PROSPECTIVE STUDY

Alternative mechanisms for prostate-specific antigen elevation: A prospective analysis of 222 transurethral resections of prostate patients

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Abstract

AIM: To investigate the relationship between prostate-specific antigen (PSA) levels and (1) bladder outlet obstruction (BOO) and (2) the severity of prostate inflammation.

METHODS: Two hundred and twenty-two consecutive patients undergoing transurethral resection of the prostate (TURP) were prospectively included. Patients with proven urinary tract infection and/or known prostate cancer were excluded. PSA levels, International Prostate Symptoms Score (IPSS), prostate weight, post residual volume and pressure flow parameters were determined. A histopathological assessment of the presence and severity of inflammation was also performed.

RESULTS: Patients had a mean age of 69.1 ± 8.6 years (45-90 years), with mean preoperative PSA levels of 4.7 ± 5.4 ng/mL (0.2-32.5 ng/mL) and IPSS of 15.7 ± 6.9 (0-32). Mean $PdetQ_{max}$ was 96.3 ± 34.4 cmH_2O (10-220 cmH_2O). The mean resected prostate weight was 39.4 ± 27.3 g (3-189 g). Correlations were observed between PSA (logarithmic) and resected prostate weight ($r = 0.54$; $P < 0.001$), PSA (logarithmic) and $PdetQ_{max}$ ($r = 0.17$; $P = 0.032$), and resected prostate weight and $PdetQ_{max}$ ($r = 0.39$; $P < 0.001$). Furthermore, low correlations were observed between PSA (logarithmic) and active ($r = 0.21$; $P < 0.0001$) and chronic ($r = 0.19$; $P = 0.005$) inflammation.

CONCLUSION: In this study we showed a correlation between BOO ($PdetQ_{max}$) and PSA (logarithmic). Furthermore, we demonstrated a weak correlation between PSA (logarithmic) and active as well as chronic prostatic inflammation.

Core tip: The goal was to investigate the relationship between prostate-specific antigen (PSA) levels and (1)

bladder outlet obstruction (BOO) and (2) the severity of prostate inflammation. We performed a prospective study on 222 consecutive patients undergoing transurethral resection of the prostate. Patients with proven urinary tract infection and/or known prostate cancer were excluded. PSA levels, International Prostate Symptoms Score, prostate weight, post residual volume and pressure flow parameters were determined. A histopathological assessment of the presence and severity of inflammation was also performed. In this study we showed a correlation between BOO ($PdetQ_{max}$) and PSA (logarithmic). Furthermore, we demonstrated a weak correlation between PSA (logarithmic) and active as well as chronic prostatic inflammation.

INTRODUCTION

Since its introduction, prostate-specific antigen (PSA) has played a key role in prostatic evaluation[1]. Elevated or rising PSA levels might indicate prostate cancer, a highly prevalent cancer in men with an incidence rate surpassing 20%[2]. The downside of PSA testing is that it is not cancer specific but merely organ specific.

Consequently, elevated PSA levels are a challenging problem for urologists in assessing or excluding potential life-threatening prostate cancer. However, to confirm the diagnosis of prostate cancer, an additional histological evaluation from prostate biopsies is still required. These biopsies can be falsely negative and cannot be repeated infinitely[3]. Furthermore, taking a prostate biopsy is a moderately invasive examination that has, although infrequently, potential life threatening complications[4].

Therefore, a better understanding of other possible mechanisms causing PSA elevation is of utmost importance as this may help to avoid unnecessary biopsies and prevent patient anxiety. Furthermore, patients do not have to be bothered with other, sometimes expensive diagnostic tests, including magnetic resonance imaging[5], advanced transrectal ultrasound imaging[6] or molecular diagnostics[7].

Benign prostate hyperplasia (BPH) is a very common condition in ageing males. As prostatic enlargement can be asymptomatic, the exact incidence of BPH is unknown, ranging between 28% and 60% of the population[8,9]. Additionally, BPH seems to be the second most common reason for surgery in men over 60[10]. Currently, the exact pathogenesis of BPH is not fully understood. Amongst other factors, including hormonal influence, prostatic inflammation could stimulate prostatic growth.

