Original Article

# A hospital-based study of prostate biopsy results in Indian males

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## Abstract

**Introduction:** The prostate is a gland belonging to the male reproductive system. Aging results in the dysfunction of the prostate that may present as inflammation, enlargement, and cancer. Additionally, the diseases of the prostate including cancers are slow in progression, and therefore, it is difficult to diagnose them early. Hence, it is increasingly important for physicians to recommend histopathological examination of the prostate gland to identify, manage, and treat prostate cancers. This study was conducted to assess prostate diseases among biopsy specimen collected from patients with signs of prostate diseases. **Materials and Methods:** This prospective study was conducted in the Department of Pathology, Deccan College of Medical Sciences, Owaisi Hospital, Hyderabad, between June 2012 and September 2014. All gross specimens ($n$ = 300) of the prostate such as the needle biopsies of the prostate, transurethral resection of the prostate (TURP) chips, and excised specimens of the prostate were included in the study. Histopathological examinations of the biopsies were performed for nuclear size, chromatin material, nucleoli, membrane thickness, irregularity, cytoplasmic granularity, staining, and cell border conspicuity. The biopsies were also assessed for lobule formation, secretions, polymorphonuclear leukocytes, lymphocytes, macrophages, connective tissue stromal cells, their arrangements, and acellular connective tissue material. **Results:** Of 300 total prostatic biopsies performed, 56 (18.66%) were identified as inflammatory lesions of the prostate (prostatitis), 98 (32.66%) revealed benign prostatic lesions (benign prostatic hyperplasia (BPH)), 112 (37.33%) were identified as BPH with premalignant lesions, and 34 (11.33%) were revealed as malignant tumors of the prostate. Chronic prostatitis (67.85%) was the common inflammatory lesion. The majority (91.42%) revealed epithelial lesions compared to stromal lesions (08.58%). BPH was predominantly (28.00%) noticed among patients in the age group of 61–70 years. Prostatic intraepithelial neoplasia (PIN) was observed majorly (53.35%) in the age group of 61–70 years. Most of the prostatic cancers were identified as adenocarcinomas. However, three variants were also categorized as small cell carcinoma, signet ring cell carcinoma, and transitional cell carcinomas. **Conclusions:** The results reveal that prostatic adenocarcinomas are predominant among the study population. Additionally, prostatic diseases including cancer are commonly noticed among people belonging to the age group of 61–70 years. More than one-third of patients showed BPH with premalignant lesions, and a majority of the study population showed evidence of chronic prostatitis.

**Keywords:** Biopsy, cancer, prostate

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## Introduction

Diseases of the prostate are a common cause of urinary problems in men. Moreover, prostatic diseases are noted to increase with age and particularly affect people over 60 years.[1] Prostate enlargement results in compression of the intraprostatic



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portion of the urethra. The most common pathological disorders of the prostate gland are inflammation, benign enlargement, premalignant lesions, and malignant tumors. Carcinoma of the prostate gland is the second most frequent cause of death in men worldwide.[2] However, low incidence was reported in Asian countries including India, Sri Lanka, and Japan as compared to Western countries including North America.[3]

Acute prostatitis typically results from microorganisms such as bacteria, fungi, and viruses being implanted in the prostate usually by intraprostatic reflux of urine from the posterior urethra or from the urinary bladder but occasionally the microbes seed the prostate through lymphohematogenous routes from distant foci of infection.[4] Histological evidence of benign prostatic hyperplasia (BPH) can be seen in approximately 20% of men at 40 years of age, which increases to 70% by the age of 60 years and 90% by the age of 80 years. Thus, BPH is a prostatic abnormality that frequently results in significant morbidity among men.[5] Prostatic intraepithelial neoplasia (PIN) represents a continuous pathological process that initially presents as a low-grade dysplasia, and over time, it transforms into carcinoma.[6] The PIN is a potential biological precursor of adenocarcinoma of the prostate with the same morphological features as cancer which, however, lacks invasive capabilities.

Prostate cancer is typically a disease of men over the age of 50 years. The incidence of prostate cancer was found to increase from 20% in men who are in their 50s to approximately 70% in men over 70 years of age. Prostate cancer is associated with complexities of diagnosis as it gradually evolves over age, could be associated with several predisposing factors such as infection, inflammation, lifestyle, diet, and genetic causes, and results in significant morbidity and mortality among the affected population.[7,8]

This study was conducted to analyze the histopathological features of prostate gland biopsies among patients attending a tertiary care teaching hospital in South India.

