abundant extracellular matrix proteins predominantly collagen fibres, were seen lying between and within the smooth muscle fascicles (Fig. 2). Stained sections of the median lobe of prostate gland showed large prostatic follicles

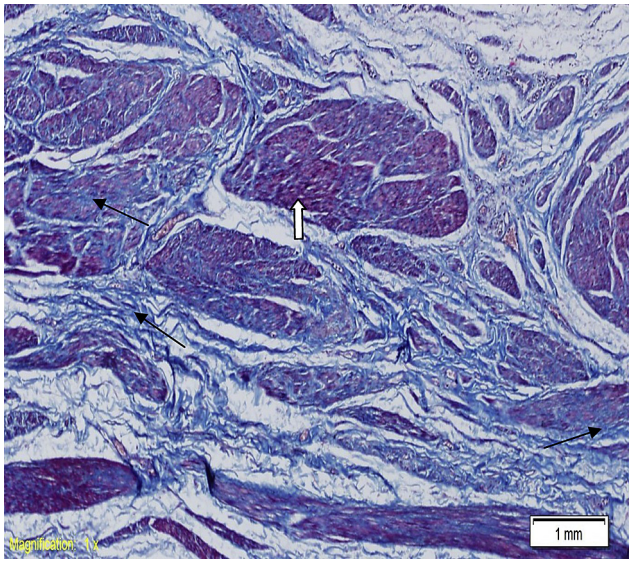

Fig. 2 – Masson's trichrome (10×): Hypertrophy of individual smooth muscle cell and increased size of muscle fascicle (white arrow) is the predominant histological alteration in bladder outlet obstruction (BOO). Excess extracellular matrix proteins, largely collagen fibres (stained blue), are seen both between and within smooth muscle fascicles (black arrows). Inflammatory exudates are also seen.

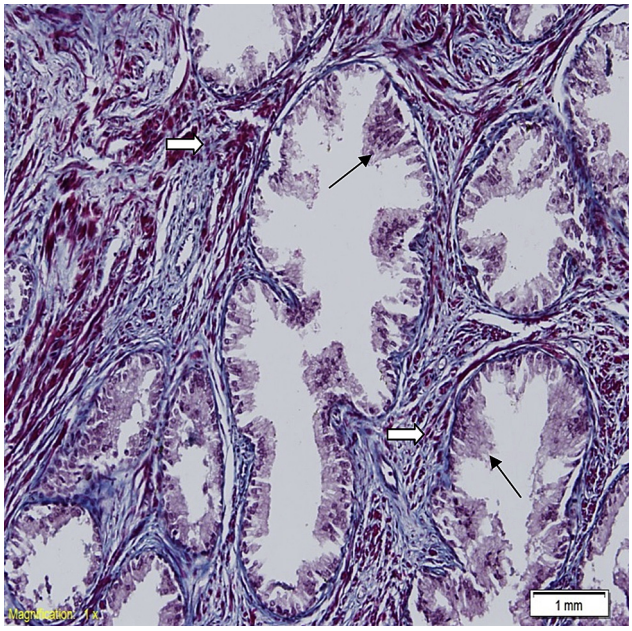

Fig. 3 – Masson's trichrome (20×): Enlarged prostatic follicles lined with epithelial hyperplasia (black arrows) amidst inflamed fibrous stroma (white arrows) which is suggestive of benign prostatic hyperplasia (BPH).

with epithelial hyperplasia and inflamed fibrous stroma suggestive of benign prostatic hyperplasia (Fig. 3).

3. Discussion

Over activity of detrusor urinae (or simply detrusor), the smooth muscle of the urinary bladder, has been found in as many as 50–60% of male patients with bladder outlet obstruction.[1] Raised intravesical pressure of the urinary bladder brought about by the combination of a fixed intravesical obstruction and the compensatory response of the detrusor to overcome the intravesical obstruction leads to a chain of events resulting in changes in smooth muscle appearance and function, extracellular matrix deposition as well as innervation of the detrusor muscle.[2–4]

In the present case study, we observed well developed large trabeculae criss-crossing the inner walls of the urinary bladder. Light microscopic studies of the specimen tissue showed smooth muscle hypertrophy, and abundant increase in the extracellular matrix proteins, chiefly collagen fibres, in and between the smooth muscle fascicles, which would account for the marked increase in the musculature of the urinary bladder. Gilpin et al while studying the morphology and morphometry of detrusor muscle fibres from patients with urodynamically obstructed bladder, described the smooth muscle compensatory hypertrophy and connective tissue infiltration of detrusor muscle bundles,[3] which is in accordance with our results observed in the present case study.

