IJBC 2022; 14(1): 18-22

Iranian Journal of Blood & Cancer

Journal Home Page: www.ijbc.ir

ORIGINAL ARTICLE

Clinical and Pathological Features of Iraqi Patients with Prostate Cancer

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ARTICLE INFO

Article History:
Received: 03.11.2021
Accepted: 17.02.2022

Keywords:
Prostate
Phosphatase
Cancer
Immunometric assay
Enzyme activity

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ABSTRACT

Background: The clinical pathological features were detected by measuring the prostatic acid phosphatase (PAP) activity according to the stages and grades of prostate cancer. We also explored the relationship between the disease and age with family history among the patients.
Methods: Data were collected from direct interview and from the patient's records. Blood samples that collected before the surgery from Baghdad hospitals. By using the ELISA device, PAP enzyme activity was determinred using Automated Chemiluminescent Immunometric Assay.
Result: Older individuals were more likely to develop prostate cancer since out of the 50 samples collected, most (46%) were ≥60 years old. 23 (46%) cases were in stage II of PCa and 29 (58%) had grade-II as the most dominant stage and grade. Family history from parents to sons had a significant role in the possibility of being positive for prostate cancer. A positive family history was found in 72% of the cases of PCa, and it was found that the enzymatic activity of the PAP enzyme rises in the presence of prostate cancer.
Conclusion: PAP enzyme is reliable in diagnosing the presence of prostate cancer, as the increase in the activity of the enzyme PAP coincides with the presence of prostate cancer.

Please cite this article as: Hussein MM, Mohammed HS. Clinical and Pathological Features of Iraqi Patients with Prostate Cancer. IJBC 2022; 14(1): 18-22.

Introduction

The prostate is a six-part integration of fibroglandular muscular tissue. The prostate gland secretes and produces an alkaline fluid, which activates and protects the sperm during ejaculation.[1-3] The size of the prostate generally increases, through life.[4, 5] Commonly the prostate alters and enlarges with increasing age.[6-8] Structural alteration of prostate malignant cells in comparison to normal or hypertrophic prostate cells are recognized and mentioned by many academics.[9] Understanding developmental features of prostate is beneficial for the management and diagnosis of early stage prostatitis and benign prostatic hyperplasia (BPH). Prostate cancer (PCa) and BPH are the most prevalent kinds of prostate disease.[10, 11] BPH refers to a benign expansion of the prostate induced by hyperplasia in the transitional zone.[12] BPH is associated with increasing age, in roughly 70% and 90% of men aged 70 and 80 years, respectively, and about 50% of men aged 50 years have being influinced.[13, 14] Aging, decreased testicular function, metabolic syndrome, family history of BPH and obesity are risk factors for BPH.[13, 15, 16] With the fifth highest fatality, PCa is the second most commonly diagnosed cancer in the males .[17-19] It is a diverse disease that can grow slowly with a long natural history and results in mortality despite therapy.[20, 21] The PCa incidence and its correlated deaths change excessively in reference to the ethnic or race. This correlation is due to the interaction of socioeconomic elements, exposure to the environmental factors, and epigenetic and biologic circumstances.[22-24] PCa is the highest diagnosed malignancy between men in the United States, and survivors of prostate cancer may keep going to be at risk of death up to 15 years beyond diagnosis.[25, 26] Human

[DOI: 10.58209/ijbc.14.1.18]

prostatic acid phosphatase (100 KD, PAcP; E.C.3.1.3.2) is produced in lysosomes of prostate epithelial cells found in high quantity in seminal fluid.[27, 28] This enzyme is found in two subunits with molecular weight of about 50 kDa for both.[29] The cellular shape (cPAP, highly expressed in the prostate cells) and the secretory shape (sPAP, expressed only in the prostate and released into the seminal fluid).[30] Actually, serum PAcP serves as a considerable warning element and biochemical marker of clinical recurrence of PCa. Moreover, a recent research emphasized that PAcP represents an indicator of progression of tumor.[31]

Materials and Methods

All blood samples were collected using a conventional phlebotomy technique. Blood samples were taken before the surgery for the purpose of measuring the enzymatic activity of people who suffer from prostate tumors. Patients suffering from liver and pancreatic diseases and those with a history of other types of malignancies were excluded. Blood was collected without anticoagulant in a serum separator vacutainer and was allowed to coagulate for 20 to 30 minutes at room temperature. Sera were separated by centrifugation and immediately all specimens were aliquoted, then stored at -70°C until batch processing.