## Materials and Methods

This prospective study was conducted in the Department of Pathology, Deccan College of Medical Sciences, Owaisi Hospital, Hyderabad, between June 2012 and September 2014. The study was approved by the Institutional Review Board of Deccan College of Medical Sciences and Allied Hospitals, Hyderabad—500058 (IEC-11-11-14). All gross specimens ($n$ = 300) of the prostate such as the needle biopsies of the prostate, transurethral resection of the prostate (TURP) chips, and excised specimens of the prostate were included in the study. Histopathological examinations of the biopsies were performed for nuclear size, chromatin material, nucleoli, membrane thickness, irregularity, cytoplasmic granularity, staining, and cell border conspicuity. The biopsies were also assessed for lobule formation, secretions, polymorphonuclear leukocytes, lymphocytes, macrophages, connective tissue stromal cells, their arrangements, and acellular connective tissue material.

### Inclusion and exclusion criteria

All the specimens for histopathological examination were received in 10% formalin containers. The specimens received were considered adequate when the needle biopsies were at least measuring two centimeters (cm) in length and submitted in duplicate. TURP chips of prostatic tissue weighing more than or equal to five grams have been accepted for the study. Additionally, the acceptance criteria for all the excised specimens included were the resected specimens with the representative central, transitional, and peripheral zones.

The submitted specimens were excluded when the needle biopsies received were less than two cm in length and with no duplicate specimen, TURP chips of prostatic tissue weighing less than five grams, and all the excised specimens that were partially resected with inconspicuous differentiation of the representative central, transitional, and peripheral zones.

### Experimental procedure for histopathological examinations

The resected specimen of the prostate was cut frontally, and representative bits from the central zone, peripheral zone, and transitional zone were extracted. All the biopsy specimens were further fixed in 10% formalin for at least 2 hours in porous stainless steel processing capsules along with proper numbering and labels.

The prostatic tissue/bits were then processed in an automated tissue processor (Yorco, New Delhi). The thin sections were suspended in a water bath and taken onto clean Mayer's albumin-coated glass slides. These sections were deparaffinized in a hot air oven and hot plate for 30 minutes.

Further, these sections were stained with hematoxylin and eosin as per the standard guidelines. The slides were cleared in xylene 1 and xylene 2 for 5 minutes each and were later mounted in dibutyl phthalate xylene (DPX) fixative. All the sections over the glass slides were thoroughly examined under a binocular compound microscope using different magnification (X) objective lenses such as scanner (4X), low power (10X), and high power (40X).

## Results

Of the total 300 prostatic biopsies performed, 56 (18.67%) were identified as inflammatory lesions of the prostate (prostatitis), 98 (32.67%) revealed benign prostatic lesions (BPH), 112 (37.33%) were identified as BPH with premalignant lesions, and 34 (11.33%) revealed malignant tumors of the prostate as shown in Table 1.

On microscopic examination, an acute prostatitis case was confirmed when the prostate contained multiple abscesses and

foci of necrosis. Other features suggestive of acute prostatitis included the presence of inflammatory cells, neutrophils, and necrotic material in the dilated acini and ducts. Additional features for acute prostatitis were neutrophilic infiltration adjacent to the stroma, and some of the acini are seen distended with a purulent exudate. Chronic prostatitis was confirmed when there was evidence of focal to diffuse infiltration of chronic inflammatory cells, lymphocytes, and plasma cells in the stroma and acini. Some of the acini are dilated showing squamous metaplasia and transitional metaplasia in their epithelial lining. Microscopic evidence of chronic inflammatory cells, lymphocytes, plasma cells, lipophages, langhans giant cells and occasional presence of stromal nodular hyperplasia was used to diagnose non- specific granulomatous prostatitis.

Among the cases of prostatitis, chronic prostatitis (67.85%) was the common inflammatory lesion followed by acute prostatitis (07.15%) and non-specific granulomatous prostatitis (25%) as shown in Table 2.

