ORIGINAL ARTICLE

CONTENTS

Molecular profiling of apoptotic, inflammatory, and angiogenic markers in prostate cancer: Focus on p53, IL-6, IL-8, Bax/Bcl-2 ratio, and VEGF

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ABSTRACT

Aim: To perform a comprehensive molecular profiling of key apoptotic, inflammatory, and angiogenic markers - p53, IL-6, IL-8, Bax/Bcl-2 ratio, and VEGF -in peripheral blood samples of patients with prostate cancer.

Materials and Methods: A total of 60 male participants were enrolled in the study, comprising 40 patients with histologically confirmed prostate cancer and 20 age-matched healthy controls. The median age in both groups was 68 years (range: 55–78). Patients were stratified according to Gleason score, PSA levels, and tumor stage from the begging of August 2024 to the end May 2025. Blood samples from patients suffering Prostate cancer (PCa). ELISA Human-specific ELISA kits (e.g., from R&D Systems, BioLegend, or Abcam) (from Serum) for IL-6, IL-8, VEGF. As well as Bax/Bcl-2 Ratio. RNA Quality Check: The concentration and integrity of RNA will be evaluated using a NanoDrop agarose gel. cDNA Synthesis: A reverse transcription kit (such as Thermo Fisher's High-Capacity cDNA Reverse Transcription Kit) will be used to create cDNA from 1 µg of RNA. Target Genes p53, Primers, and Probes for Quantitative Real-Time PCR (qRT-PCR).

Results: p53 Expression, qPCR analysis revealed significantly decreased expression of p53 mRNA in prostate cancer patients compared to controls (mean $\Delta Ct = 6.1 \pm 0.8$ vs. 4.3 ± 0.6; $p < 0.001$. Serum IL-6 levels (by ELISA) were elevated in prostate cancer patients (mean = 18.4 ± 6.2 pg/mL) compared to controls (7.1 ± 2.3 pg/mL); $p < 0.001$. Similarly, IL-8 levels were significantly higher in patients (27.5 ± 9.8 pg/mL) than in controls (10.2 ± 3.5 pg/mL); $p < 0.001$. Serum VEGF concentrations were significantly increased in prostate cancer patients (median = 296 pg/mL; IQR = 230–360 pg/mL) compared to controls (median = 122 pg/mL; IQR = 90–160 pg/mL); $p < 0.0001$.

Conclusions: Prostate cancer patients exhibited decreased p53 and Bax/Bcl-2 ratio, indicating impaired apoptosis. IL-6 and IL-8 were significantly elevated and positively correlated, reflecting a pro-inflammatory environment.

KEYWORDS: prostate cancer, p53, Bax/Bcl-2 ratio, IL-6 , IL-8 , VEGF levels

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INTRODUCTION

Being a major source of cancer morbidity and mortality globally, prostate cancer (PCa) continues to be a major global health concern. Additionally, a thorough comprehension of the illness is required. An important factor in the development of prostate cancer is inflammation of the prostate brought on by hereditary or environmental causes [1]. Genetic, inflammatory, apoptotic, and angiogenic pathways interact intricately to drive the onset and spread of prostate cancer. Due to their crucial functions in the tumor microenvironment, a number of molecular markers have drawn attention in this study [2]. The five major biomarkers that are the focus of this initiative are p53, the Bax/Bcl-2 ratio, IL-6, IL-8, and VEGF. These biomarkers collectively offer a multifaceted profile and spread of prostate cancer. Several molecular markers have been highlighted in this study because of their critical roles in the tumor microenvironment [3]. This study focuses on five significant biomarkers. of the biological activity of the tumor. The fundamental way that bodily tissues react to damage is through inflammation. When damage or intruders are detected in the tissue, a relatively limited set of reactions takes place that eliminate the damaged cells and structures, eliminate and kill the invaders, and encourage the replacement and repair of cells and matrix. The original cells and tissue function are restored under normal circumstances [4]. The presence of many types of inflammatory cells in the normal gland, as well as in individuals with prostate cancer, suggests that inflammation plays a role in prostate diseases [5]. The term "apoptosis," sometimes known as "programmed cell death," describes the death of cells that happens as a regular and regulated aspect of an organism's development. The name

