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HISTOPATHOLOGICAL SPECTRUM OF BENIGN PROSTATIC HYPERPLASIA - A ONE YEAR STUDY

Pathology

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ABSTRACT

Aims and objectives: To study the age distribution and histopathological spectrum of benign prostatic hyperplasia

Material and methods: This is a retrospective study of 37 cases of Benign Prostatic Hyperplasia during the period of January 2017 to December 2017.The gross specimens were in the form of transurethral resections of prostate(TURP). All the cases were analysed according to age and microscopic examination. Morphological patterns and features of benign prostatic hyperplasia were described.

Results and conclusion: The most common morphological pattern seen was mixed type with both glandular and stromal hyperplasia with papillary and branching pattern.

KEYWORDS

Benign Prostatic Hyperplasia, Histopathological spectrum.

INTRODUCTION

Prostate gland is an exocrine gland and the largest accessory reproductive organ in male. It is the most commonly affected organ in males with increasing age, accounting for significant morbidity and mortality. The most important categories of prostatic diseases are inflammatory lesions, Nodular hyperplasia and Carcinoma. Prostate has three major glandular regions- peripheral zone, central zone and transitional zone. Prostatic hyperplastic lesions are common in transition zone and peripheral zone is the main site for carcinomas. Transurethral resection of prostate(TURP) is a common urological procedure primarily used for the surgical management of benign prostatic hyperplasia(BPH).

BPH represents nodular enlargement of the prostate caused by proliferation of both glandular and stromal components. The incidence of BPH increases with age, being only 8% during the fourth decade, 50% in the fifth decade and upto 75% in the eighth decade. Prostatitis occurs in approximately 10% to 15% of men. It may be classified as acute, chronic and granulomatous and is a common finding associated with BPH.(1)

The present study was conducted with an aim to enumerate histomorphological spectrum of BPH in TURP specimens.

MATERIALS AND METHODS

This is a retrospective study of 37 cases of Benign Prostatic Hyperplasia during the period of January 2017 to December 2017 from Sree Balaji Medical College and Hospital, Chennai. The gross specimens were in the form of transurethral resections. The tissue was fixed in 10% neutral buffered formalin. Sections were cut at 3 micron thickness and were subsequently stained by haematoxylin and eosin stain. All the cases were analysed according to age and microscopic examination. Morphological patterns and features of benign prostatic hyperplasia were described.

RESULTS

All the 37 TURP specimens received were gray-tan to gray-black in colour and soft to firm in consistency. They were analysed as per age and for histomorphological patterns. Results are tabulated below.

Table 1: Age distribution

Age(years)	No. of cases	Percentage
51-60	06	16.2
61-70	19	51.4
71-80	09	24.3
81-90	03	8.1
Total	37	100

In the current study, most of the patients(51.4%) were in 6th decade followed by 7th decade(24.3%). This is similar to study done by Mathi A et al (2), Puttaswamy K et al(3),Arya RC et Al(4) and Kumar M et al(5).

Table 2: Pattern of hyperplasia

Pattern	No. of cases	Percentage
Pure stromal nodules	01	2.7
Mixed pattern	32	86.5
Predominantly epithelial hyperplasia	04	10.8
Total	37	100

The most common pattern of hyperplasia was mixed pattern (Fig. 2) with both glandular and stromal hyperplasia (86.5%). The findings encountered agree with a similar study on benign disorders of the prostate in Kuwait(6) and histopathological pattern of prostatic diseases in Nigeria(7).