BPH (70%) was the most common prostatic lesion identified in the study population. The age groups most affected were 51–60 years (22.66%) and 61–70 years (28%). The age-wise distribution of BPH lesions is shown in Table 3.

BPH was identified using characteristics such as the cut surface revealing varying sizes of nodules and honeycombed architecture. Some of them showed cystic spaces and corpora amylacea. Microscopically, prostatic hyperplasia was observed in the inner zone of the prostate. The acini are increased in number and size with many acini showing dilatation and invagination. Most of the acini are lined by columnar cells with poorly defined borders and abundant double layers. Most of the cases showed uneven hyperplasia of glandular and stromal nodules compressing the adjacent structures.

Among the 210 BPH lesions noted in the study, the majority (91.42%) revealed epithelial lesions compared to stromal lesions (08.57%). Among the BPH cases, PIN was observed in 53.33% of cases which is the highest incidence among the epithelial type of BPH lesions. Simple BPH (24.76%) was the next most common epithelial type of BPH lesion. Basal cell hyperplasia (BCH) was found in eight (3.80%) cases. Microscopically, BCH was identified by the presence of small groups of acini and proliferating basal cells. The stroma of BCH appears more cellular and shows the proliferation of fibroblasts and myofibroblasts.

Two (00.95%) cases of clear cell cribriform hyperplasia (CCCH) were observed in this study. Microscopically, CCCH with BHP was identified when a nodular growth pattern is noticed along with acini that were distended by the proliferation of clear cells which are in papillary or cribriform arrangement. The acini are lined by cuboidal to columnar cells with abundant clear cytoplasm. The nuclei are small round and uniform in appearance.

In this study, 10 (04.76%) cases of atrophy associated with BPH were observed in 210 BPH lesions. In most cases, atrophy was observed in the peripheral zones. By microscopic examination, atrophy was classified into simple lobular atrophy, cystic atrophy, and sclerotic atrophy. The details of prostatic lesions and types of BPH are elaborated in Table 4.

Microscopically, the PIN was identified following the observation at low magnification of foci of cytologic abnormality as sharply demarcated clusters of glandular units that were distinguished from the adjacent tissue of deeply stained and thickened epithelium. Definitive diagnosis and grading of severity for each focus were performed at high magnification. Deviation from normal epithelium was identified as PIN-1 or low-grade prostatic intraepithelial neoplasia (LGPIN) [84 (75%)]. Microscopic characteristics found included a prominent

**Table 1: Distribution of prostatic lesions**

| Name of the prostatic lesion | Number (%); total=150 |
|---|---|
| Prostatitis | 56 (18.67) |
| Benign prostatic lesions (BPH) | 98 (32.67) |
| BPH with premalignant lesions | 112 (37.33) |
| Malignant tumors of the prostate | 34 (11.33) |

**Table 2: Incidence of various types of prostatitis**

| Type of prostatitis | Number (%) |
|---|---|
| Acute prostatitis | 4 (7.15) |
| Chronic prostatitis | 38 (67.85) |
| Non-specific granulomatous prostatitis | 14 (25) |

**Table 3: Age-wise distribution of BPH cases**

| Age group in years | Cases (*n*) | Cases showing BPH (*n*%) |
|---|---|---|
| 41 – 50 | 20 | 06 (02) |
| 51 – 60 | 92 | 68 (22.66) |
| 61 – 70 | 108 | 84 (28.00) |
| 71 – 80 | 64 | 42 (14.00) |
| >80 | 16 | 10 (03.33) |
| Total | 300 | 210 (70) |

BPH=benign prostatic hyperplasia

**Table 4: Types of prostatic lesions and BPH among the cases**

| Type of prostatic lesion | Cases (*n*%) |
|---|---|
| Epithelial lesions | 192 (91.42) |
| Simple BPH | 52 (24.76) |
| BPH with BCH | 08 (03.80) |
| BPH with CCCH | 02 (00.95) |
| BPH with atrophy | 10 (04.76) |
| BPH with sclerosing adenosis | 08 (03.80) |
| BPH with PIN | 112 (53.33) |
| Stromal lesions | 18 (8.57) |
| Stromal hyperplasia | 18 (8.57) |
| Total | 210 (100) |

