

A Retrospective Histopathological Evaluation of Prostatic Lesions and Assessing Adenocarcinoma of Prostate According to Modified Gleason Grading System

Anil Kumar

Assistant Professor, Department of Pathology, Krishna Mohan Medical College and Hospital, Mathura, U.P. , India

Received: 14-05-2022 / Revised: 11-06-2022 / Accepted: 29-07-2022

Corresponding author: Dr. Anil Kumar

Conflict of interest: Nil

Abstract:

Aim and objectives: The aim of the present study was to assess histopathological features of lesions of prostate and to classify tumours of prostate as per recommendations of WHO and to analyse cases of Adenocarcinoma of prostate according to Modified Gleason grading system.

Methods: An observational study conducted in the Department of Pathology for the period of one year and 200 patients were included in the study.

Results: All prostatic specimens were broadly classified into benign 180 (90%) and malignant 19 (9.5%). We reported 1 (0.5%) case of Prostatic Intra-epithelial Neoplasia (PIN). Maximum cases of BPH 80 (40%) were seen in the 61-70 years age group. Cases of BPH with co-existing chronic prostatitis were 12 (6%) and that with acute prostatitis were 6 (3%). Less frequent findings were BPH with basal cell hyperplasia 6 (3%) and BPH with squamous metaplasia 2 (1%). We reported 19 cases of adenocarcinoma prostate with modified Gleason Grading system.

Conclusion: Maximum number of cases of adenocarcinoma was seen in 61-70 years age group. It is necessary to study all prostate biopsies in order to identify premalignant lesions, proliferative activity and grade of inflammation. Histopathological diagnosis and grading plays a definitive role in the management of prostatic carcinoma.

Keywords: Needle core biopsy, TURP, benign prostatic hyperplasia, prostate carcinoma

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

In normal adult prostate is a pear - shaped glandular organ that weighs up to 20gms. [1] It is an exocrine gland and forms a significant component of seminal fluid. In adult, prostatic parenchyma can be divided into four biologically and anatomically distinct zones or regions: peripheral, central, transitional and periurethral zones. Histologically it consists of glands lined by basal cuboidal cells and inner secretory columnar cells (double layered).[1] Most of

the patients present with complaints related to micturition and incontinence. Of the diseases which affects the prostate, the most frequently encountered in clinical practice are Benign Prostatic Hyperplasia, Carcinoma of prostate and prostatitis.[2] Pathological lesions of the prostate occur more frequently after the age of 50 years and constitute a significant cause of morbidity and mortality in males [3] of advancing age. The incidence of prostatic

lesions increases with advancing age with 8% occurring during 4th followed by 50% in the 5th decade and rising dramatically to 75% in the 8th decade. Patients usually present with symptoms of urinary dribbling/incontinence, hesitancy, urinary retention,[4] and rarely hematuria.[5] Three pathologic processes mainly affect the prostate gland: Inflammatory (prostatitis), benign prostatic hyperplasia (BPH), and tumors (pre-malignant and malignant lesions). Of these three, BPH is the most common and occurs so often in an advanced age that they can almost be construed as a “normal” aging process. Prostatic carcinoma (CaP) is also an extremely common lesion in men. Prostatitis may be acute or chronic bacterial prostatitis, chronic abacterial prostatitis, or granulomatous prostatitis.[2] Histologic evidence of BPH can be seen in approximately 20% of men by 40 years of age, a figure that increases to 70% by age 60 and to 90% by age 80.[6] The development of the histologic features of BPH is dependent on the bioavailability of testosterone and its metabolite, dihydrotestosterone.[7]

Hence, the aim of study was to study the incidence, age distribution, gross and histopathological features of lesions of prostate and to classify tumours of prostate as per recommendations of WHO and to analyse cases of Adenocarcinoma of prostate according to Modified Gleason grading system.

Material & Methods

An observational study conducted in the Department of Pathology Department of Pathology, Krishna Mohan Medical college and Hospital, Mathura, UP, India from December 2018 November 2019 to and 200 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.