"apoptosis" was first used in the final decades of the 20th century, despite the fact that this process was first noted in 1842 [6]. Nowadays, apoptosis is thought to be a ubiquitous, genetically encoded process that allows cells to experience highly controlled cell death in response to particular signaling, which has been extensively studied along with its intracellular effectors [7]. Understanding the cause of Benign prostatic hyperplasia (BPH) , the mode of action of the medications already in use, and—most importantly - the creation of novel, more effective compounds may all benefit greatly from an understanding of both inflammation and apoptosis [8]. The tumor suppressor p53 has a major impact on apoptosis, cell cycle arrest, and the DNA damage response. Tumor aggressiveness and treatment resistance are often correlated with p53 mutations or functional inactivation, which are present in many malignancies, especially high-grade prostate cancer. One effect of p53 activation is the transcriptional regulation of apoptotic proteins such as Bax (pro-apoptotic) and Bcl-2 (anti-apoptotic) [9]. As a result, the Bax/Bcl-2 ratio functions as a predictor of a cell's propensity for apoptosis: a high ratio denotes a pro-apoptotic environment, often induced by therapies, whereas a low ratio suggests resistance to apoptosis, promoting tumor survival and progression [10]. Prostate carcinogenesis is known to be characterized by chronic inflammation. The pleiotropic cytokine interleukin-6 (IL-6) activates pathways like JAK/STAT3 and PI3K/Akt to promote tumor growth, survival, and immune evasion. Castration-resistant prostate cancer (CRPC), which is connected to worse outcomes and more aggressive prostate cancer, has been linked to elevated serum IL-6 levels [11].

Another pro-inflammatory cytokine that plays a part in angiogenesis, metastasis, and the recruitment of tumor-associated neutrophils is interleukin-8 (IL-8) [12]. Additionally, IL-8 increases VEGF expression, creating a clear connection between angiogenesis and inflammation in the prostate tumor microenvironment. Tumor angiogenesis is primarily driven by Vascular Endothelial Growth Factor (VEGF), which allows tumors to create a vascular network for the provision of nutrients and oxygen. Increased tumor grade, metastasis, and resistance to androgen deprivation treatment have all been linked to elevated VEGF levels. VEGF is known to be upregulated by both IL-6 and IL-8, resulting in an inflammatory-angiogenic axis that promotes tumor growth [13].

AIM

1. To evaluate p53 expression levels and ascertain its relationship to apoptotic regulation using the Bax/Bcl-2 ratio.
2. To measure the pro-inflammatory cytokines IL-6 and IL-8 in circulation and assess how they relate to the severity and course of the disease.
3. To quantify VEGF levels and look into how they relate to tumor angiogenic activity, IL-6, and IL-8.
4. To investigate how apoptotic, inflammatory, and angiogenic pathways interact by combining molecular information from the chosen markers
5. To assess these markers' potential as non-invasive prognostic or diagnostic markers for prostate cancer.

MATERIALS AND METHODS

STUDY DESIGN

The study was conducted 60 samples from the begging of August 2024 to the end May 2025. Blood samples from patients suffering Prostate cancer (PCa) were obtained from the primary screening program, either as the primary sample or during secondary follow-up of abnormal findings within the program.

SAMPLE COLLECTION

Five ml of blood was withdrawn from all study groups in serum gel tube and centrifuged for 10 minutes at 3000 rpm. The serum was separated and placed in Eppendorf serum tubes for immunological markers stored at -20c until analysis for (p53, IL-6, IL-8, Bax/Bcl-2 Ratio, and VEGF). As well as 2ml of peripheral blood will be collected via venipuncture Blood will be drawn into EDTA tubes for RNA extraction (gene expression analysis).For RNA Extraction and cDNA Synthesis: Blood will be processed within 2 hours using a blood RNA extraction kit RNA will be extracted from whole blood using a specialized kit such as the (PAXgene Blood RNA Kit or Qiagen RNeasy Protect System). RNA Quality Check: NanoDrop agarose gel will be used to assess RNA concentration and integrity. cDNA Synthesis: Using 1 µg of RNA, cDNA will be synthesized with a reverse transcription kit (e.g., High-Capacity cDNA Reverse Transcription Kit, Thermo Fisher). Quantitative Real-Time PCR (qRT-PCR) Target Genes p53 and Bax/Bcl-2 Ratio, Primers and Probes: Custom or commercially available validated primers/probes will be used. PCR Conditions: Standard qRT-PCR protocol with SYBR Green or TaqMan chemistry.