BPH=benign prostatic hyperplasia, BCH=basal cell hyperplasia, CCCH=cases of basal cell hyperplasia, PIN=prostatic intraepithelial neoplasia

increase in nuclear size and cell crowding accompanied by irregularity in nuclear spacing. Multiple foci of PIN were observed at low magnification. Twelve cases (10.71%) of PIN-2 were observed in 112 cases of BPH with PIN. In this study, the cardinal diagnostic feature for PIN-3 was the presence of large prominent eosinophilic nucleoli in the majority of cells. Nuclear hyperchromatism was similar to that observed in PIN-2 but chromatin margination beneath the nuclear membrane was more prominent and seen in more nuclei. Marked nuclear enlargement was seen in the majority of cells. The degree of nuclear crowding was severe, with bridges of epithelial cells extended and showing a cribriform, and trabecular pattern. PIN 2 and PIN 3 are also reported as high-grade prostatic intraepithelial neoplasia (HGPIN). Sixteen (14.28%) cases of PIN-3 were identified among the total cases of BPH with PIN. The majority of the cases of BPH with PIN were noticed in the age group of 61–70 years as shown in Table 5.

A total of 34 (11.33%) cases of prostatic cancer were identified, all of which were epithelial in origin (100%) as shown in Table 6.

All the 34 prostatic malignancies in the study were recognized as adenocarcinomas. Of these, five variants were categorized as shown in Table 7.

**Table 5: Age-wise distribution of BPH with PIN types among the cases**

| Age group in years | Cases of BPH with PIN | LGPIN PIN-1 | HGPIN | |
|---|---|---|---|---|
| | | | PIN-2 | PIN-3 |
| <40 | 00 | 00 | 00 | 00 |
| 41-50 | 12 | 12 | 00 | 00 |
| 51-60 | 20 | 12 | 04 | 04 |
| 61-70 | 50 | 44 | 02 | 04 |
| 71-80 | 12 | 08 | 02 | 02 |
| >80 | 18 | 08 | 04 | 06 |
| Total | 112 | 84 | 12 | 16 |

BPH=benign prostatic hyperplasia, PIN=prostatic intraepithelial neoplasia

**Table 6: Frequency distribution of malignant tumors of prostate**

| Type | Number of cases | Percentage% |
|---|---|---|
| Epithelial type | 34 | 100% |
| Stromal type | 0 | 0 |
| Lymphoma and sub-types | 0 | 0 |
| Metastatic tumors | 0 | 0 |
| Total | 34 | 100% |

**Table 7: Variants of adenocarcinoma**

| Variants of adenocarcinoma | Number of cases (*n*=) |
|---|---|
| Small cell minor type | 26 |
| Trabecular type | 2 |
| Small cell carcinoma | 2 |
| Signet ring cell carcinoma | 2 |
| Transitional cell carcinoma | 2 |
| Total | 34 |

Histopathological examinations of cancerous tissue revealed larger than the normal size or smaller than the normal cell size with a firm to variable consistency. The cut section showed dry, fibrous areas and homogenous solid areas containing yellowish zones as shown in Figure 1. Microscopically, the tumors showed glands that are closely packed with a little stroma as shown in Figure 2. Additionally, in between them in some cases, the glands had haphazardly distributed stroma. These glands are tiny to small, and some were simple large and fused glands. Few showed columns, cords, or solid sheets of cells with no gland formation. The cell varied from cuboidal to columnar cells. Cellular anaplasia is slight, and giant cells and mitotic figures were absent. The nuclei were small, the nuclear membrane was delicate, and chromatin was homogenously distributed. The stromal invasion was observed at the base of the acinus where there was an outgrowth of cells.

The age of the patient with cancer diagnosis ranged from 52 years to 86 years wherein the youngest patient was 52 years and the oldest patient was 86 years. No malignancy case was detected among patients below 50 years of age. The incidence of cancer varied with the age group: 61–70 years (0.98%), 71–80 years (4.88%), and 81–90 years (4.92%).