Moreover, data on the association between inflammation, prostatic volume, PSA levels and acute urinary retention risk have been published[11].

Therefore, the aim of this study was to investigate the relationship between PSA and the degree of prostate inflammation and to investigate whether PSA levels can be used as a biomarker for bladder outlet obstruction (BOO).

MATERIALS AND METHODS

The study was approved by the hospital's Ethics Committee (07.58/uro07.02) and was conducted according to the established GCP criteria. In this prospective study, 222 consecutive patients undergoing transurethral resection of the prostate (TURP) between May 2008 and June 2010 were included. A single high volume surgeon in a non-academic referral center operated on all patients. As indicated in the EAU guidelines[12], surgery was performed only on patients with a clear indication for TURP, including patients who did not improve after medical therapy, patients with (recurrent) acute urinary retention, high post void residual volume, obstruction characterised by pressure flow analysis or post renal kidney insufficiency. However, patients who were treated with 5ARIs were excluded because of the possible impact on PSA values. Patients with a proven urinary tract infection were excluded, except for patients with catheters, which are colonised by definition. Additionally, patients with known prostate cancer were excluded in order to prevent influence on PSA by cancer cells.

Before surgery, PSA levels were determined for all patients by GPs. The International Prostate Symptom Score (IPSS) was determined. Full urodynamic studies were performed in 154 patients using Laborie Medical Technologies INC/UDS-64-IIs and were evaluated by PIs. Urodynamic studies were not performed in cases of acute urinary retention or high post-residual volume. All patients were treated with low-dose quinolone prophylaxis for 48 hours, starting the day before urodynamic testing. Filling was done standing with a filling speed of 35 mL/minute, using a 6F-filling catheter (double lumen). When indicated, pressure flow analysis was performed according to the International Continence Society criteria[13]. Endoscopic procedures were performed under loco-regional anesthesia using an Olympus resectoscope 26 (6%) or 28 Charrière (94%), depending on the estimated prostate volume. The resected prostate specimens were weighed and the fragments were embedded until four cassettes, each containing 2 g of tissue, were filled. Each additional 10 g of prostate tissue was used to fill an extra cassette[14]. The tissue was fixed in formalin and embedded in paraffin. One slide was made from every paraffin block and examined after staining by hematoxylin and eosin. The inflammatory infiltrate was first divided into an active (mixed infiltrate of lymphocytes, plasma cells and polynuclear cells) and chronic (mononuclear infiltrate of lymphocytes and plasma cells) component, after which the density of both components

Reference tissue (A0 and C0)

| A1 | A2 | A3 |
| C1 | C2 | C3 |

Figure 1 Scores for active and chronic inflammation. A: Active inflammation; C: Chronic inflammation. Semi-quantitative scoring scale from 0 (no infiltrate) to 3 (severe infiltrate).
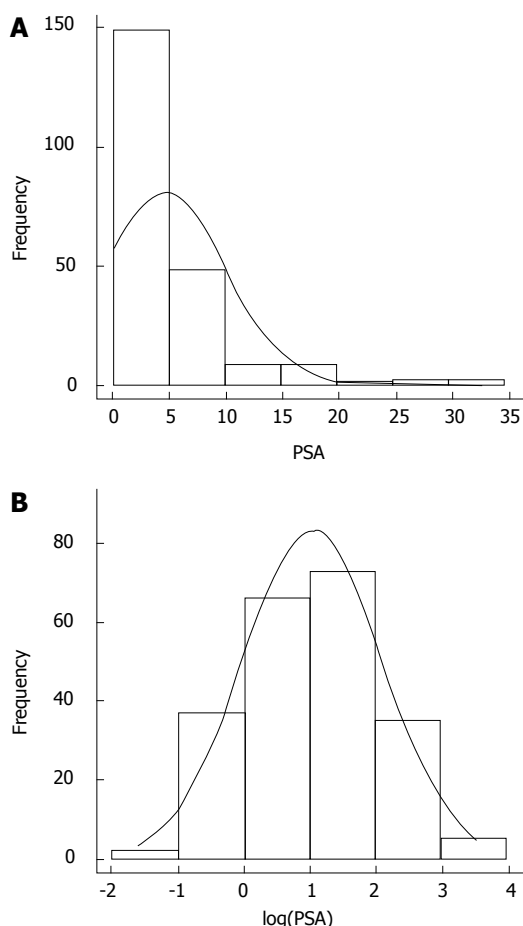
Figure 2 Histogram for prostate-specific antigen (A) and log prostate-specific antigen (B) with a normal density curve superimposed. PSA: Prostate-specific antigen.