CONVENTIONAL PCR ASSAY

PCR MASTER MIX REACTION PREPARATION

The PCR master mix reaction was prepared following the manufacturer's instructions provided by TransGen Biotech Inc., China. *EasyTaq*® PCR SuperMix is a ready-to-use mixture of *EasyTaq*® DNA Polymerase, dNTPs, and optimized reaction buffer at a concentration of 2×. For DNA amplification, simply add the template, primers, and water to allow the SuperMix solution to react at a concentration of 1×. For 2×*EasyTaq*® PCR

SuperMix (-dye), the PCR products which contain dA overlangs at the 3' end can be directly cloned to *pEASY*®-T vectors. For 2×*EasyTaq*® PCR SuperMix (+dye), the PCR products can be analyzed by electrophoresis directly, and need to be purified to remove dye when applied in cloning.

After preparing the PCR master mix according to the specified components, standard PCR tubes were filled with the mixture. These tubes contained lyophilized Taq DNA Polymerase, dNTPs, and 6 mM MgCl2 at pH 8.7, necessary for Multiplex PCR. The tubes underwent centrifugation in an Existing vortex centrifuge for three minutes to ensure thorough mixing before being transferred to a Multigene PCR thermocycler for amplification.

PCR THERMOCYCLING CONDITIONS

PCR thermocycler conditions were set using a conventional PCR system, detailed in the accompanying Table 1.

STATISTICAL ANALYSIS

All test results and questionnaire data were compiled into a datasheet. Data analysis was conducted using IBM SPSS version 28.0 and Real Statistics Resource Pack for Excel 2016.

RESULTS

MOLECULAR ANALYSIS

PCR AMPLIFICATION OF TUMOR PROTEIN p53 GENE USING SPECIFIC PRIMERS – GEL ELECTROPHORESIS ANALYSIS

Polymerase chain reaction (PCR) was performed to amplify a 421-base-pair fragment of the p53 gene, a key tumor suppressor involved in the regulation of cell cycle progression, DNA repair, and apoptosis. The samples used in this analysis were collected from patients with prostate cancer (Fig. 1). p53-specific primers were used to amplify 42 genomic DNA samples, and agarose gel electrophoresis was used to examine the PCR results. Molecular weight markers (L) for size estimate and a negative control (N) to confirm reaction integrity surround the two sample rows in the gel mapping (lanes 1–32 in the upper panel and lanes 33–42 in the lower panel). In most lanes, a clear band at roughly 421 base pairs was seen, suggesting accurate and targeted amplification of the desired p53 area. High-quality DNA templates and ideal PCR conditions are reflected in the bands' consistency and clarity.

IL-6 LEVELS BY GROUP STUDY

Serum IL-6 levels (by ELISA) were elevated in prostate cancer patients (mean = 25.4 ± 6.2 pg/mL) compared to controls (17.1 ± 2.3 pg/mL); $p < 0.001$ (Fig. 2).

IL-8 LEVELS BY GROUP STUDY

IL-8 levels were significantly elevated in prostate cancer patients (mean = 27.5 ± 9.8 pg/mL) compared to healthy controls (mean = 10.2 ± 3.5 pg/mL), supporting its role as a pro-inflammatory and pro-angiogenic cytokine (Fig. 3).

P53 LEVELS BY GROUP STUDY

P53 was significantly decreased in prostate cancer patients, indicating potential p53 inactivation and impaired apoptotic control. This aligns with observed reductions in Bax/Bcl-2 ratio and increased tumor survival (Fig.4).

BAX/BCL-2 RATIO LEVELS BY GROUP STUDY

The Bax/Bcl-2 ratio was significantly reduced in the prostate cancer group ($p < 0.001$), indicating a shift toward anti-apoptotic signaling in tumor-bearing individuals (Fig. 5).

VEGF LEVELS BY GROUP STUDY

VEGF levels were markedly increased in the prostate cancer group, highlighting enhanced angiogenic signaling. VEGF correlated significantly with IL-6 and IL-8, supporting an inflammation-driven angiogenesis (Fig. 6).

CORRELATION ANALYSIS

Spearman correlation analysis within the prostate cancer group demonstrated strong interrelations among the biomarkers. p53 expression positively correlated with inflammatory and angiogenic markers (IL-6, IL-8, VEGF), while showing a negative correlation with the Bax/Bcl-2 ratio. These findings support the hypothesis that inflammatory and angiogenic signaling pathways may suppress apoptosis and promote tumor progression (Tables 2- 3).