## Discussion

The American Cancer Society estimates have indicated that there was an annual increase of 3% in prostate cancer cases from 2014 to 2019.[9] The value of a detailed morphological study of lesions of the prostate gland lies not only in early diagnosis of prostatic cancer which is the second most frequent cause of death from cancer in men but also in predicting and planning the modality of treatment and assessing the prognosis.[10]

Disorders of the prostate gland are generally observed in older persons. However, the diagnosis requires radiological evidence including ultrasonography, magnetic resonance imaging (MRI),

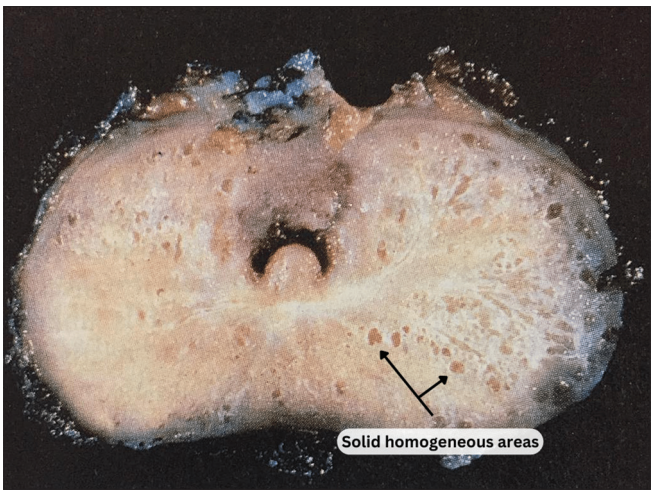


**Figure 1:** Cut section showing dry, fibrous areas and homogeneous solid areas containing yellowish zones

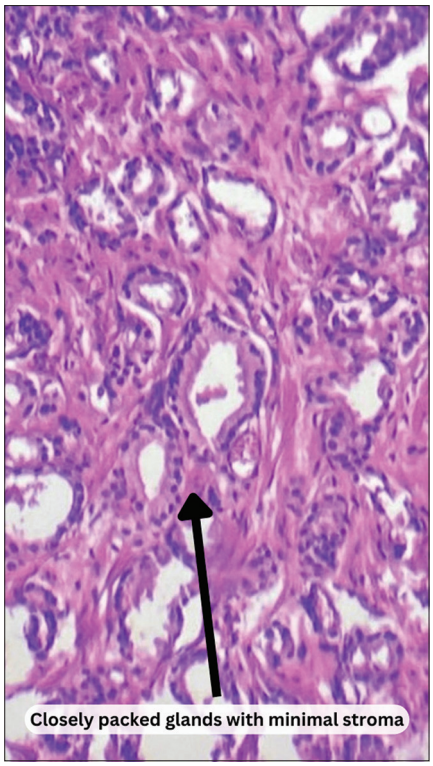


**Figure 2:** High-power view—mixed small and large carcinoma

serological evidence such as prostate-specific antigen (PSA), cathepsin D, thrombospondin, and histopathological examination of biopsy specimens.[11-13]

Biopsy of the prostate gland was suggested as a gold standard method for the diagnosis of prostate cancer.[14] There are different approaches to prostate biopsy including transrectal extended systematic biopsy, transrectal saturation biopsy, transperineal template mapping biopsy, transperineal biopsy schemes, transperineal freehand technique, and robotic and radiologically guided biopsy. A prostate biopsy has been recommended to assess the risk stratification of prostate cancer.[15]

A previous study from India had predicted an increase in the incidence of prostate cancer. This study also observed that the isolated incidences do not necessarily predict the real picture due to a lack of appropriate reporting guidelines. Moreover, there are no reliable population-based data on the prevalence of pancreatic diseases including cancer.[16]

In this study, we observed 210 of 300 (70%) cases of benign prostatic lesions. Most of these benign lesions observed were among patients above 50 years of age. A study from the same geographical region reported 80% of prostatic lesions as benign and 20% as malignant lesions.[17]

Similar findings were reported in a previous study from Chennai, South India. However, this study found higher rates (23.58%) of prostate carcinoma unlike the results of this study (17%).[18] Interestingly, a study from the same region reported higher rates (92%) of BPH and lower rates (8%) of prostate cancer when compared to the results of this study.[19] Results of a study from Ahmedabad, Western India, showed a similar (72%) prevalence of BPH and a higher incidence of prostate cancer [28%].[20]

A study from Jammu and Kashmir, North India, reported lower rates (3%) of prostatic cancer and 91% with BPH among the 245 prostate gland biopsy specimens analyzed.[21]