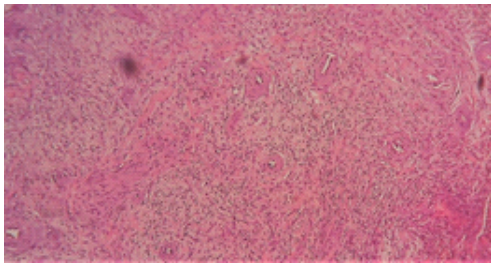

Fig . 1- Stromal hyperplasia

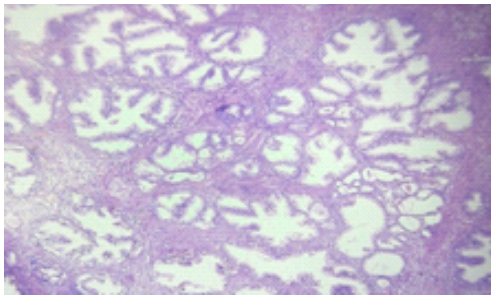

Fig.2 – Glandular hyperplasia with papillary hyperplasia

Table 3: Different Histopathological features

Histopathological features	No. of cases	Percentage
Papillary hyperplasia	18	48.6
Cystic change	22	59.5
Basal cell hyperplasia	03	8.1
Clear cell metaplasia	04	10.8
Squamous metaplasia	01	2.7
Transitional cell metaplasia	04	10.8
Inflammation(Prostatitis)	19	51.4
Myxoid change in stroma	03	8.1
Prostatic intraepithelial neoplasia(PIN)	01	2.7

Non-specific prostatitis (Fig. 3) was observed in 51.4% cases. Kohnen et al(8) and Anim et al(9) found nonspecific prostatitis in 90-98% cases of BPH.

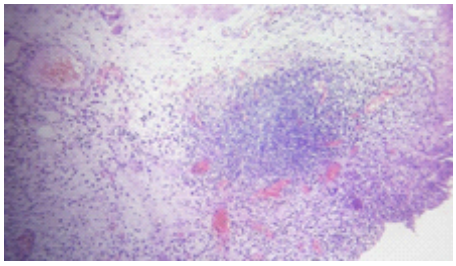

Fig.3 –Nonspecific prostatitis with lymphocytic infiltrate

Di Silverio F et al. studied 3942 patients with benign prostatic hyperplasia and observed that focal acinar/cystic atrophy(Fig. 4) was found significantly increased according to the patient age in decades. In concurrence with the above study this study also showed 59.5% cases with cystic/acinar atrophy showing an increased incidence towards the higher age group(10).

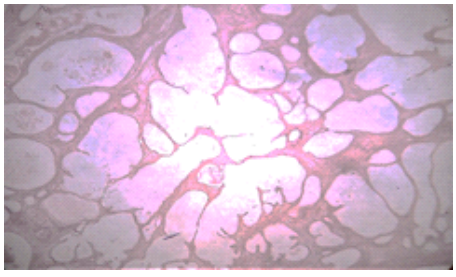

Fig.4 – Cystic atrophy

The second most common morphological change seen is papillary hyperplasia (48.6%) (Fig. 2). 8.1% cases showed basal cell hyperplasia(Fig. 6). Clear cell metaplasia(Fig. 5a) and transitional cell metaplasia (Fig. 5b) was seen in 10.8% cases each and squamous metaplasia(Fig. 5c) in 2.7% i.e. one case. Myxoid change in stroma was observed in 8.1% cases. One case i.e 2.7% showed focus of High grade PIN.

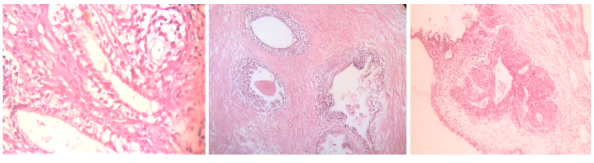

Fig. 5a,5b,5c – Metaplasias

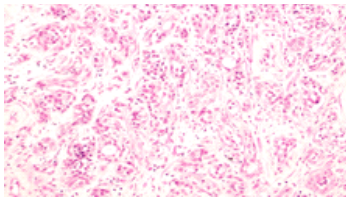

Fig. 6- Basal cell hyperplasia

DISCUSSION

The current study was done retrospectively on TURP specimens received in a tertiary hospital. All the 37 cases studied showed benign nodular hyperplasia of prostate. Majority were observed in the 6th and 7th decade.

Benign prostatic hypertrophy is the usual name applied to a common benign disorder of the prostate that, when extensive, results in varying degrees of urinary obstruction, sometimes requiring surgical intervention. The disease represents a nodular enlargement of the gland caused by hyperplasia of both glandular and stromal components. This results in an increase in the weight of the organ well beyond the 20 g regarded as normal for adult individuals. The clinical incidence is higher after the 5th decade and increases with age. Nodular hyperplasia usually begins in the 'inner' gland, i.e., the portions around the urethra and specifically where the ejaculatory ducts enter the urethra, which are also referred to as the periurethral and transitional zones. This fact supports the interpretation that this portion of the gland reacts differently to hormonal stimuli than the outer portion. In most instances the nodules congregate on both sides of the urethra, resulting in so-called lateral lobe hyperplasia. In others the disease results in a midline dorsal nodule at the bladder neck protruding into the bladder lumen. With increased growth, the periphery of the organ is pushed aside and compressed. In only about 5% will a focal lesion of nodular hyperplasia be found in the peripheral zone of the organ.