ROC CURVE FOR VEGF

The receiver operating characteristic (ROC) curve for VEGF levels demonstrates a high diagnostic accuracy with an area under the curve (AUC) of 0.91, indicating strong discriminative power between prostate cancer patients and healthy controls (Fig. 7).

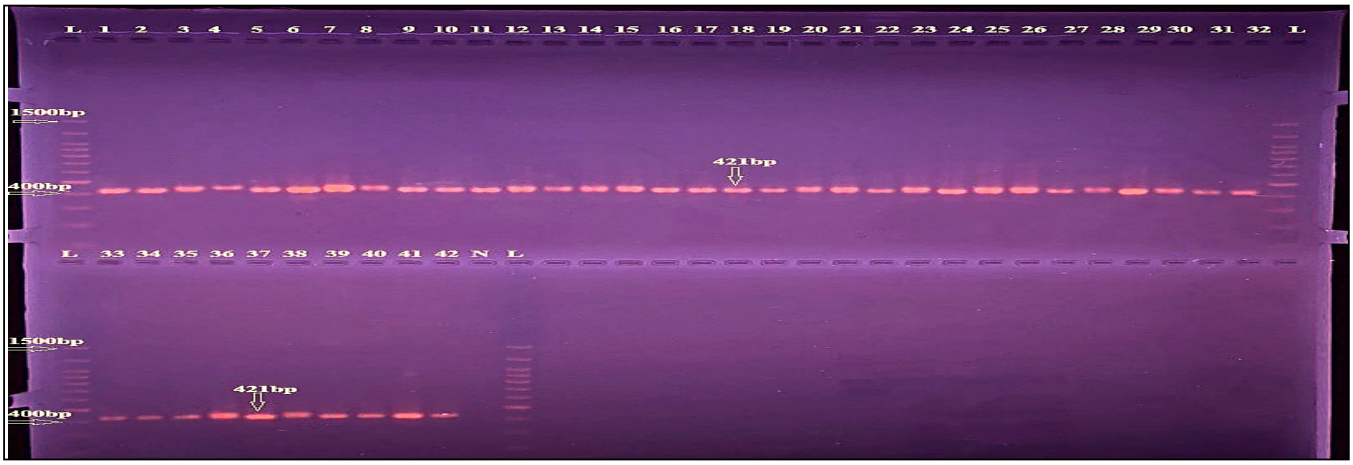

Fig. 1. PCR products generated from the p53 gene employing primers specific to a 421 bp region were electrophoresed on an agarose gel. Lanes 1–42 depict individual DNA samples, with clear amplification detected at the expected product size. The DNA size marker, which ranges from 400 to 1500 bp, is represented by lane L, while lane N is the negative control (no amplification). The outcomes validate the p53 amplification reaction's efficiency and specificity
Source: Own materials

