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Immunological Interactions and Inflammatory Changes in the Pathways of Benign Prostatic Hyperplasia and Prostate Cancer

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Abstract

The prostate is the unity of the accessory glands in the male reproductive system. It plays a crucial role in maintaining sperm vitality by secreting components that form part of the semen. Prostate diseases, such as prostatic hyperplasia (BPH) and prostate cancer (PCa), are associated with inflammatory changes that affect male fertility. Research suggests that prostatitis may be a precursor to the development of these conditions. The study included 84 men aged 35-79 years, divided into three groups: benign prostatic hyperplasia (HP), prostate cancer (PCa), and a control group. Cytokine concentrations of IL-6, IL-8, TNF-α, and C-reactive protein were restrained using ELISA. The results showed significant differences at a confidence level of $P<0.05$ between groups in IL-8 levels, with the prostate cancer group recording the highest concentration. No significant differences at a confidence level of $P<0.05$ were observed in IL-6 levels. Significant differences were noted in TNF-α and C-reactive protein levels between groups at a confidence level of $P<0.05$. Previous research highlights the role of cytokines in the development of prostate cancer, but data on their specific role is limited. Elevated IL-6 and IL-8 levels are associated with illness progression. Moreover, elevated TNF-α and C-reactive protein points are linked to the assistance of benign and malignant prostatic hyperplasia. According to the result and statical analysis study offers extra sign of the title role of cytokines IL-8 and TNF-α in the distinguishing prostate cancer from other conditions underscoring the importance of targeting these molecules in diagnosis and treatment.

Introduction:

The prostate is one of the attachment glands of the man generative system, located around the urethra just after it exits the bladder. Its primary function is to secrete components and fluids that form part of the semen and help maintain sperm vitality. The functional relationship between the prostate and testes dates back over two hundred years. In 1786, John Hunter described in his book "Observations on the Glands between the Rectum and the Bladder [1]. The testes contribute to

sperm production (gametes), the addition glands secrete various proteins such as prostatic kallikrein (KLKs) and seminalgellin from the seminal vesicles, growth factors like testosterone, and insulin-like growth factor 3 from Leydig cells [2]. Recent data also indicate prostatitis as a precursor to benign prostatic hyperplasia (BPH) [3] and prostate cancer (PCa) [4]. Prostate diseases can directly affect male fertility, whether young or old, by altering the gland's metabolic state or causing inflammatory conditions [5]. Benevolent prostatic hyperplasia (BPH) is one of the most common conditions among people starting in their fourth decade of life. It affects about 30-40% of men, with prevalence increasing linearly to 70-80% in those over 80 years old [6][7]. Prostate cells are prone to tumor formation, often occurring in the post-midlife stages [8]. The peripheral zone of the prostate in adult men constitutes more than 70% of the prostate tissue mass and significantly contributes to normal prostate function. Moreover, it is the most common site for tumor development, with 80% of tumors originating in this area [9]. Prostatitis can affect a man's quality of life through urinary symptoms, pain, and sexual dysfunction [10]. The prostate has several natural define devices in contrast to infection, together with the manufacture of antimicrobial substances and mechanical expulsion of the prostatic urethra through urination and ejaculation [11]. Risk factors for bacterial prostatitis include urethral stricture, surgical interventions in the urethra, transrectal prostate biopsy, and sexually transmitted urethritis [12]. Cytokines play a key part in promoting and regulating the immune response, including cell movement, diversity, proliferation, and the production of other cytokines [13]. Cytokines act on signing particles and cells, directing them to sites of irritation, infection, and injury, and influencing lymphocyte growth and other biotic functions. Cytokines can perform at their location of production (autocrine), on in-line cells (paracrine), or distant cells (endocrine) [14]. Many types of cytokines have been exposed, including chemokines, interferons (IFN), interleukins (IL), lymphokines, and tumor necrosis factor (TNF) [15]. Cytokines are extracellular peptides/glycoproteins with low molecular weight that direct signals between cells, formed by numerous resistant cells, exclusively T cells, neutrophils, and macrophages. Tumor necrosis factor-alpha (TNF-α) is a strong anti-inflammatory cytokine that touches many cell kinds and plays a central role in the progress of chronic inflammatory diseases such as rheumatoid arthritis [16]. It is known for its robust anti-tumor activity in vivo [17] and in cell culture experiments [18]. TNF-α is a peptide intermediary of many cellular rejoinders, including apoptosis, necrosis, and proliferation [19], produced by a precursor known as Transmembrane TNF-α, which is uttered on the cell outward as a second type of peptide in activated macrophages, lymphocytes, and other cell types, be made up of 233 amino acids with a weight of 26 kilodaltons [20]. The function of TNF-α depends on its interaction with its receptors, which can bind to similar and different molecules, enabling it to activate both communal and different pathways. TNF-R1 is related to actuating inflammatory and cell death pathways, while TNF-R2 is associated with tissue overhaul and angiogenesis [21].

