

**A Study Assessing Histopathological Features and Classify Tumors of Prostate as Per Recommendations of WHO: An Observational Study**Sawan Kumar<sup>1</sup>, Ankit Gaba<sup>2</sup>, Rajeev Bhardwaj<sup>3</sup><sup>1</sup>Assistant Professor, Department of Pathology, Lord Buddha Koshi Medical College and Hospital, Saharsa, Bihar, India<sup>2</sup>Associate Professor, Department of Pathology, Shree Narayan Medical Institute and Hospital, Saharsa, Bihar, India<sup>3</sup>Assistant Professor, Department of Pathology, Lord Buddha Koshi Medical College and Hospital, Saharsa, Bihar, India

Received: 10-05-2023 / Revised: 16-06-2023 / Accepted: 23-07-2023

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Conflict of interest: Nil

**Abstract****Aim:** The aim of the present study was to assess the incidence, age distribution, gross and histopathological features of lesions of prostate and to classify tumours of prostate as per recommendations of WHO.**Methods:** An observational study conducted in the Department of Pathology for the period of 2 years and 100 patients were included in the study.**Results:** Most prostatic samples were benign (90%), whereas 9% were cancerous. Our analysis found 1 case of Prostatic Intra-epithelial Neoplasia (PIN). BPH was most common in those aged 61–70, with 39 cases (39%). Six BPH patients (6%) had chronic prostatitis and two (2%) had acute prostatitis. Three BPH patients (3%) had basal cell hyperplasia and one had squamous metaplasia. We recorded 10 prostate cancer cases utilizing modified Gleason Grading. Gleason's score 7 was the most common in 3 of 10 adenocarcinomas.**Conclusion:** Adenocarcinoma was most common in those aged 61–70. All prostate biopsies must be examined for premalignant abnormalities, proliferative activity, and inflammation. Successful prostate cancer therapy depends on histology detection and classification.**Keywords:** Needle core biopsy, TURP, benign prostatic hyperplasia, prostate carcinoma.

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**Introduction**

The prostate gland in a typical adult is a glandular organ with a pear-shaped structure, weighing around 20 grams. It functions as an exocrine gland and constitutes a substantial portion of seminal fluid. In adult individuals, the prostatic parenchyma may be categorized into four separate zones or areas based on biological and anatomical characteristics: peripheral, central, transitional, and periurethral zones. The histological composition of the structure comprises glands that are bordered by basal cuboidal cells and inner secretory columnar cells, forming a double layer. [1] A significant proportion of the patients exhibit symptoms associated with both urinary retention and urinary overflow. The disorders that often impact the prostate include Benign Prostatic Hyperplasia, Prostate Carcinoma, and Prostatitis [2], which are regularly seen in clinical practice. Two Prostate pathological lesions have a higher incidence rate beyond the age of 50 years and represent a substantial contributor to both morbidity and death among men of increasing age.<sup>3,4</sup> The prevalence of

prostatic lesions rises as individuals age, with 8% occurring in the fourth decade, followed by 50% in the fifth decade, and a significant increase to 75% in the eighth decade. Patients often exhibit signs of urine dribbling/incontinence, hesitation, urinary retention, and in rare cases, hematuria. [5] The prostate gland is primarily affected by three pathological processes: inflammation (prostatitis), benign prostatic hyperplasia (BPH), and cancers (pre-malignant and malignant lesions). Among these three conditions, benign prostatic hyperplasia (BPH) is the most prevalent and frequently manifests in older individuals, prompting the perception of it as a typical aspect of the aging process. Prostatic carcinoma (CaP) is a very prevalent condition in males. Prostatitis may manifest as either acute or chronic bacterial prostatitis, chronic abacterial prostatitis, or granulomatous prostatitis. [2] The presence of BPH may be seen in about 20% of men by the age of 40, which rises to 70% by the age of 60 and further climbs to 90% by the age of 80 years. [6] The

histologic characteristics of BPH are influenced by the abundance of testosterone and its metabolite, dihydrotestosterone. [7]

The objective of the research was to examine the occurrence, age distribution, physical characteristics, and histological aspects of prostate lesions. Additionally, the study aimed to categorize prostate tumors based on the guidelines provided by the World Health Organization (WHO) and analyze instances of prostate adenocarcinoma using the Modified Gleason grading system.

### Material & Methods

An observational study conducted in the Department of Pathology, Lord Buddha Koshi Medical College and Hospital, Saharsa, Bihar, India for the period of 2 years and 100 patients were included in the study.