was scored 0-3 according to a semi-quantitative scoring system: 0 being no infiltrate and 3 severe infiltrate (Figure 1). Two senior pathologists analyzed all tissue sections of each patient independently and were blinded for clinical data. They scored the mean value of the infiltrate, considering all tissue sections.

Statistical analysis

In an initial step, data were analyzed with descriptive statistics and Pearson correlation was used to investigate simple correlations between different variables. Appropriate linearity tests and lack of fit tests were performed to ensure that these relationships were indeed linear and adequate. Secondly, in a more in depth analysis the relationship between PSA levels and the different potential covariates was analyzed with a multiple regression model. Since the histogram of PSA is severely skewed

Table 1 Characteristics of the 222 consecutive patients undergoing transurethral resection of the prostate between May 2008 and June 2010

	n	Mean	SD	Min	Max
PSA (ng/mL)	218	4.7	5.4	0.2	32.5
Age (yr)	222	69.1	8.6	45	90.0
UF (mL/s)	204	10.7	6.5	1	47.2
IPSS	222	15.7	6.9	0	32.0
PRV (mL)	208	83.8	130.3	0	890
P/F (cm H$_2$O)	154	96.3	34.4	10	220
Operation time (min)	222	31.1	11.9	10	110
TURP weight (g)	222	39.4	27.3	3	189

SD: Standard deviation; PSA: Prostate specific antigen; UF: Uroflow; IPSS: International prostate symptoms score; PRV: Post residual volume; P/F: Pressure flowmetry; TURP: Transurethral resection of the prostate; Min: Minimum; Max: Maximum.

due to the prevalence of lower PSA values in a normal population (Figure 2), PSA values were transformed with a natural log transformation, resulting in a more realistic normality assumption of the log PSA than the original PSA scale. Selection of the variables included in the model for log PSA was based on the AIC criterion and the adjusted r-square. With respect to the variable active inflammation, it might not seem realistic from a clinical point of view to assume that the difference between any consecutive levels of the active inflammation was the same or the increase was linear in nature. For this reason, the choice was made to treat inflammation as a categorical rather than a continuous variable. Furthermore this choice resulted in a better fit of the model.

RESULTS

Patient characteristics

The patients were between 45 years and 90 years old (mean age: 69.1 ± 8.6 years) (Table 1) and had an indication for TURP. Mean preoperative PSA value was 4.7 ± 5.4 ng/mL (0.2 to 32.5 ng/mL). Mean IPSS was 15.7 ± 6.9 (0 to 32). Mean peak flow was 10.7 ± 6.5 mL/s (1 to 47 mL/s). Post residual volume ranged from 0 to 890 mL with a mean value of 83.8 ± 130.3 mL. When indicated ($n = 154$), pressure flow analysis was executed and showed a mean value of PdetQ$_{max}$ of 96.3 ± 34.4 cmH$_2$O (10 to 220 cmH$_2$O). The mean resected prostate weight was 39.4 ± 27.3 g (3 to 189 g). Mean operating time was 31.1 ± 11.9 min (10 to 110 min).

