

RESEARCH ARTICLE

Evaluation of Environmental Risk Factors for Prostate Cancer in a Population of Iranian Patients

Farkhondeh Pouresmaeili¹, S Jalil Hosseini^{2*}, Farah Farzaneh³, Arezoo Karimpour¹, Eznollah Azargashb⁴, Mohammad Yaghoobi⁵, Maryam Kamarehei⁶

Abstract

Background: The Prostate cancer is the 2nd most common cancer worldwide for males, and the 5th most common cancer overall, with an estimated 900,000 new cases diagnosed in 2008 (14% of the total in males and 7% of the total overall) aim of this study was to assess some of the most proposed environmental factors influencing the incidence of prostate cancer among Iranian men. Smoking, opioids, occupation and living location were considered as studied risk factors of the prostate cancer in this research. **Material and methods:** Two groups of affected men with prostate cancer and controls aged 50-75 years referred to medical clinics were subjects in this case-control study. Living and working place, smoking and drug consuming habits were assessed for any associations with prostate cancer. **Results:** The largest number, of patients, in order, belonged to Tehran, provincial capitals, major industrial cities, small towns and villages, respectively. The disease showed links with smoking and drugs with a significant difference between controls and patients (P value <0.0001). **Conclusions:** Our recent evidence duplicates previously done researches confirming the serious adverse effects of smoking and drugs on the prostate cancer occurrence in Iranian men. Living place bearings some hazardous behaviors which increases the rate of diseases as well as advanced chance for associated cancers like prostate.

Asian Pac J Cancer Prev, 15 (24), 10603-10605

Introduction

Prostate cancer (PCa), a disease of the elderly, is currently a growing public medical problem with rising incidence and mortality rate among men (Crawford, 2003; RS, 1993). Furthermore, it is recognized as the second leading cause of cancer death in males (Heidenreich et al., 2013). A few risk factors for developing PCa are currently well-known (Doolan et al., 2014). It has been shown that this common malignancy probably originates from a combination of factors including advanced age, ethnicity, family history, endogenous hormones and specific diseases (Quinn and Babb, 2002; Crawford, 2003). The metabolic syndrome, an important public health problem, and specially diabetes mellitus are examples of metabolic disorders involved in increased risk of PCa (Long et al., 2012; Ozbek et al., 2014).

On the other hand, exogenous factors such as food consumption, pattern of sexual behavior, alcohol consumption, exposure to ultraviolet radiation and chronic inflammation influence the risk of PCa progression (Boffetta and Nyberg, 2003; Cerhan et al., 1999; Crawford, 2003; Zhou et al., 2013). Wide variations in PCa incidence and mortality patterns result from differences

in detection practices, treatment, and environmental and genetic factors (Quinn and Babb, 2002; Center et al., 2012). Cigarette smoking as a potential modifiable risk factor may affect PCa etiology by exposure to carcinogens including N-nitroso bis (2-hydroxypropyl) amine (BHP) (Pour, 1981). On the other hand, smoking has been shown to alter serum steroid hormones levels particularly bioavailable testosterone levels (English et al., 2001; Joseph et al., 2004).

The highest estimated PCa mortality rates are observed mainly in North America and also western and northern Europe; however, Asian countries are among the lowest worldwide (Center et al., 2012). A few surveys have been conducted on carcinoma of prostate incidence in Iran (Akbari et al., 2008; Askari et al., 2014; Hosseini et al., 2009; Mazdak et al., 2012; Moslemi et al., 2011; Pourmand et al., 2007). Despite the demonstrated role of some potential risk factors (socioeconomic position and dietary patterns) in PCa, the link between cigarette smoking and this malignancy among Iranian people remains a matter of debate (Mazdak et al., 2012; Rohani-rasaf et al., 2013). Further studies are needed to determine the probable smoking role in PCa progression in Iran. The aim of the present study was to evaluate the relationship

¹Department of Medical Genetics and Infertility and Reproductive Health Research Center, ²Infertility and Reproductive Health Research Center, ³Department of Obstetrics and Gynecology, ⁴Department of Community Medicine, ⁵Urology Department, Faculty of Medicine, Shahid Beheshti University of Medical Sciences, ⁶Institute of Biochemistry and Biophysics, University of Tehran, Tehran, Iran *For correspondence: jhosseinee@gmail.com

between cigarette smoking and carcinoma of the prostate.