A Libyan study identified that the majority (82%) of prostate biopsies were BPH and 18% showed evidence of prostatic adenocarcinoma. Similar to the findings of the current study, this study also noted that the 60-70 years age patients were predominantly affected by BPH.[22] From Nepalese study results, the occurrence of BPH and malignant lesions was found more common among 69.6 ± 8.1 years and 72.9 ± 5.2 years, respectively. This study reported 90% of BPH cases and 8% of malignant lesions among prostate biopsies.[23]

In a study from Nigeria, histological examination of prostate biopsies revealed higher rates (39.3%) of prostate cancer and a significant proportion of them showed evidence of BPH (42.5%).[24]

The diagnosis of prostatic lesions including prostate cancer appears to be complex due to several reasons including non-specific clinical features, low awareness, and lack of infrastructure and technology, especially in the low socioeconomic regions of sub-Saharan African regions.[25] Therefore, in countries such as India, prostate biopsies may prove to be extremely important in the diagnosis and management of prostatic lesions including cancer. Additionally, it was recently identified that benign mimics of prostate cancer could make prostate biopsy examinations even more complex and it is advisable that conclusions about the presence or absence of cancer should be made cautiously.[26,27]

## Study limitations

This study was confined to a small geographical region, and the results obtained from this study may not reflect the population characteristics. Additionally, the biopsy results were not correlated with serological biomarkers of cancer such as PSA and radiological features, among others.

# Conclusions

The study results revealed that patients above 50 years of age have higher chances of developing prostatic lesions. BPH was the most common lesion followed by prostatitis and carcinoma of the prostate. The majority of the prostatic lesions were epithelial in origin. BPH with PIN lesions may be premalignant in nature, and early detection of these lesions could help in the diagnosis of carcinoma of the prostate and assist in a better prognosis for the patients. Therefore, a careful gross biopsy specimen examination, tissue processing, paraffin block making, section cutting, and proper staining with nuclear and cytoplasmic stains, followed by a thorough and comprehensive visualization under

progressively increasing objective magnifications of microscope and meticulous histopathological evaluation, constitute the basic approach toward a standard diagnosis of the prostatic pathology including cancer.

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## Conflicts of interest

There are no conflicts of interest.