Correlations with PSA

We investigated the relationship between log PSA and several parameters, including IPSS, maximum uroflow, post voided residual volume, pressure flowmetry parameter PdetQ$_{max}$ and the weight of the resected prostate tissue (Table 2). A significant correlation between log PSA and the resected prostate weight was found ($r = 0.54$; $P < 0.001$). Furthermore, log PSA was correlated with PdetQ$_{max}$ ($r = 0.17$; $P = 0.032$). A negative correlation was encountered between log PSA and IPSS ($r = $

Table 2 Correlation between log (prostate specific antigen) and the corresponding predictor variables

Variable	Linearity (*P* value)	Significance (*P* value)	Correlation (*r*)
IPSS	0.488	0.04	-0.14
Age (decades)	0.563	< 0.0001	0.29
UF (mL/s)	0.929	0.986	0.00
PRV (mL)	0.766	0.276	0.08
P/F (cm H$_2$O)	0.443	0.032	0.17
TURP weight (g)	0.504	< 0.0001	0.54

IPSS: International prostate symptoms score; UF: Uroflow; PRV: Post residual volume; P/F: Pressure flowmetry; TURP: Transurethral resection of the prostate.

Table 3 Multiple regression model results

Effect	Estimate(st.err)	*P* value
Age (decades)	0.18 (0.069)	0.010
IPSS	-0.02 (0.008)	0.008
Active_0	-1.58 (0.47)	0.001
Active_1	-0.96 (0.53)	0.073
Active_2	-0.78 (0.54)	0.146
Active_3	-0.80 (0.55)	0.150
TURP weight	0.034 (0.006)	< 0.0001
R^2 adj	0.377	

IPSS: International prostate symptoms score; Active 0: No active inflammation; Active 3: Most severe inflammation; TURP: Transurethral resection of the prostate.

-0.14; $P = 0.04$). No correlation was observed between log PSA and peak flow on uroflowmetry ($r = 0.00$; $P = 0.99$). The correlation between log PSA and the post residual volume ($r = 0.08$; $P = 0.28$) was not significant. Additionally, we did not find any correlation between IPSS and PdetQ$_{max}$ ($r = 0.051$; $P = 0.53$). Last but not least, a significant correlation between the weight of the resected tissue and PdetQ$_{max}$ ($r = 0.39$; $P < 0.001$) was observed. These relationships were confirmed in a multiple regression model (Table 3).

Correlations with inflammation (active and chronic)

We also investigated the relationship between inflammation (active and chronic) and log PSA, IPSS, age and the resected prostate tissue weight (Tables 4 and 5). A significant correlation between log PSA and active ($r = 0.21$; $P < 0.0001$) and chronic ($r = 0.19$; $P = 0.005$) inflammation was observed. Age was related to active ($r = 0.24$; $P < 0.0001$) but not to chronic ($r = 0.09$; $P = 0.08$) inflammation. Similarly, the weight of the resected prostate tissue was related to active inflammation ($r = 0.13$; $P = 0.011$) but not to chronic inflammation ($r = 0.1$; $P = 0.34$). However, IPSS was not correlated with active inflammation ($r = 0.03$; $P = 0.6$) or chronic inflammation ($r = -0.03$; $P = 0.91$). Additionally, categorization of IPSS into 3 categories did not result in any association with active and chronic inflammations (Table 6).

We also evaluated the influence of pre-operatively placed suprapubic catheters, trans-urethral catheters or previous prostate puncture biopsies and TURP on the relationship with active and chronic inflammation (Table 7). Only a weak negative correlation was found ($r = -0.16$; $P = 0.003$) between active inflammation and the presence of a suprapubic catheter. No significant correlation was observed with the other variables (Table 7). However, for some categories of degree of inflammation no data were obtained, complicating comparisons.

DISCUSSION

PSA measurement is one of the cornerstones in prostate evaluation. Besides BPH and acute prostatitis, prostate cancer can be one of the reasons for PSA elevation, sensitizing and alarming many patients and care givers. Dealing with a patient with elevated/rising PSA levels is always a challenge, especially in ruling out potential life-threatening prostate cancer.