We also studied the link between bone density and PCa.

Materials and Methods

In this case- control study, 74, 75, 59 patients with PCa and 129, 108 and 95 healthy control subjects with no history of any type of cancer were selected (Table 1). The age of two groups was between 50-75 years who were referred to the hospitals under Shahid Beheshti University of Sciences coverage. We examined the association of a number of risk factors with PCa. The raw results of the measurement of these variables were analyzed using SPSS software.

Results

Statistical analysis was considered significant (p<0.05). In evaluation of risk factors associated with PCa, the largest number of patients with a priority belonged to Tehran (41.2%), provincial capitals and major industrial cities (36.8%), small towns and villages (22.1%), respectively (Table 2).

When study the disease association with smoking and drugs, a significant difference between controls and patients with p<0.0001 was observed (Table 3). Moreover, the serum levels of Prostate-Specific Antigen (PSA) difference between the two groups was also considered to be significant with p<0.0001.

Serum level of PSA is a biological factor that is affected by PCa involvement. Our statistics show the

Table 1. Mean of Under Study People Age

Groups	N	Mean	Std. Deviation	Std. Error Mean
Age Case	74	68.46	7.208	0.838
Control	116	68.16	9.335	0.867

Table 2. The Investigated Relationship between The Residency and The Risk of Prostate Cancer

Residency	Groups		Total	Asymp sig p value
	Case	Control		
	Count	Count		
	(% within groups)	(% within groups)		
Tehran	28 (41.20)	50 (64.10)	78 (53.40)	0.021
Industrial town	25 (36.80)	17 (21.80)	42 (28.80)	0.021
Small town	15 (22.10)	11 (14.10)	26 (17.80)	0.015
Total	68 (100.00)	78 (100.00)	146 (100.00)	

Table 4. A Comparison between PSA Plasma Level in The Control and Patient Groups

PSA	Independent Samples Test							
	Levene's Test for Equality of Variances		T-test for Equality of Means					
	F	Sig p value	T	df	Sig (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference Lower Upper
Equal variations assumed	66.838	0	15.148	167	0	13.50949	0.89181	11.74882 15.27.15
Equal variations not assumed			10.163	51.509	0	13.50949	1.32923	10.84158 16.17739

increased level of this plasma factor in the affected individuals under the study (Table 4). One of the other risk factors investigated in connection with PCa was Bone Mineral Intensity (BMI) which its average value in patient group was 27. 2678 and in control group was 26. 4646 with a p value of 0.443. As is clear, there was no meaningful association between this factor and the cancer appearance.

Discussion

PCa is the most common reported cancer among Iranian men after stomach cancer (Mohagheghi et al., 2009; Mousavi et al., 2009). Its incidence rates is consistently increasing due to rise in life expectancy and the numbers of old-aged people and it is expected that in the future, the numbers of cancer cases will increase rapidly in Iran (Moslemi et al., 2011). Identification of mechanisms affecting malignancy of prostate etiology and development results in designing potent treatments. Since it is a multi-factorial disease induced by genetic (Yang et al., 2013) and environmental factors, there are numerous factors which are known to cause or increase the incidence of PCa (Doolan et al., 2014). A proven association between prevalence and mortality rate of prostate cancer and smoking has led many to consider smoking as a significant exogenous risk factor for PCa progress (Huncharek et al., 2010). As malignancy of prostate is a major cause of morbidity and mortality in Iran (Moslemi et al., 2011) and there is no evidence to show incidence of PCa among smokers and also non-smokers, we studied cigarette smoking influence on PCa development.