Apparatus and Method Description

Tests were performed by ELISA according to the method; Automated Chemiluminescent Immunometric Assay. The patient's samples were added to a solid phase which is coated with a mouse monoclonal antibody particular for PAP. To construct an antibody sandwich complex, a goat-anti-PAP-alkaline phosphatase conjugate is added. Washing was used to eliminate any extra conjugate and an adamantyl dioxetane phosphate substrate was added to make chemiluminescence. Light emission was commensurate with PAP concentration in the specimen. The chemicals used in this analytical reagent were purchased from Mayo Clinic Laboratories Rochester, United States.

Results

46% of the 50 patients with PCa were ≥60 years old, while out of 50 samples with BPH, 40% were ≥60 years (Table 1). The results of this study showed positive family history for PCa in 72% and 64% in BPH cases (Table 2).

The results illustrated that most patients with PCa were in stage II (46%) and 58% of them had tumors graded as grade II (Tables 3-7).

Table 1: Age range of patients with PCa

Age range (Year)	Number of PCa	%	Number of BPH	%	Number of control cases	%
20-29	0	0	2	4	3	6
30-39	2	4	7	14	7	14
40-49	10	20	9	18	8	16
50-59	15	30	12	24	12	24
≥60	23	46	20	40	20	40
Total	50	100	50	100	50	100

P<0.001

Table 2: Family history of patients with PCa and BPH

Family history	No. of PCa	Percentage %	No. of BPH	Percentage %
Yes	36	72	32	64
No	14	28	18	36
Total	50	100	50	100

P=0.003

Table 3: Classification of prostate tumor stages

Stage of tumor	No. of cases	%
I	5	10
II	23	46
III	13	26
IV	9	18
Undetected	-	-
Total	50	100

P<0.001

Table 4: Classification of prostate cancer grade by grade of tumoral cells

Tumor grade	Number of cases	%
1	5	10
2	29	58
3	16	32
Total	50	100

P<0.001

[DOI: 10.58209/ijbc.14.1.18]

Table 5: Enzyme activity in patients with PCa and control group

Enzyme Activity	No.	Mean ± Std	Std.Error	P value
Pca	50	5.89±3.25	0.459	P<0.001
Control	50	0.44±0.22	0.031	

Table 6: Enzyme activity in patients with BPH and control group

Enzyme Activity	No.	Mean±Std	Std.Error	P value
BPH	50	4.55±2.15	0.459	P<0.001
Control	50	0.44±0.22	0.304	

Table 7: Enzyme activity for samples of PCa and BPH

Enzyme Activity	No.	Mean±Std	Std.Error	P value
Pca	50	5.89±3.25	0.459	P=0.005
BPH	50	4.55±2.15	0.459	

Discussion

This study showed that 23 patients with PCa were ≥60 years. Its incidence rises with age over 65 years.[32-35] Estimates from this study were positive family history in cases of PCa (72%) and BPH (64%) because of common genes and specific environmental carcinogens exposures and shared lifestyle manners which is consistent with a previous study.[36-39] Cancer grade and stage are important factors to identify and follow up prostate cancer treatment. As shown in this research, 46% of cases of PCa were in stage II and pathological grade II was reported in 58%. These results were compatible with In 1938 was first mentioned by Gutman that the rise of serum PAP were observed in patients with prostate cancer. The hypotheses of this research adopt the same as what was had found in previous studies.[40-45] We found an excessive increase in the activity of the PAP enzyme in patients with PCa compared to patients with BPH. which in turn we found an increase in the concentration and activity of the PAP enzyme compared to the control group of healthy person and this is according to what was shown in the tables above.