Relationship between PSA and BPH and/or BOO

Multiple papers have been published indicating the relationship between PSA levels and BOO. In patients with clinical BPH, PSA levels are shown to be positively correlated with the five year cumulative risk of invasive BPH treatment[15]. Additionally, in men with BPH, prostate volume and PSA have been evaluated as predictors of acute urinary retention[16,17]. Consequently, elevated PSA levels are predictive for the need of BPH-related surgery. PSA has also been shown to be a strong predictor of future prostate growth[18], which is related to a higher risk of acute urinary retention and, subsequently, to the need for BPH-related surgery. A broader approach for the use of PSA seems justified. In a multi-center study in men suffering from LUTS, a correlation between PSA and the category of BOO was observed[19], Above a PSA-level of 4 ng/mL, mild or definite BOO was observed (in 89% of the cases) and below 2 ng/mL, the chances of a patient not suffering from BOO were one in three[19]. In previous papers, we have shown that BOO can be expected in a very particular group of patients with elevated and/or rising PSA, (multiple) negative multisided prostate biopsies and minor LUTS (mild + moderate IPSS)[20,21]. In a retrospective analysis of 82 consecutive patients, 95.9% were clearly obstructed (PdetQ$_{max}$ ≥ 40 cm H$_2$O)[18]. Similar results were obtained in a prospective analysis ($n = 33$), with a mean PdetQ$_{max}$ of 80.3 cm H$_2$O[21]. A positive correlation between PSA velocity and PdetQ$_{max}$ was found ($r = 0.5014$; $P = 0.006$)[22]. As already mentioned, rising PSA levels can also be observed in patients with prostate cancer. Notwithstanding the presence of a tumor in these patients, the elevated PSA levels can also be influenced by BOO, implying that PSA should be included in the pre-treatment workup and the post-therapy evaluation of these patients. Therefore, PSA should be considered an additional indicator in the BOO decision tree.

We found some interesting correlations in this pro-

Table 4 Correlation between active inflammation and prostate related continuous variables

	Active inflammation				*P* value	Correlation, *r*
	0	**1**	**2**	**3**		
Log PSA, *n*	25	91	88	14		
Mean (SD)	1.4 (1.6)	4.4 (4.3)	5.9 (6.4)	4.6 (6.6)	<0.0001	0.21
IPSS, *n*	25	94	88	15		
Mean (SD)	14.4 (6.3)	16.2 (7.0)	15.3 (7.0)	16.6 (7.1)	0.595	0.03
Age, *n*	25	94	88	15		
Mean (SD)	59.7 (5.4)	70.1 (8.1)	70.8 (8.2)	68.4 (9.5)	<0.0001	0.24
PRV, *n*	25	90	81	15		
Mean (SD)	75.0 (103.9)	83.8 (110.6)	68.9 (110.0)	202.8 (303.2)	0.001	0.09
TURP weight, *n*	25	94	88	15		
Mean (SD)	24.7 (10.7)	39.5 (27.4)	44.2 (30.4)	32.6 (14.0)	0.011	0.13

SD: Standard deviation; PSA: Prostate specific antigen; IPSS: International prostate symptoms score; PRV: Post residual volume; TURP: Transurethral resection of the prostate.

Table 5 Correlation between chronic inflammation and prostate related continuous variables

	Chronic inflammation				*P* value	Correlation, *r*
	0	**1**	**2**	**3**		
Log PSA, *n*	4	109	91	14		
Mean (SD)	1.0 (0.7)	4.0 (5.0)	5.7 (5.9)	4.2 (4.4)	0.005	0.19
IPSS, *n*	4	110	93	15		
Mean (SD)	14.8 (7.9)	16.0 (6.5)	15.3 (7.2)	15.7 (8.3)	0.91	-0.03
Age, *n*	4	110	93	15		
Mean (SD)	61.5 (5.2)	68.4 (8.75)	70.5 (8.31)	67.6 (9.3)	0.08	0.09
PRV, *n*	4	105	86	13		
Mean (SD)	68.5 (137.0)	80.3 (129.5)	83.8 (121.8)	116.9 (191.4)	0.88	0.05
TURP weight, *n*	4	110	93	15		
Mean (SD)	25.0 (5.2)	36.8 (25.4)	42.5 (28.9)	40.1 (31.6)	0.34	0.1

SD: Standard deviation; PSA: Prostate specific antigen; IPSS: International prostate symptoms score; PRV: Post residual volume; TURP: Transurethral resection of the prostate.