Microscopically, the earliest change is a stromal proliferation about small sinusoidal spaces in the periurethral regions and, to a lesser degree, in the periductal and intralobular areas. This stromal proliferation (which in the periductal areas may have a concentric or an eccentric quality) contains more smooth muscle and less elastic tissue than the normal stroma. This is followed by hyperplasia of the glandular component, so that in the well developed disease the nodules are composed of varying proportions of both elements. These proportions are somewhat different in patients with symptomatic and those with asymptomatic nodular hyperplasia. The glands are dilated or even cystic and often contain an inspissated secretion of glycoprotein nature (corpora amylacea), which is sometimes calcified. The epithelium ranges from flat to columnar, sometimes facing each other in the same gland ('functional polarization'); the cytoplasm is pale, and the nuclei are regular and centrally located. The nucleoli are inconspicuous. Papillary infoldings are common. A continuous basal cell layer is seen immediately above a well-developed basement membrane. Small clusters of lymphocytes are common in the interstitium and around the ducts. They are probably the result rather than the cause of the hyperplasia; a diagnosis of chronic prostatitis is not warranted because of their mere presence. Many morphologic variations of this basic theme exist, some of them resulting from the overgrowth of one component over the other and others from the emergence of distinctive patterns. Interestingly, many of the latter bear a notable resemblance to lesions of the breast and have been named accordingly. They include sclerosing adenosis, fibroadenoma-like and phylloides tumor-like proliferations, leiomyoma-like and fibromyxoid nodules, and the presence of bizarre cells in the stroma. Transitional cell metaplasia, squamous metaplasia and clear cell metaplasia may be observed. Squamous metaplasia is seen at the periphery of infarcts. Cribriform hyperplasia and basal cell hyperplasia might be also seen. Basal cell hyperplasia may be the progenitor of the so-called 'adenoid basal cell tumor'.

Acute prostatitis is rarely seen in surgical specimens. Chronic prostatitis is more common, but it is important to distinguish the true infectious processes of this organ from the inconsequential mononuclear infiltrates often seen accompanying nodular hyperplasia.

Prostatic intraepithelial neoplasia (PIN) is the currently preferred term for a process involving prostatic ducts and acini, which has also been described as intraductal or ductal–acinar dysplasia. It is often multicentric and may even extend to the prostatic utricle. It is grouped into two categories low grade PIN and high grade PIN based on the severity of the following alterations: cell crowding and stratification; nuclear enlargement, pleomorphism, and chromatin pattern; and nucleolar appearance. The key feature in distinguishing high grade from low-grade PIN is the nuclear (and particularly the nucleolar) appearance, regardless of architecture. Morphologic variations of PIN include tufting, micropapillary, cribriform, and flat/atrophic (equivalent of 'clinging' in the breast) patterns at the architectural level, and inverted (hobnail) and foamy types at the cytologic level. Association between high grade PIN and prostatic adenocarcinoma is well documented.

In the current study, as mentioned in the given literature, majority of the cases had both glandular and stromal hyperplasia with presence of papillary infoldings, cystic changes and non-specific prostatitis in many. High grade PIN was also reported in one case. Other changes like basal cell hyperplasia, clear cell metaplasia, transitional cell metaplasia and squamous cell metaplasia were also noted.(1,11).

CONCLUSION

The aim of the current study was to study the age distribution and the histopathological spectrum of BPH. According to this study, 6th and 7th decades are the most common groups of men presenting with BPH. Histopathologically, mixed pattern with both glandular and stromal

hyperplasia is most common in BPH. Papillary hyperplasia and cystic change are the most common morphological changes seen. BPH with non-specific prostatitis was also seen in more than 50% cases. PIN is relatively uncommon in TURP specimens which is also observed in the current study. It is suggested that the presence of high-grade PIN in the adenomatous zone in cases of nodular hyperplasia requires a follow-up for concurrent or subsequent invasive carcinoma. Identification of premalignant lesions and incidental prostate cancer can improve the treatment outcome of patients.

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