Interleukins are a collection of veiled proteins with various organizational and purposeful roles. These proteins fix to their receptors and take part in communication between white blood cells, being closely linked to immune activation and cell division. They are mainly produced by helper CD4+ T lymphocytes, monocytes, macrophages, and endothelial cells [22]. The IL-6 family includes IL-6, IL-7, ciliary neurotrophic factor (CNTF), leukemia inhibitory factor (LIF), oncostatin M (OSM), cardiotrophin 1 (CT-1), cardiotrophin-like cytokine (CLC), and IL-27. These cytokines are categorized into a single family because each cytokine's receptor compound holds either one or two subunits of IL-6 and IL-27 [23]. The IL-6 family participates in various functions, including B cell stimulus and hepatic acute-phase protein initiation, with metabolic and neurological roles also

accredited to this family of cytokines. Chemokines switch the migration and transport of homeostatic immune cells such as neutrophils, B lymphocytes, and monocytes among the bone marrow, blood, and close tissues, thus categorizing them as chemotactic cytokines [24]. There are 50 human chemokines classified into tetrad relations, with the two main families being CC chemokines, where cysteine residues are together, and CXC chemokines, where cysteine remains are detached by one amino acid residue. IL-8, or CXCL8, is the most famous CXC chemokine responsible for recruiting neutrophils and maintaining inflammatory responses. Monocyte chemoattractant protein-1 (MCP-1), CCL2, and CCL11 are samples of CC chemokines that recruit various leukocytes, especially monocytes, and eosinophils [25]. Some trainings have confirmed the role of cytokines in the evolution of varied malignancies, but data related to prostate cancer are limited [26]. The limited data on cytokine involvement in prostate cancer are attributed to the multi-step process of prostate cancer development and progression, which involves multiple growth factors, hormones, and cytokines [27]. The aim of this project to ensure the role of some cytokines in the developed of prostate hyperplasia and cancer.

Methods

The study included 84 men from consultations with specialists in Samarra and the Oncology Centre at Tikrit Military Hospital, aged 35-79 years, from September 2023 to April 2024, divided into three groups: Group 1: Benign Prostatic Hyperplasia (HP) consisting of 48 men. Group 2: Prostate Cancer (PCa) involving of 12 men. Group 3: Control Group (Co) containing of 24 men. The concentrations of immune variables IL-6, IL-8, TNF-α, and C-reactive protein were measured using the ELISA technique, a widely used method in scientific research and medicine for determining and measuring specific compounds in biological samples grounded proceeding antibody-antigen contacts through the enzyme-linked immunosorbent assay [28]. The procedures followed were according to the method provided by MybioSourceChina for IL-6, IL-8, TNF-α [29,30,31], and DemeditecGermany for C-reactive protein tests [32].

Statistical Analysis

Arithmetical investigation of immune parameters stood made with one-way ANOVA, a statistical method used to compare means of three or more independent (unrelated) groups to determine if there are significant differences between them at a confidence level of $P<0.05$. The null hypothesis, which assumes that all means are the same, is tested. One-way ANOVA relies primarily on the F-test, which forms the basis for determining whether there are significant differences between means of multiple groups [33].

Results

Table (1) shows the statistical analysis for immune variables including IL-6, IL-8, TNF-α, and C-reactive protein for the study groups. The results for IL-8 showed significant differences between the three study groups at a confidence level of $P<0.05$, with the prostate cancer group recording the highest mean of 123.0±26.1 pg/ml, followed by the control group with a second mean of 99.4±19.3, and the benign prostatic hyperplasia group with a mean of 75.8±14.8. No significant differences were observed in IL-6 levels across the trio clusters at a sureness level of $P<0.05$. The IL-6 concentrations in the benign prostatic hyperplasia group had the highest mean of 6.060±2.010 pg/ml, followed by the control group with a mean of 5.830±1.807, and the prostate cancer group with a mean of 3.142±1.445. Furthermore, significant differences were observed in TNF-α levels among the three groups at a confidence level of $P<0.05$. The prostate cancer group had the highest mean value of 75.50±9.11 pg/ml, followed by the benign prostatic hyperplasia group with a second

mean of 43.92±8.05, and the control group with the lowest mean of 23.17±3.86. Similarly, important variances were well-known in C-reactive protein heights among the three groups at a confidence level of P<0.05, with the benign prostatic hyperplasia group having the highest mean of 10.99±3.88 µg/ml, followed by the prostate cancer group with a mean of 6.34±2.41, and the control group with the lowest mean of 2.6±0.746.