## References

1. Fiard G, Stavrinides V, Chambers ES, Heavey S, Freeman A, Ball R, *et al.* Cellular senescence as a possible link between prostate diseases of the ageing male. Nat Rev Urol 2021;18:597-610.
2. Guimaraes CTS, Sauer LJ, Romano RFT, Pacheco EO, Bittencourt LK. Prostate benign diseases. Top Magn Reson Imaging 2020;29:1-16.
3. Zhu Y, Freedland SJ, Ye D. Prostate cancer and prostatic diseases best of Asia, 2019: Challenges and opportunities. Prostate Cancer Prostatic Dis 2020;23:197-8.
4. Khan FU, Ihsan AU, Khan HU, Jana R, Wazir J, Khongorzul P, *et al.* Comprehensive overview of prostatitis. Biomed Pharmacother 2017;94:1064-76.
5. Lawrentschuk N, Ptasznik G, Ong S: Benign prostate disorders. Feingold KR, Anawalt B, Blackman MR, *et al.* (ed): MDText.com, Inc., South Dartmouth (MA); 2000. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279008/. [Last accessed on 2024 Jan 24].
6. Joniau S, Goeman L, Pennings J, Van Poppel H. Prostatic intraepithelial neoplasia (PIN): Importance and clinical management. Eur Urol 2005;48:379-85.
7. Wasim S, Lee SY, Kim J. Complexities of prostate cancer. Int J Mol Sci 2022;23:14257.
8. De Nunzio C, Lombardo R. Best of 2022 in prostate cancer and prostatic diseases. Prostate Cancer Prostatic Dis 2023;26:5-7.
9. Siegel RL, Miller KD, Wagle NS, Jemal A. Cancer statistics, 2023. CA Cancer J Clin 2023;73:17-48. Available from: https://unitouchcdn.digiscapetech.com/jfmpc/10.4103/jfmpc.jfmpc_1339_23/Author_Proofing_Uniprr/jfmpc_1339_23_R3.pdf. [Last accessed on 2024 Jan 24].
10. Hugosson J, Månsson M, Wallström J, Axcrona U, Carlsson SV, Egevad L, *et al.* Prostate cancer screening with PSA and MRI followed by targeted biopsy only. N Engl J Med 2022;387:2126-37.
11. Handke AE, Albers P, Schimmöller L, Bonekamp D, Asbach P, Schlemmer HP, *et al.* [Systematic or targeted fusion-guided biopsy]. Urologie 2023;62:464-72.
12. Eldred-Evans D, Burak P, Connor MJ, Day E, Evans M, Fiorentino F, *et al.* Population-based prostate cancer screening with magnetic resonance imaging or ultrasonography: The IP1-PROSTAGRAM study. JAMA Oncol 2021;7:395-402.
13. Gentile F, La Civita E, Ventura BD, Ferro M, Bruzzese D, Crocetto F, *et al.* A neural network model combining [-2] proPSA, freePSA, total PSA, cathepsin D, and thrombospondin-1 showed increased accuracy in the identification of clinically significant prostate cancer. Cancers (Basel) 2023;15:1355.
14. Mahapatra QS, Mohanty P, Nanda A, Mohanty L. Histomorphological study of prostatic adenocarcinoma and its mimics. Indian J Pathol Microbiol 2019;62:251-60.
15. Gravestock P, Shaw M, Veeratterapillay R, Heer R: Prostate cancer diagnosis: Biopsy approaches. Barber N, Ali A (ed): Exon publications, Brisbane (AU); 2022. Available from: https://doi.org/10.36255/exon-publications-urologic-cancers-prostate-cancer-biopsy. [Last accessed on 2024 Jan 24].
16. Hariharan K, Padmanabha V. Demography and disease characteristics of prostate cancer in India. Indian J Urol 2016;32:103-8.
17. Krishna T. A clinico-histopathological spectrum of prostatic lesions at a tertiary care teaching institute. IP J Diagn Pathol Oncol 2020;5:300-5. 10.18231/j.jdpo. 2020.058.
18. Josephine A. Clinicopathological study of prostatic biopsies. J Clin Diagn Res 2014;8:FC04-6.
19. Muthuvel E, Vimal CR, Srinivasan C. Clinicopathological study of associated lesions in benign prostatic hyperplasia and prostatic adenocarcinoma in surgical biopsy specimens. Annals Pathol Lab Med 2018;5:158-64. 10.21276/APALM.1608.
20. Sabalpara DM, Parikh S, Parikh B. Histopathological study of prostatic lesions. Natl J Integr Res Med. 10:58-63. Available from: https://nicpd.ac.in/ojs-/index.php/njirm/article/view/2590. [Last accessed on 2024 Jan 24].
21. Sharma A, Sharma M, Gandhi S, Khajuria A, Goswami KC. Histomorphological spectrum of prostatic lesions: A retrospective analysis of transurethral resection of prostate specimens. Int J Res Med Sc 2017;5:2373-8. 10.18203/2320-6012.ijrms20172095.
22. Garalla HM, Darraz KMA, Essa MMA. A retroprospective clinicopathological study of prostatic lesions in surgical specimens. Libyan Int Med Univ J 2022;7:51-6. 10.1055/s-0042-1759622.
23. Bhatta S, Hirachan S. Prostatic lesions: Histopathological study in a tertiary care hospital. JMMIHS 2018;4:12-9.
24. Amin SM, Jibrin PG, Olah FG, Oguntebi E, Abubakar ND, Lawal IO. Clinicopathological analysis of prostatic lesions in a tertiary hospital in Nigeria. Ann Trop Pathol 2020;11:25-8. 10.4103/atp.atp_14_19.
25. Bosland MC, Shittu OB, Ikpi EE, Akinloye O. Potential new approaches for prostate cancer management in resource-limited countries in Africa. Ann Glob Health 2023;89:14.
26. Egevad L, Delahunt B, Furusato B, Tsuzuki T, Yaxley J, Samaratunga H. Benign mimics of prostate cancer. Pathology 2021;53:26-35.
27. Verhoef EI, van Cappellen WA, Slotman JA, Kremers GJ, Ewing-Graham PC, Houtsmuller AB, *et al.* Three-dimensional architecture of common benign and precancerous prostate epithelial lesions. Histopathology 2019;74:1036-44.