Med Oncol (2012) 29:823–826
DOI 10.1007/s12032-011-9940-x

ORIGINAL PAPER

Prostate-specific antigen: its relationship with alcohol intake and tobacco

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Received: 18 February 2011 / Accepted: 30 March 2011 / Published online: 12 April 2011

Abstract To determine the influence of alcohol and tobacco consumption on serum prostate-specific antigen (PSA) levels. 59 men participated in this study: 20 with prostate tumors (PT) and 39 without tumor diagnosis (prostate controls, PC) (mean 66 and 58 years, respectively). PSA was analyzed in serum samples and its values were compared through the Kruskal–Wallis nonparametric test. Alcohol and tobacco consumption was also considered. PSA mean value was higher than 4 ng/ml in PT, whereas in PC it was lower than that value. Statistically significant differences were found when comparing PSA between PT and PC ($P < 0.05$). PSA was higher in alcohol and tobacco consumers than in non-consumers in PT group ($P < 0.05$). For PC, PSA mean values were higher in non-smokers than in smokers. Statistically significant differences were observed for serum PSA when compared between PT and PC groups considering alcohol and tobacco consumption ($P < 0.05$). Serum PSA values appear to be influenced by alcohol and tobacco consumption.

Keywords Alcohol · Prostate-specific antigen · Prostate · Tobacco · Tumors

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Introduction

Prostate cancer is the most common non-skin cancer affecting men and also the third cause of cancer death in the world [1, 2]. Since 1994, the prostate-specific antigen (PSA) is the routine marker for early diagnosis, prognosis and prostate tumor follow up, and for controlling the effectiveness of hormone therapy [3–6]. Normal serum values are considered up to 4 ng/ml [6, 7].

The benign and malign prostate diseases are associated with an increased serum PSA level. According to Baum and Lipp [8], the serum PSA value increases on average 0.3 ng/ml/g of prostate tissue in men with benign prostatic hyperplasia. Thus, the greater the gland size, the higher the PSA value. However, this increase in PSA level is lower than that found in patients with clinical diagnosis of prostate cancer.

Studies carried out in humans indicate that various prostate diseases non-associated factors could affect serum PSA values [9–12]. In this regard, higher levels of PSA were reported in older patients with a statistically significant correlation between prostate volume, age, and PSA

level [13]. According to Gray et al. [11], tobacco consumption among other clinical and demographic factors has a significant effect on the serum PSA level in patients without clinical evidence of prostate cancer. Former and current smokers showed lower PSA values as compared to non-smokers.

Experimentally, Gumus et al. [14] observed dilatations in the endoplasmic reticulum cisternae of prostate acinar cells and disarrangements in the Golgi complex in rats with acute alcohol intake and killed after 3, 24, and 72 h. Nevertheless, just the group treated with alcohol and killed after 3 h showed a significant increase in serum PSA levels.

However, no references were found about the influence of alcohol (ethanol) intake on the serum PSA level in neither healthy nor prostate tumor patients as well as about the effects of tobacco in patients with the abovementioned tumor. Thus, the objective of this study was to determine the influence of alcohol and tobacco consumption on serum PSA in healthy and prostate tumor patients.

Patients, materials, and methods

Patients

Fifty nine men attending the Urology Department of the Privado hospital, in Córdoba, Argentina, participated in the study. 20 men (mean = 66 years) presented diagnosis of prostate tumors (PT). The clinical and ultrasound diagnoses were confirmed by histopathological examination. Benign prostate hyperplasia and adenocarcinoma were the benign and malignant prostate tumors, respectively.

All patients were incorporated into the study immediately after the tumor diagnosis and before starting any treatment. In addition, 39 men (mean = 58 years) were included as a control (PC) group. They had neither diagnosis nor tumor evidence in any location. PC was matched for age (±5 years) with respect to PT, respectively.

Volunteers have participated in the study in concordance to the ethical principles for human research (Helsinki Declaration) and signed an informed consent approved by the Institutional Committee for Ethics in Health Research (CIEIS) of the involved hospitals.

Materials

Medical history

Data such as age, birth date, habitual medicine intake, clinical–pathological, and family background were registered. Information about tobacco smoking like mode (cigarette or pipe), amount, and consumption time as well as type, frequency, and time of alcoholic beverage intake was also included.

Samples

Blood samples from all participants were obtained and serum aliquots were stored at −20°C.

Reagents

Elecsys total test (ECLIA electrochemiluminescence immunoassay), (Roche DiagnostiSC GmbH, D-68298 Mannheim, Germany) was used to analyze the serum PSA.

Methods

PSA analysis

In vitro quantitative detection of total serum PSA levels was carried out by using an Elecsys 2010 autoanalyzer. The assays were performed according to the manufacturer's specifications. Values are expressed as ng/ml.