Conclusion

PAP enzyme is reliable in diagnosing the presence of prostate cancer, as the increase in the activity of the enzyme PAP coincides with the presence of prostate cancer, which occurs commonly in men of advanced ages, and that family history is important to warn prostate cancer because of a close relationship between family history and the presence of cancer.

Conflict of Interest: None declared.

References

1. Prashanth Anamthathmakula and Wipawee Winuthayanon Mechanism of semen liquefaction and its potential for a novel non-hormonal contraception†2020 Aug; 103(2): 411–426.Published online 2020 May 14.
2. Verze P, Cai T, Lorenzetti S. The role of the prostate in male fertility, health and disease. Nat Rev Urol 2016; 13:379–386.
3. Drabovich AP, Saraon P, Jarvi K, Diamandis EP. Seminal plasma as a diagnostic fluid for male reproductive system disorders. Nat Rev Urol 2014; 11:278–288.
4. Kim EH, Larson JA, Andriole GL (2016). "Management of Benign Prostatic Hyperplasia". AnnualReviewofMedicine (Review).
5. Üçer O (1 December 2011). "Giant prostatic hyperplasia: Case report and literature review". Dicle Medical Journal / Dicle Tıp Dergisi. 38 (4): 489–491.
6. Berry SJ, Coffey DS, Walsh PC, Ewing LL. The development of human benign prostatic hyperplasia with age. J Urol. 1984;132:474–9.
7. Xia SJ, Xu XX, Teng JB, Xu CX, Tang XD. Characteristic pattern of human prostatic growth with age. Asian J Androl. 2002;4:269–71.
8. McNeal JE. The zonal anatomy of the prostate. Prostate. 1981;2:35 49.
9. Korček, M., Sekerešová, M., Makarevich, A. V., Gavurová, H., Olexíková, L., Pivko, J., Barreto, L."Morphological and functional alterations of the prostate tissue during clinical progression in hormonally naïve, hormonally treated and castration resistant patients with metastatic prostate cancer". Oncology Letters 20.5 (2020): 201.
10. Shi-Jun Zhang,Hai-Ning Qian, Yan Zhao, Kai Sun, Hui-Qing Wang, Guo-Qing Liang,Feng-Hua Li, and Zheng LiRelationship between age and prostate sizeAsian J Androl. 2013 Jan; 15(1): 116–120. Published online 2012 Dec 10
11. Emberton M, Andriole GL, de la Rosette J, Djavan B, Hoefner K, et al. Benign prostatic hyperplasia: a progressive disease of aging men. Urology. 2003;61:267–73.
12. Yiyu Huang , Jiaxin LI , Shan Yang , Daozhang Yuan and Shusheng Wang ,Efficacy and safety of transurethral split of prostate for benign prostatic hyperplasia: a meta-analysis Huang et al. BMC Urology (2020) 20:141.
13. Skinder D, Zacharia I, Studin J, Covino J. Benign prostatic hyperplasia. J Am Acad Physician Assist. 2016;29(8):19–23.
14. McVary KT. BPH: epidemiology and comorbidities.

[DOI: 10.58209/ijbc.14.1.18]