Our research in a population of 190 Iranian men (74 prostate cancer affected and 116 control) from different areas of the country showed results consistent with

Table 3. The Association between Cigarette Smoking, Drug and The Risk of Prostate Cancer

	Groups		Total	Asymp sig (2-sided) p value
	Case	Control		
	Count	Count		
	(% within groups)	(% within groups)		
Addiction				
Smoking	44 (59.50)	27 (26.00)	71 (39.90)	0
Smoking and Opiat	1 (1.40)	1 (1.00)	2 (1.10)	0
No addiction	29 (39.20)	76 (73.10)	105 (59.00)	0
Total	74 (100.00)	104 (100.00)	178 (100.00)	

previously done investigations (Huncharek et al., 2010; Bae et al., 2013).

Significant percentage of prostate cancer affected people who were cigarette smokers and addicted (59.5%, 1.4%) was obviously higher than the rest of the patient population who were not addicted (39.2%). This is an emphasis on the effectiveness and positive influence of smoking on the disease etiology. There are several hypothetical mechanisms through which cigarette smoking may enhance prostate cancer risk (Bae et al., 2013; English et al., 2001; Huncharek et al., 2010; Joseph et al., 2004).

On the other hand, high rate of prostate cancer incidence in industrial cities compared with small towns provides the evidence of lifelong exposures to a polluted environment effects on cancer occurrence (Winkelstein and Kantor, 1963; Calderon-Garciduenas et al., 2013). Previous reports have shown that air pollution besides other factors including the systemic inflammation, vitamin D insufficiency, urban violence could affect bone density during the time (Calderon-Garciduenas et al., 2013) which is in parallel with present study that showed the higher rate of the patients were living in the big and industrial cities and is providing the evidence that the higher concentration of chemical particles residing in the air pollution of these kind of areas are possible active risk factors inducing the cancer formation and/ or progression.

To submit a stronger evidence of this kind, there is urgent need to investigate a bigger sample size of prostate cancer affected individuals.

Acknowledgements

We appreciate those patients who generously accepted our invitation to cooperate us in this research. This work was supported by Shahid Beheshti university of Medical Sciences-Research Deputy.

References

Akbari ME, Hosseini SJ, Rezaee A, et al (2008). Incidence of genitourinary cancers in the Islamic Republic of Iran: a survey in 2005. *Asian Pac J Cancer Prev*, **9**, 549-52.

Mohagheghi MA, Mosavi-Jarrahi A, Malekzadeh R, et al (2009). Cancer incidence in Tehran metropolis: the first report from the Tehran Population-Based Cancer Registry, 1998-2001. *Arch Iran Med*, **12**, 15-23.

Askari F1, Parizi MK, Jessri M, et al (2014). Dietary patterns in relation to prostate cancer in Iranian men: a case-control study. *Asian Pac J Cancer Prev*, **15**, 2159-63.

Bae JM, Li ZM, Shin MH, Kim DH, et al (2013). Cigarette smoking and prostate cancer risk: negative results of the Seoul Male Cancer Cohort Study. *Asian Pac J Cancer Prev*, **14**, 4667-9.

Boffetta P, Nyberg F (2003). Contribution of environmental factors to cancer risk. *Br Med Bull*, **68**, 71-94.

Calderon-Garciduenas L, Mora-Tiscareno A, Francolira M, et al (2013). Exposure to urban air pollution and bone health in clinically healthy six-year-old children. *Arh Hig Rada Toksikol*, **64**, 23-34.

Center MM, Jemal A, Lortet-Tieulent J, et al (2012). International variation in prostate cancer incidence and mortality rates. *Eur Urol*, **61**, 1079-92.

Carhan JR, Parker AS, Putnam SD, et al (1999). Family history and prostate cancer risk in a population-based cohort of Iowa men family history and prostate cancer risk in a population-based cohort of Iowa men. *Cancer Epidemiol Biomarkers Prev*, **8**, 53-60.