Table 6 Relationship between International Symptoms Score (categorized) and inflammation

		IPSS Categorized			*P* value
		Mild	**Moderate**	**Severe**	
Active inflammation	0	4	16	5	0.68
	1	11	47	36	
	2	15	43	30	
	3	2	7	6	
Chronic inflammation	0	1	1	2	0.13
	1	10	65	35	
	2	18	41	34	
	3	3	6	6	

IOSS: International Prostate Symptoms Score. 0: No inflammation; 3: Most severe inflammation.

spective analysis of 222 consecutive patients undergoing TURP for bothersome LUTS. A significant correlation was shown between log PSA and PdetQ$_{max}$. In line with the literature[22], log PSA was also correlated with prostate volume (the resected prostate weight) ($r = 0.54$; $P < 0.001$). Interestingly, a negative correlation between IPSS and log PSA was observed ($r = -0.14$; $P = 0.04$), confirming the findings of our previous studies. This negative correlation could partly be explained by the correlation between high PSA levels and high detrusor pressures due to an increased compensation level of the detrusor for the urethral resistance, resulting in high flow rates and less symptoms.

On the other hand, no statistically significant correlation was observed between log PSA and post voiding residual volume, and between IPSS and the weight of the resected tissue. This might mean that the response of the detrusor to obstruction is more related to the symptoms than the degree of obstruction as such. Last but not least, a highly significant correlation between the weight of the resected tissue and PdetQ$_{max}$ ($r = 0.39$; $P < 0.001$) was observed, implying that bigger prostates are more frequently associated with obstruction and therefore are more prone to BPH-related surgery.

Relationship between PSA and prostate inflammation

The role of chronic prostate inflammation in PSA elevation in asymptomatic men is still unclear. Many papers have covered this subject with various and sometimes contradictory outcomes and conclusions. In a prospective analysis of a small group of asymptomatic patients ($n = 51$) who underwent prostate puncture biopsies, a statistically significant correlation was found between the inflammatory process and the PSA values ($P = 0.02$)[23].

Table 7 Correlation between inflammation (active and chronic) and risk factors for inflammation

		Active inflammation				P value	Correlation, r_c	Chronic inflammation				P value
		0	1	2	3			0	1	2	3	
SPC	Yes	1	6	6	5	0.003	-0.16	0	8	7	3	0.34
	No	124	88	82	10			4	102	86	12	
TUC	Yes	0	1	3	0	0.710	1	0	2	2	0	1
	No	25	93	85	15			4	108	91	15	
PPB	Yes	2	29	27	4	0.130	1	1	25	34	2	0.08
	No	23	65	61	11			3	85	59	13	
Re-TURP	Yes	2	13	11	2	0.930	1	0	14	12	2	1
	No	23	81	77	13			4	96	81	13	

[1]No significant correlation. SPC: Presence of a suprapubic catheter; TUC: Presence of a transurethral catheter; PPB: Prostate biopsy punctures; Re-TURP: Previous transurethral resection of the prostate.

Similarly, in a retrospective analysis of 238 men, a correlation between PSA and inflammation was observed[24]. In an analysis of the prostate puncture biopsy of 80 asymptomatic patients, the extent of inflammation was positively correlated with total PSA levels ($P < 0.001$)[25]. In all these studies, histological examination was performed on prostate puncture biopsies in asymptomatic men, limiting the results obtained.

In contrast to the previously mentioned reports, multiple papers claimed the opposite and concluded that there is no correlation between PSA and inflammation. In a retrospective analysis of cancer negative prostate biopsies ($n = 233$), it was concluded that the degree of chronic inflammation did not correlate with PSA levels[26].

Chronic prostatitis was encountered in 68.3% of 284 patients with negative prostate puncture biopsies, while active prostatitis was observed in 8.4% of patients[27]. However, inflammation did not correlate with total PSA levels. In a prostate puncture biopsy driven evaluation ($n = 49$), the presence of inflammation did not correlate statistically with the PSA levels[28]. Finally, Nickel *et al*[29] studied a cohort of 80 patients without a history of prostatitis who underwent TURP. After histological examination on the resected tissue, prostatic inflammation was found to be extremely common. On the other hand, no correlation was observed between inflammation and PSA levels.