***Table** (1) Mean values for IL-6, IL-8, TNF, and C-reactive protein for benign prostatic hyperplasia, prostate cancer, and control groups.*

Studied Variables	Prostate Cancer mean±S.D n=12	Hyperplasia mean±S.D n=48	Control mean±S.D n=24
IL-6 pg/ml	3.142 ± 1.445 a	6.060 ± 2.010 a	5.830 ± 1.807 a
IL-8 pg/ml	123.0 ± 26.1 a	75.8 ± 14.8 b	99.4 ± 19.3 c
TNFα pg/ml	75.50 ± 9.11 a	43.92 ± 8.05 b	23.17 ± 3.86 c
C-reactive ug/ml	6.34 ± 2.410 b	10.99 ± 3.880 a	2.60 ± 0.746 c

Note: - the similar litters mean no significance, the different litters there are significance

Discussion

Katongole et al., study indicated no significant and notable differences in the concentrations of various cytokines, including IL-6 and IL-8, in the sera of prostate malignancy patient protagonists compared to healthy individuals. However, a significant correlation was found between prostate-specific antigen (PSA) and cytokine attentions of IL-6 and IL-8 at a confidence level of p<0.05. That study concluded that elevated stages of IL-6 and IL-8 provide additional evidence for predicting prostate cancer, and these raised levels stay related using developed PSA levels and illness harshness [34]. On the other hand, another study confirmed the protagonist of cytokines cutting-edge the onset and development of prostate melanoma [26]. A notable role was observed for several interleukins and cytokines, especially interleukin 8 (IL-8), which remained knowingly high in in-patient roles with prime prostate cancer linked to good persons [35]. Cole and Boer reported that IL-6 levels were elevated in prostate cancers and the growth microenvironment and this remained related to unfortunate sickness outcomes and growth variation [36]. Maynard et al., recognized IL-8 as a cytokine with in height countenance in both main and metastatic prostate cancer-affected roles compared to healthy persons [35]. Additionally, Furtosi [37] discussed the relationship between insulin resistance and certain inflammatory markers in prostate cancer patients, noting significantly elevated points of cytokine IL-6 and tumour necrosis factor-alpha (TNF-α) in those with prostate cancer and benign prostatic hyperplasia. Supporting this, another study [38] emphasized the limited title role of C-reactive protein in predicting prostate malignancy suitcases, suggesting it is less useful for patients undergoing treatment for urological cancers. In Iraq, a study found increased levels of various inflammatory markers, including IL-6, IL-8, PSA, and C-reactive protein, in individuals with benign prostatic hyperplasia compared to healthy individuals. This study showed statistically significant differences between those with prostate issues and healthy individuals. Increased levels of C-reactive protein were also associated with higher lymphocyte counts [39]. Furthermore, another study [40] reported elevated TNF levels in

patients with benign and malignant prostatic hyperplasia. An increase in C-reactive protein levels was also observed among individuals with benign and malignant hyperplasia [41].

Conclusion

The study highlights that IL-8 and TNF-α planes are meaningfully advanced in prostate malignance patients associated to benign prostatic hyperplasia and control groups, suggesting their potential as biomarkers for prostate cancer. IL-6 showed no significant differences across the groups, while C-reactive protein was higher in benign prostatic hyperplasia but less useful for prostate cancer prediction. These findings support the role of IL-8 and TNF-α in distinguishing prostate cancer from other conditions.

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التداخلات المناعية والتغيرات الالتهابية في مسارات تطور تضخم البروستاتا الحميد وسرطان البروستاتا

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الخلاصة:

تعتبر البروستاتا إحدى الغدد التبعية في الجهاز التناسلي الذكري، وتلعب دورًا مهمًا في الحفاظ على حيوية الحيوانات المنوية من خلال إفراز مكونات تشكل جزءًا من السائل المنوي. ترتبط أمراض البروستاتا مثل تضخم البروستاتا الحميد (BPH) وسرطان البروستاتا (PCa) بتغيرات التهابية تؤثر على خصوبة الذكور. تشير الأبحاث إلى أن التهاب البروستاتا قد يكون مقدمة لتطور هذه الحالات. الدراسة شملت 84 رجلاً تتراوح أعمارهم بين 35-79 سنة، تم تقسيمهم إلى ثلاث مجموعات: تضخم البروستاتا الحميد (HP)، سرطان البروستاتا(PCa) ، ومجموعة السيطرة. تم قياس تراكيز السيتوكينات IL-6 ، IL-8، TNF-α، و C-reactive proteinباستخدام تقنية ELISA..أظهرت النتائج فروقًا معنوية بين المجاميع في مستويات IL-8، حيث سجلت مجموعة سرطان البروستاتا أعلى تركيز. بينما لم تُظهر مستويات IL-6 فروقًا معنوية. كما لوحظت فروق معنوية واضحة في مستويات TNF-α و C-reactive proteinبين المجموعات. تشير الأبحاث السابقة إلى دور السيتوكينات في تطور سرطان البروستاتا، لكن البيانات المتعلقة بدورها محدودة. يرتبط ارتفاع مستويات IL-6 و IL-8 بزيادة في مستضد البروستاتا النوعي (PSA) وتفاقم المرض. علاوة على ذلك، يرتبط ارتفاع مستويات TNF-α و C-reactive proteinبتضخم البروستاتا الحميد والخبيث. تقدم هذه الدراسة أدلة إضافية على دور السيتوكينات في تطور أمراض البروستاتا، مما يعزز من أهمية استهداف هذه الجزيئات في التشخيص والعلاج.