**Inclusion Criteria:** All Prostate Specimens received during study period were included in the study.

**Exclusion Criteria:** Patients not giving consent were excluded from the study.

105 specimens were received over the course of the investigation. 5 specimens were excluded based on

the above-mentioned exclusion criteria due to insufficient biopsies and poor preservation. As a result, the current investigation comprised a total of 100 prostatic specimens.

Age of patients, presenting symptoms, Digital Rectal Examination (DRE) findings, and pertinent tests such as serum PSA levels, USG, and clinical diagnosis were all taken down from the case records. The gross specimens received were of needle core biopsies and transurethral resection of prostate (TURP) chips. The received specimens were fixed in 10% neutral buffered formalin solution and routine paraffin processing followed by hematoxylin and eosin staining was done. All the specimens were analysed according to age, medical history, histopathological pattern and final diagnosis. Thorough examination of slides was done under light microscope. Various lesions of prostate were listed, diagnosed according to various histopathological patterns and were classified with reference to age. Following histopathologic assessment, the tumors were classified according to WHO recommendation, and histologic grading was done using modified Gleason's system.

### Results

**Table 1: Age wise distribution of cases**

Age (years)	Benign	Malignant	PIN
41-50	12	1	-
51-60	25	1	-
61-70	36	3	01
71-80	10	3	-
81-90	7	1	-
Total	90	9	01

All prostatic specimens were broadly classified into benign 90 (90%) and malignant 9 (9%). We reported 1 (1%) case of Prostatic Intra-epithelial Neoplasia (PIN). Maximum cases of BPH 39 (39%) were seen in the 61-70 years age group.

**Table 2: Distribution of Histopathological lesions**

Histopathological pattern	Number of cases	Percentage(%)
BPH alone	70	70
BPH with acute prostatitis	2	2
BPH with chronic prostatitis	6	6
Stromal hyperplasia only	2	2
BPH with squamous metaplasia	1	1
BPH with basal cell hyperplasia	3	3
Benign prostatic tissue	5	5
Prostatic intraepithelial Neoplasia (PIN)	1	1
Adenocarcinoma of Prostate	10	10

Cases of BPH with co-existing chronic prostatitis were 6 (6%) and that with acute prostatitis were 2 (2%). Less frequent findings were BPH with basal cell hyperplasia 3 (3%) and BPH with squamous metaplasia 1 (1%). We reported 10 cases of adenocarcinoma prostate with modified Gleason Grading system.

**Table 3: Distribution of cases according to Gleason's score**

Gleason's score	Number of cases	Percentage (%)
6	1	10
7	3	30
8	2	20
9	2	20
10	2	20
Total	10	100

The most common score obtained was Gleason's score 7 in 3 cases out of the total 10 adenocarcinoma cases.

### Discussion

The cause of prostatic cancer is largely unknown, and preventing the disease is difficult. Genetic factors contribute to this phenomenon. [8,9] The large differences in the incidence of clinically apparent carcinoma imply that dietary and environmental variables may possibly have a role in the disease's genesis and progression. [10] Moore's term "Nodular Hyperplasia" is a more exact designation than the popular moniker BPH. The nodular expansion of the gland is a consequence of hyperplasia occurring in both the glandular and stromal components. In NH, there is excessive growth of the epithelium and fibromuscular stroma in the transition zone and periurethral region. [11] Nodular hyperplasia is a very prevalent condition among the elderly. Beginning in the fourth decade of life, the prevalence of NH rises rapidly, reaching approximately 100% frequency by the ninth decade. The age-specific frequency exhibits a remarkable degree of comparability across populations worldwide. [12]

The prostatic specimens were categorized into two main groups: benign, accounting for 90% of the samples, and malignant, comprising 9%. A single instance (1%) of Prostatic Intra-epithelial Neoplasia (PIN) was identified in our study. Maximum cases of BPH 39 (39%) were seen in the 61-70 years age group similar to Matapurkar et al. [13] Malignant lesions were encountered predominantly in age group 61-70 years that are similar to Sharma et al. [14] We reported 1 (1%) case of Prostatic Intra-epithelial Neoplasia (PIN) similar to Neha Angurana's study [15] (50.5%), followed by prostatic adenocarcinoma (14.4%) and BPH with chronic prostatitis (6.9%). While in stromal pattern, the sections showed more stromal elements than glands or were made up almost entirely of stromal elements. Before the identification of the hyperplastic nature of BPH, the prostatic enlargement in older men has been variably believed to indicate neoplastic development, compensatory hypertrophy, a reaction to inflammation or arteriosclerosis. [16]