In experimental animal models, smooth muscle hypertrophy was observed as the predominant histological alteration in most forms of bladder outlet obstruction, and was largely responsible for the tremendous increase in muscle mass of the urinary bladder.[5–8] Damaser observed that rapid increase in bladder weight may reach 8-fold depending on the severity and duration of bladder outlet obstruction.[6] Studies conducted by Mostwin et al and Nyirady et al in animals with bladder outlet obstruction have shown that relative ischaemia in the detrusor muscle of the urinary bladder was created by the raised intravesical pressure seen with bladder outlet obstruction.[7,8] A study by Buttyan revealed that the growth factors were shown to be enhanced, for example the fibroblast growth factor, or were suppressed such as the transforming growth factor (beta) in the urinary bladder in association with relative ischaemia, which accounted for the excess deposition of extracellular matrix proteins, largely collagen fibres in and between smooth muscle fascicles,[9] as was seen in the present case study.

The light and electron microscopic structure of biopsy samples of trabeculated urinary bladder from patients with bladder outflow obstruction due to prostatic hypertrophy were compared with the morphology of control bladder specimens in a study by Gosling and Dixon.[10] They observed that irrespective of age of patients, detrusor muscle from trabeculated bladders contained many muscle bundles in which the constituent cells were of relatively small diameter and were widely separated from each other by dense masses of connective tissue containing collagen fibrils, and electron dense microfibrils which were in continuity with the basal laminae of the smooth muscle cells. In the control bladder specimens, the detrusor muscle bundles were composed of smooth muscle cells closely packed together with very little intervening connective tissue.[10] In a stereological study of collagen and elastic fibre system in the detrusor muscle of bladders from controls and patients with infravesical obstruction, Rubinstein et al found that the components of connective tissue, collagen and elastic fibres were increased (collagen = 4.89 ± 2.64%; elastin = 10.63 ± 2.00%; $p < 0.0001$) in the detrusor muscle of patients with benign prostatic hyperplasia (BPH), when compared to controls (collagen = 2.32 ± 1.25%; elastin = 8.94 ± 1.19%; $p < 0.0001$).[11]

Gosling, in a study on the morphology of bladder structure in response to bladder outflow obstruction, observed that many smooth muscle cells were surrounded by large amounts of connective tissue and some of the muscle cells were involved in collagen synthesis.[12] Buoro observed that myofibroblast derived smooth muscle cells were partially responsible for deposition of collagen fibres induced by partial bladder outlet obstruction in rabbit model.[13]

Saito and Lin demonstrated that long standing bladder outlet obstruction resulted in smooth muscle hypocontractility, which eventually impaired the emptying of the urinary bladder in animal models.[14,15] Mirone et al described the correlation between detrusor collagen content and urinary symptoms in patients with prostatic obstruction. They observed that collagen content in bladder detrusor muscle specimens were significantly higher (50.45% ± 8.22%) in BPH patients having severe symptoms as compared to BPH patients with moderate symptoms (43.09% ± 7.05%).[16]

We also observed enlarged prostatic follicles amidst inflamed fibrous stroma of the prostate, which was suggestive

of benign prostatic hyperplasia as the cause of bladder outlet obstruction in the present case study.

Changes in extracellular matrix deposition and detrusor smooth muscle appearance, metabolism, contractility, and detrusor innervation, develop in conjunction with the ischaemia experienced during bladder outlet obstruction. These factors would contribute to the physiologic alterations in urinary bladder function noted with infravesical obstruction, giving rise to a myriad of signs and symptoms associated with bladder outlet obstruction in clinical settings like benign prostatic hyperplasia commonly affecting elderly males.

Conflicts of interest

All authors have none to declare.

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