Methodology

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	24 (13.33%)	02 (10%)	-
51-60	50 (27.77%)	03 (15%)	-
61-70	72 (40%)	07 (35%)	01
71-80	20 (11.11%)	06 (30%)	-
81-90	14 (7.77%)	01 (5%)	-
Total	180 (100%)	19 (100%)	01

All prostatic specimens were broadly classified into benign 180 (90%) and malignant 19 (9.5%). We reported 1 (0.5%) case of Prostatic Intra-epithelial Neoplasia (PIN). Maximum cases of BPH 80 (40%) were seen in the 61-70 years age group.

Table 2: Distribution of Histopathological lesions

Histopathological pattern	Number of cases	Percentage (%)
BPH alone	140	70
BPH with acute prostatitis	6	3
BPH with chronic prostatitis	12	6
Stromal hyperplasia only	4	2
BPH with squamous metaplasia	2	1
BPH with basal cell hyperplasia	6	3
Benign prostatic tissue	11	5.5
Prostatic intraepithelial Neoplasia (PIN)	1	1
Adenocarcinoma of Prostate	19	9.5

Cases of BPH with co-existing chronic prostatitis were 12 (6%) and that with acute prostatitis were 6 (3%). Less frequent findings were BPH with basal cell hyperplasia 6 (3%) and BPH with squamous metaplasia 2 (1%). We reported 19 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	2	10.52
7	7	36.84
8	5	26.31
9	3	15.78
10	2	10.52
Total	19	100

The most common score obtained was Gleason's score 7 in 7 cases out of the total 19 adenocarcinoma cases.

Discussion

The aetiology of prostatic cancer is mostly unclear, disease prevention is challenging. Hereditary factors play a part in this.[8,9] The wide variations in the prevalence of clinically evident carcinoma suggest that nutritional and environmental factors may

potentially play a role in the disease's development and progression.[10] Moore's word "Nodular Hyperplasia" is a more precise designation than the common name BPH. Hyperplasia of both glandular and stromal components results in a nodular expansion of the gland. The epithelium and fibromuscular stroma in the transition zone and periurethral region overgrow in NH.[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. In populations all around the world, the age-specific frequency is strikingly comparable.[12]

Maximum cases of BPH 41 (41%) 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] All prostatic specimens were broadly classified into benign 180 (90%) and malignant 19 (9.5%). We reported 1 (0.5%) case of Prostatic Intra-epithelial Neoplasia (PIN). Maximum cases of BPH 80 (40%) were seen in the 61-70 years age group. We reported 1 (0.5%) 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 recognition of the hyperplastic nature of BPH, the prostatic enlargement in elderly men had been variously interpreted to reflect neoplastic process, compensatory hypertrophy, a response 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 12 (6%) and that with acute prostatitis were 6 (3%). Less frequent findings were BPH with basal cell hyperplasia 6 (3%) and BPH with squamous metaplasia 2 (1%). We reported 19 cases of adenocarcinoma prostate with modified Gleason Grading system. Maximum number of cases was reported in 7th decade which is similar to studies conducted by Sharma et al[14] and Matapurkar et al.[13] We reported 14 cases of adenocarcinoma prostate with modified Gleason Grading system. The most common predominant grades observed in

this study were grade 3 and grade 4 was comparable to The most common score obtained was Gleason's score 7 in 7 cases out of the total 19 adenocarcinoma cases.

Conclusion

We concluded that prostatic lesions are more common in age group of 61-70 years. Benign conditions are more common than malignant conditions. Among the histological types of prostatic lesions, BPH is predominant type, followed by BPH with prostatitis. It is necessary to reassess periodically all prostate biopsies carefully in order to identify premalignant lesions, proliferative activity, grade of inflammation. Efforts should be made to apply modified Gleason's system in case of adenocarcinoma of prostate to improve management. Histopathological diagnosis and grading plays an important role in the management of prostatic cancer. For satisfactory management of patient, a high degree of the awareness of the advances along with team approach has become imperative.

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 Pa thol. 2010; 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. 2010; 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 5α -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 prostrate 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.