Pure stromal hyperplasia with nodule formation was first reported by Reischauer in 1925. [17]

Cases of BPH with co-existing chronic prostatitis were 6 (6%) and that with acute prostatitis were 2 (2%). Less common observations were BPH with basal cell hyperplasia 3 (3%) and BPH with squamous metaplasia 1 (1%). We reported 10 instances of adenocarcinoma prostate using modified Gleason Grading system. Maximum number of cases was found in 7th decade which was comparable to research performed by Sharma et al [14] and Matapurkar et al. [13] We reported 14 instances of adenocarcinoma prostate using modified Gleason Grading system. The most frequent score obtained was Gleason's score 7 in 3 instances out of the total 10 adenocarcinoma cases.

### Conclusion

We found that 61-70-year-olds had greater prostatic lesions. Malignancies are rarer than benignities. Histologically, BPH is the most common prostatic lesion, followed by BPH with prostatitis. Prostate biopsies must be carefully reassessed to detect premalignant abnormalities, proliferative activity, and inflammation. Modified Gleason's method should be used to control prostate cancer. Histopathological diagnosis and grading contribute to prostate cancer treatment. High knowledge of advancements and teamwork are essential for patient management.

### References

1. Rosai J. Rosai and Ackerman's surgical pathology e-book. Elsevier Health Sciences; 2011 Jun 20.
2. Cotran RS, Kumar V, Collins T. Robbins pathologic basis of disease. In Robbins pathologic basis of disease 1999 (pp. xv-1425).
3. Sumaya, Das M, Nagesha KR. Spectrum of histopathological lesions of prostate in a tertiary care center. Int J Clin Diagn Pathol. 2020; 3(1):110-3.
4. Yelave R, Shahnaaz Z, Pawar V. Histopathological study of prostatic lesions in a tertiary care hospital. J Diagn Pathol Oncol. 2020; 5(2):200-7
5. Shah R, Karki S, Shah N, Dhakal S, Singh SK, Chaudhari RK. Histopathological Study of Prostatic Diseases in BPKIHS, Nepal: A Hospital Based Study.

6. Kumar V, Abbas AK, Fausto N, editors. Prostate. In: Robbins Pathologic Basis of Disease. 8th ed. Philadelphia: Saunders Company; 1999.
7. Bartsch G, Rittmaster RS, Klocker H. Dihydrotestosterone and the concept of 5 $\alpha$ -reductase inhibition in human benign prostatic hyperplasia. *European urology*. 2000 Mar 17; 37(4):367-80.
8. Carter BS, Bova GS, Beaty TH, Steinberg GD, Childs B, Isaacs WB, Walsh PC. Hereditary prostate cancer: epidemiologic and clinical features. *The Journal of urology*. 1993 Sep;150(3):797-802.
9. Woolf CM. An investigation of the familial aspects of carcinoma of the prostate. *Cancer*. 1960 Jul;13(4):739-44.
10. Carter HB, Piantadosi S, Isaacs JT. Clinical evidence for and implications of the multistep development of prostate cancer. *The Journal of urology*. 1990 Apr 1;143(4):742-6.
11. Rosai J. Male reproductive system. In: Rosai & Ackerman's. *Surgical Pathology*. 9th ed. Vol.1, Missouri: Mosby; 2004:1361-1412.
12. Bostwick DG, Amin MB. Male reproductive system. In: Damjanov I, Linder J, editors. *Anderson's pathology*. Vol.1, 10th Ed., Missouri: Mosby; 1996.p.2197-2222.
13. Matapurkar BG, Taneja OP. Incidence of carcinoma prostate. *Ind. J of Cancer*, 1969, 172-182.
14. Sharma GC, Mathur SC, Sharma ML. Occult carcinoma in benign hypertrophy of prostate (Clinicopathological study of 100 cases). *Ind. J Surg.*, 1972, 152-155.
15. Neha Angurana. Pattern of prostate diseases-a histopathological study in jammu, *international journal of basic and applied medical sciences* ISSN: 2277-2103 (online)
16. Mostofi FK. Benign hyperplasia of the prostate gland. In Campbell MF, Harrison JH eds. *Urology*, 3rd ed. Philadelphia: WB Saunders, 1970, 10-65.
17. Peterson RO, Sesterhenn IA, Davis CJ. Editors. Prostate. In: *Urologic Pathology*. Lippincott Williams and Wilkins, 2009, 451-559.