Crawford ED (2003). Epidemiology of prostate cancer. *Urology*, **62**, 3-12.

Doolan G, Benke G, Giles G (2014). An update on occupation and prostate cancer. *Asian Pac J Cancer Prev*, **15**, 501-16.

English KM, Pugh PJ, Parry H, et al (2001). Effect of cigarette smoking on levels of bioavailable testosterone in healthy men. *Clinical Science (Lond)*, **100**, 661-5.

Heidenreich A, Bastian PJ, Bellmunt J, et al (2014). EAU guidelines on prostate cancer. Part II: Treatment of advanced, relapsing, and castration-resistant prostate cancer. *Eur Urol*, **65**, 467-79.

Hosseini M, SeyedAlinaghi S, Mahmoudi M, et al (2010). A case-control study of risk factors for prostate cancer in Iran. *Acta Med Iran*, **48**, 61-6.

Huncharek M, Haddock KS, Reid R, et al (2010). Smoking as a risk factor for prostate cancer: a meta-analysis of 24 prospective cohort studies. *Am J Public Health*, **100**, 693-701.

Joseph MA, Wei JT, Harlow SD, et al (2004). Relationship of serum sex-steroid hormones and prostate volume in African American Men. *Prostate*, **329**, 322-9.

Long XJ, Lin S, Sun YN, Zheng ZF (2012). Diabetes mellitus and prostate cancer risk in Asian countries: a meta-analysis. *Asian Pac J Cancer Prev*, **13**, 4097-100.

Mazdak H, Mazdak M, Jamali L, et al (2012). Determination of prostate cancer risk factors in Isfahan, Iran: a case-control study. *Med Arh*, **66**, 45-8.

Moslemi MK, Lotfi F, Tahvildar SA (2011). Evaluation of prostate cancer prevalence in Iranian male population with increased PSA level, a one center experience. *Cancer Manag Res*, **3**, 227-31.

Mousavi SM, Gouya MM, Ramazani R, et al (2009). Cancer incidence and mortality in Iran. *Ann Oncol*, **20**, 556-63.

Ozbek E, Otunctemur A, Dursun M, et al (2014). The metabolic syndrome is associated with more aggressive prostate cancer. *Asian Pac J Cancer Prev*, **15**, 4029-32.

Pour PM (1981). A new prostatic cancer model: systemic induction of prostatic cancer in rats by a nitrosamine. *Cancer Lett*, **13**, 303-8.

Pourmand G, Salem S, Mehraei A, et al (2007). The risk factors of prostate cancer: a multicentric case-control study in Iran. *Asian Pac J Cancer Prev*, **8**, 422-8.

Quinn M, Babb P (2002). Patterns and trends in prostate cancer incidence, survival, prevalence and mortality. Part I: international comparisons. *BJU Int*, **90**, 162-73.

Rohani-Rasaf M1, Abdollahi M, Jazayeri S et al (2013). Correlation of cancer incidence with diet, smoking and socio-economic position across 22 districts of Tehran in 2008. *Asian Pac J Cancer Prev*, **14**, 1669-76.

RS P (1993). Epidemiology of prostate cancer; with emphasis on familial clusters. *Cancer Bull*, **45**, 384-9.

Winkelstein W, Kantor S (1963). Particulate air pollution, thus replicating findings in the Nashville economic status. *Nonlinearity Biol Toxicol Med*, **59**, 259-92.

Yang B, Chen WH, Wen XF, et al (2013). Role of DNA repair-related gene polymorphisms in susceptibility to risk of prostate cancer. *Asian Pac J Cancer Prev*, **14**, 5839-42.

Zhou XF, Ding ZS, Liu NB (2013). Allium vegetables and risk of prostate cancer: evidence from 132,192 subjects. *Asian Pac J Cancer Prev*, **14**, 4131-4.