Am J Manag Care. 2006;12(5 Suppl):S122–8.
15. Luigi Cormio, ,Beppe Calò, Ugo Falagario, Manuela Iezzi, Alessia Lamolinara, Paola Vitaglione, Giovanni Silecchia, Giuseppe Carrieri, Vincenzo Fogliano, Stefano Iacobelli, Pier Giorgio Natali & Mauro Piantelli Improvement of urinary tract symptoms and quality of life in benign prostate hyperplasia patients associated with consumption of a newly developed whole tomato-based food supplement: a phase II prospective, randomized double-blinded, placebo-controlled studyJournal of Translational Medicine volume 19, Article number: 24 (2021)
16. Christopher J McNally, Mark W Ruddock, Tara Moore, and Declan J McKenna Biomarkers That Differentiate Benign Prostatic Hyperplasia from Prostate Cancer: A Literature ReviewCancer Manag Res. 2020; 12: 5225–5241.Published online 2020 Jul.
17. Bosch JL, Tilling K, Bohnen AM, Bangma CH, Donovan JL. Establishing normal reference ranges for prostate volume change with age in the population-based Krimpen-study: prediction of future prostate volume in individual men. Prostate.
18. Loeb S, Kettermann A, Carter HB, Ferrucci L, Metter EJ, et al. Prostate volume changes over time: results from the Baltimore Longitudinal Study of Aging. J Urol. 2009;182:1458–62.
19. Ferlay J, Soerjomataram I, Ervik M, et al. GLOBOCAN 2012 v1.0, Cancer Incidence and Mortality Worldwide: IARC CancerBase No. 11. International Agency for Research on Cancer, Lyon. 2013. http:// globocan.iarc.fr. Accessed 24 May 2018.
20. DeVita VT Jr, Lawrence TS, Rosenberg SA (eds): Cancer of the prostate, in Cancer: Principles and Practice of Oncology (ed 11). Philadelphia, PA, Wolters Kluwer, 2019, 1087.
21. James A. Eastham, MD ; Glenn Heller, PhD ; Susan Halabi, PhD ; J. Paul Monk III, MD ; Himisha Beltran, MD; Martin Gleave, MD, Cancer and Leukemia Group B 90203 (Alliance): Radical Prostatectomy With or Without Neoadjuvant Chemohormonal Therapy in Localized, High-Risk Prostate Cancer; Journal of Clinical Oncology > List of Issues > Volume 38, Issue 26 > ORIGINAL REPORTS Genitourinary Cancer ... J Clin Oncol. 2020 Sep 10;volume38(26):3042-3050.
22. Dess RT, Hartman HE, Mahal BA, et al. Association of black race with prostate cancer-specific and other-cause mortality. JAMA Oncol 2019;5:975-983.
23. Mahal BA, Alshalalfa M, Spratt DE, et al. Prostate cancer genomic-risk differences between African-American and white men across Gleason scores. Eur Urol 2019;75:1038-1040.
24. Brandon A. Mahal, M.D ,Mohammed Alshalalfa, Ph.D. Kevin H. Kensler, Sc.D. Ilkania Chowdhury-Paulino, B.A. Racial Differences in Genomic Profiling of Prostate Cancer The New England Journal of Medicine Downloaded from nejm.org on August 24, 2021. For personal use only. No othN Engl J Med2020;383:1083-1085.
25. Alyssa N. Troeschel, MPH, Terryl J. Hartman, PhD, MPH, RD, Eric J. Jacobs, PhD, Victoria L. Stevens, PhD, Ted Gansler, MD, MPH," Postdiagnosis Body Mass Index, Weight Change, and Mortality From Prostate Cancer, Cardiovascular Disease, and All Causes Among Survivors of Nonmetastatic Prostate Cancer" J Clin Oncol. 22020 Jun 20; 38(18): 2018–2027. Published online 2020 Apr 6.
26. Husson O, van Steenbergen LN, Koldewijn EL, et al. : Patients with prostate cancer continue to have excess mortality up to 15 years after diagnosis. BJU Int 114:691-697, 2014.
27. Muniyan, Sakthivel; Chaturvedi, Nagendra K.; Dwyer, Jennifer G.; LaGrange, Chad A.; Chaney, William G.; and Lin, Ming-Fong, "Human prostatic Acid phosphatase: structure, function and regulation." (2013). Journal Articles: Biochemistry & Molecular Biology. Paper 46
28. P. Vihko, M. Kontturi, and L. K. Korhonen, "Purification of human prostatic acid phosphatase by affinity chromatography and isoelectric focusing. Part I," Clinical Chemistry, vol. 24, no. 3, pp. 466–470, 1978.
29. Luchter-Wasyl E., Ostrowski W. Subunit structure of human prostatic acid phosphatase. Biochim. Biophys. Acta. 1974;365:349–359.
30. M. F. Lin, M.-S. Lee, X.-W. Zhou et al., "Decreased expression of cellular prostatic acid phosphatase increases tumorigenicity of human prostate cancer cells," The Journal of Urology, vol. 166, no. 5, pp. 1943–1950, 2001.
31. Taira A, Merrick G, Wallner K, Dattoli M. Reviving the acid phosphatase test for prostate cancer. Oncology. 2007; 21:1003–10.
32. Ferlay J EM, Lam F, Colombet M, Mery L, Pineros M, Znaor A, Soerjomataram I. et al. Global cancer observatory: cancer today. Lyon, France: International Agency for Research on Cancer. Available from: https://gco.iarc.fr/today, Accessed 02 February 2019. [Internet]
33. Perdana NR, Mochtar CA, Umbas R, Hamid AR. The Risk Factors of Prostate Cancer and Its Prevention: A Literature Review. Acta Med Indones. 2016;48(3):228–238.
34. SEER Cancer Statistics Review, 1975-2013 [Internet]. National Cancer Institue, Bethesda, MD. 2016. Available from: https://seer.cancer.gov/csr/1975_2015/. Accessed 04 February 2019. [Internet]. SEER, 2018.
35. Moyad, M.A.; Newton, R.U.; Tunn, U.W.; Gruca, D. Integrating diet and exercise into care of prostate cancer patients on androgen deprivation therapy. Res. Rep. Urol. 2016, 8, 133–143.
36. Rawla, P. Epidemiology of Prostate Cancer. World J. Oncol. 2019, 10, 63–89
37. Carroll PR, Grossfeld GD, editors. Prostate cancer. Hamilton, London: Decker Inc.; 2002.
38. Sridhar G, Masho SW, Adera T, Ramakrishnan V, Roberts JD. Association between family history of prostate cancer. JMH. 2010;7:45–54
39. Family History and Probability of Prostate Cancer, Differentiated by Risk Category: A Nationwide