We performed a prospective analysis in 222 patients undergoing TURP according to the EAU guidelines[30] but without proven infection, known prostate cancer and a history of chronic prostatitis. We made a histological distinction between active and chronic inflammation, each with 4 subcategories. Additionally, since we used TURP tissue and not prostate puncture biopsy derived tissue, we were able to evaluate a substantial prostatic tissue volume. Our results showed that most patients have some degree of inflammation. Especially in the active inflammation group, a correlation between log PSA and inflammatory parameters was observed ($r = 0.21$; $P < 0.0001$). However, we did not encounter a statistically relevant correlation between symptoms (IPSS) and inflammation. The correlation between age and active inflammation ($r = 0.24$; $P < 0.0001$) is interesting to note, which was not the case for the chronic inflammation group ($r = 0.09$; $P = 0.08$). A possible explanation for this finding could be the accumulation of debris in the prostate during the lifetime, resulting in more frequent active inflammations. This finding may also be the explanation for the correlation between prostate weight (TURP) and active ($r = 0.13$; $P = 0.011$) and chronic ($r = 0.1$; $P = 0.34$) inflammation.

In conclusion, elevated and/or rising PSA levels with regard to underlying prostate cancer and prostatic inflammations are well known. In this paper, we have shown that PSA could also be indicative of BOO, not only with respect to observational data but also with regard to statistically relevant correlations between PSA and PdetQmax. Therefore, PSA levels should be taken into account and could even be used as a biomarker when a treatment strategy is determined or executed for patients suffering from clinical BPH. Additionally, the results obtained in this study indicate that inflammation can be correlated with PSA levels. This implies that elevated PSA levels should also be considered as predictive for prostate inflammation as well as for prostate cancer.

ACKNOWLEDGEMENTS

COMMENTS

Background

Since its introduction, prostate-specific antigen (PSA) has played a key role in prostatic evaluation. However, it is not a cancer specific parameter but merely organ specific. Consequently, elevated PSA levels are a challenging problem for urologists in assessing or excluding potential life-threatening prostate cancer. A better understanding of other possible mechanisms causing PSA elevation is required. Therefore, the aim of this study was to investigate the relationship between PSA and the degree of prostate inflammation and to investigate whether PSA levels can be used as a biomarker for bladder outlet obstruction (BOO).

Research frontiers

The cause of elevated PSA serum levels is not always prostate cancer. In the area of interpreting PSA elevation, the research hotspot is to have an insight in to other mechanisms that can be responsible for a PSA elevation, apart from prostate cancer.

Innovations and breakthroughs

In this study the authors showed a correlation between BOO ($PdetQ_{max}$) and PSA (logarithmic). Furthermore, the authors demonstrated a weak correlation between PSA (logarithmic) and active as well as chronic prostatic inflammation.

Applications

PSA could also be indicative of BOO, not only with respect to observational data but also with regard to statistically relevant correlations between PSA and $PdetQ_{max}$. Therefore, PSA levels should also be taken into account and could even be used as a biomarker when a treatment strategy is determined or executed for patients suffering from clinical benign prostate hyperplasia (BPH). Additionally, the results obtained in this study indicate that inflammation can be correlated with PSA levels. This implies that elevated PSA levels should also be considered as predictive for prostate inflammation as well as for prostate cancer.

Terminology

BOO: a blockage at the base of the bladder that reduces or prevents the flow of urine into the urethra, the tube that carries urine out of the body. This condition is most common in aging men. It is often caused by BPH. As a man ages, his chance of developing these diseases increases dramatically; Prostate inflammation: inflammation of the prostate gland. There are four types of prostatitis: acute bacterial prostatitis, chronic bacterial prostatitis, chronic prostatitis without infection, asymptomatic inflammatory prostatitis; PSA (prostate-specific antigen): a protein manufactured exclusively by the prostate gland. PSA is produced for the ejaculate where it liquefies the semen and allows sperm cells to swim freely. Elevated levels of PSA in blood serum are associated with benign prostatic hyperplasia and prostate cancer.

Peer review

This prospective analysis investigates the association of high PSA levels and bladder outflow obstruction and prostatic inflammation. Its results, as described in the discussion, add to the literature and the controversy that exists in these issues among other studies.

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