Alcohol intake

The information on alcoholic beverage intake obtained through the medical history was processed by the *Interfood v.1.3* software [15] in order to get the daily alcohol intake. It is expressed as grams of alcohol per day (g/day).

Statistical analysis

The Kruskal–Wallis nonparametric test was employed to compare the serum PSA levels between PT and PC groups and to correlate those values with alcohol and tobacco consumption variables ($P < 0.05$). The Infostat statistical software package was used [16].

Results

The serum PSA mean values in the PT group was 4.86 ± 5.61 ng/ml (mean ± SD), whereas it was lower than 4 ng/ml in the PC group (2.27 ± 2.35 ng/ml). Statistically significant differences were found when comparing the mean PSA values between PT and PC ($P < 0.05$).

Table 1 presents the serum PSA concentration in relation to the tumor histopathology. PSA was higher than 4 ng/ml in patients bearing benign ($n = 15$) and malignant ($n = 5$) PT. No statistically significant differences were observed between both groups.

Table 1 Serum PSA values according to tumor histopathology

Histopathology	PT	*P*
Benign	4.87 ± 6.23	0.69
Malign	4.84 ± 3.75	

Values are expressed as ng/ml and correspond to mean ± SD

PT prostate tumor

P value corresponds to the Kruskal–Wallis test

Table 2 Serum PSA values according to alcohol and tobacco consumption by group

Variables	PT	PC	*P*
Alcohol consumption			0.04
No	0.30	1.80	
Yes	5.10	2.31	
Tobacco consumption			0.03
No	3.63	2.77	
Yes	7.14	1.69	

Values are expressed as ng/ml and correspond to mean ± SD

PT prostate tumor, *PC* prostate controls

P value corresponds to the Kruskal–Wallis test

Daily ethanol intake was 340.76 g/day in PC group and 190.46 g/day in PT group. Red wine was the most frequently consumed beverage in both groups. 94% of patients reported alcohol consumption for over 10 years.

Cigarette was the only way of tobacco smoking in all groups. 40% of persons belonging to the whole sample smoked 10–20 cigarettes per day and 24% of them smoked more than 20 cigarettes per day. With respect of consumption time, 76% of all participants had smoked for over 10 years.

Table 2 shows the PSA mean values according to alcohol and tobacco consumption. PSA mean values were higher in alcohol and tobacco consumers than in non-consumers in PT group. For PC, PSA mean values were higher in non-smokers than in smokers. Statistically significant differences were observed for serum PSA when compared between PT and PC groups considering alcohol and tobacco consumption ($P < 0.05$).

Discussion

A significant increase of serum PSA was observed in relation to alcohol consumption in both groups. Although no similar studies have been found in humans, experimental studies carried out in rats have shown that alcohol—at different application times—coincidently increases the serum PSA levels [14]. These findings could be associated with the disorganization of the epithelial cell layers and the disruption of the epithelial basement membrane of prostate tissue that Baum and Lipp [8] found in this gland pathology. They suggest that abnormal prostate conditions allow PSA molecules to diffuse more easily from the acini of the epithelial glands to the adjacent capillaries and thus to the blood stream.

Serum PSA has shown to be higher in smokers of more than ten cigarettes per day for over 10 years than in non-smokers in PT group. No references about the influence of tobacco consumption on the serum PSA level in men bearing PT were found. In healthy people, Gray et al. [11] observed lower PSA levels in smokers than in non-smokers coincidently with our finding in PC group. That author suggests that it could be related to the influence of smoking on the testosterone serum concentrations which have a secondary effect on serum PSA in healthy patients. However, the mechanisms of action of both toxic habits on serum PSA are still unknown. Elzanaty et al. [17] found that the abnormal quality and quantity of PSA can result in a depression of sperm motility and subinfertility. Martini et al. [18] observed that alcohol and tobacco consumption has a detrimental effect on the human seminal quality such as a significant reduction in seminal volume, sperm concentration, percentage of motile spermatozoa, and an increase in the percentage of the nonmotile viable gametes.

These results would indicate that certain prostate disease non-associated factors like alcohol and tobacco consumption have an influence on serum PSA values. However, further research extended to other geographical contexts as well as studies tending to understand the action mechanisms of alcohol and tobacco on the expression of this tumor marker are required. It would contribute to a better clinical use of PSA.

Acknowledgments The authors thank the following persons and funding institutions: Dr. Marcelo Ruggieri, Casilda Rupérez and *Secretaría de Ciencia y Tecnología. Universidad Nacional de Córdoba*. Res. no. 123/04, 197/05, 162/06, 069/08, 214/10, and *Ministerio de Ciencia y Tecnología-Provincia de Córdoba* Res. no. 121/08, Córdoba, Argentina.

Conflict of interest None of the authors have any potential conflicts of interests to disclose.

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