[DOI: 10.58209/ijbc.14.1.18]

Population-Based Study Ola Bratt, Linda Drevin, Olof Akre, Hans Garmo, Par StattinJNCI J Natl Cancer Inst (2016) 108(10): djw11oi: 10.1093/jnci/djw110 Fi0

40. Jun Li, Joseph A. Djenaba, Ashwini Soman, Sun Hee Rim, and Viraj A. Master Recent Trends in Prostate Cancer Incidence by Age, Cancer Stage, and Grade, the United States, 2001–2007Prostate Cancer. 2012; 2012: 691380. Published online 2012 Nov 27. doi: 10.1155/2012/691380 PMCID: PMC3515924 PMID: 23251806
41. Lin DW, Porter M, Montgomery B. Treatment and survival outcomes in young men diagnosed with prostate cancer: a population-based cohort study. Cancer. 2009;115(13):2863–2871.
42. Cooperberg MR, Broering JM, Carroll PR. Time trends and local variation in primary treatment of localized prostate cancer. Journal of Clinical Oncology. 2010;28(7):1117–1123.
43. A. B. Gutman and E. B. Gutman, "An "acid" phosphatase occurring in the serum of patients with metastasizing carcinoma of the prostate gland," The Journal of Clinical Investigation, vol. 17, no. 4, pp. 473–478, 1938.
44. J. A. Whitesel, R. E. Donohue, J. H. Mani et al., "Acid phosphatase: its influence on the management of carcinoma of the prostate," The Journal of Urology, vol. 131, no. 1, pp. 70-71, 1984.
45. H. Y. Kong and J. Byun, "Emerging roles of human prostatic acid phosphatase," Biomolecules & Therapeutics, vol. 21, no. 1, pp. 10–20, 2013.

[DOI: 10.58209/ijbc.14.1.18]