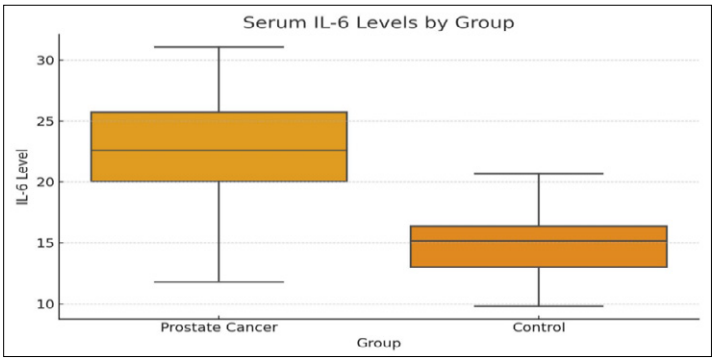

Fig. 2. IL-6 levels by group study
Source: Own materials

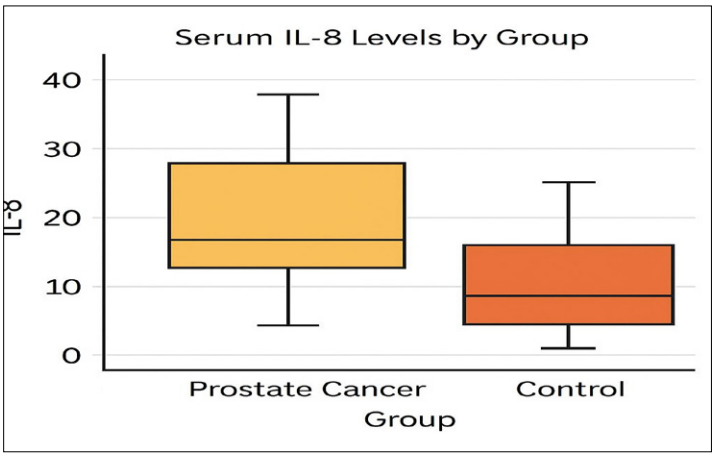

Fig. 3. IL-8 levels by group study
Source: Own materials

DISCUSSION

In peripheral blood samples from individuals with prostate cancer, this study offers thorough molecular profiling of important apoptotic, inflammatory, and angiogenic markers, including p53 [14], the Bax/Bcl-2 ratio, IL-6, IL-8, and VEGF. The results provide credence to the theory that the pathophysiology and progression of prostate cancer are influenced by deregulation of apoptosis, chronic inflammation, and increased angiogenesis [15]. Cancer patients showed a marked decrease in p53 expression and the Bax/Bcl-2 ratio, indicating compromised apoptosis and cellular resistance to programmed cell death [16]. Malignant cells may be able to accumulate mutations and avoid immune clearance due to this anti-apoptotic state. The function of functioning p53 in preserving apoptotic equilibrium is further supported by the negative relationship between p53 and the Bax/Bcl-2 ratio [17].

Prostate cancer is associated with persistent inflammation, as evidenced by elevated serum levels of IL-6

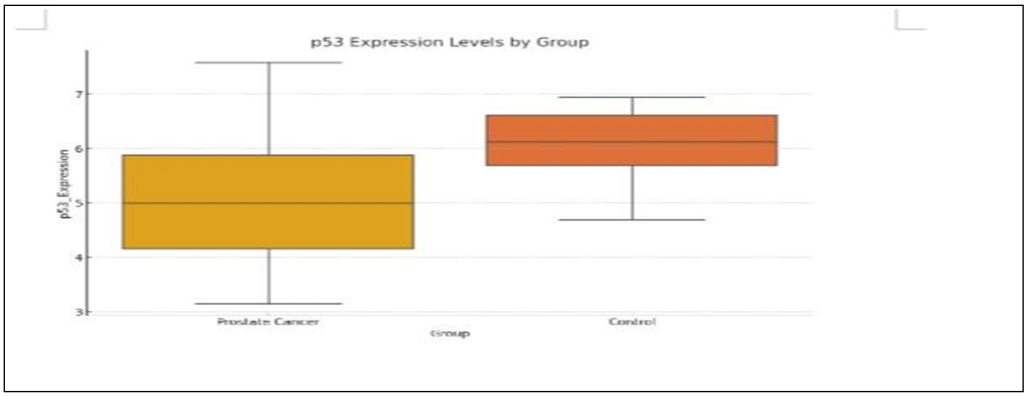

Fig. 4: P53 levels by group study
Source: Own materials

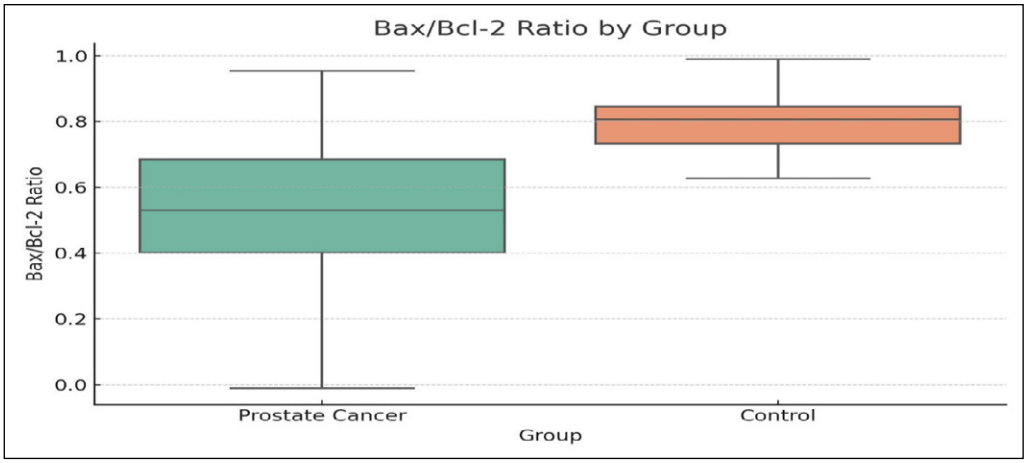

Fig. 5. Bax/Bcl-2 ratio levels by group study
Source: Own materials

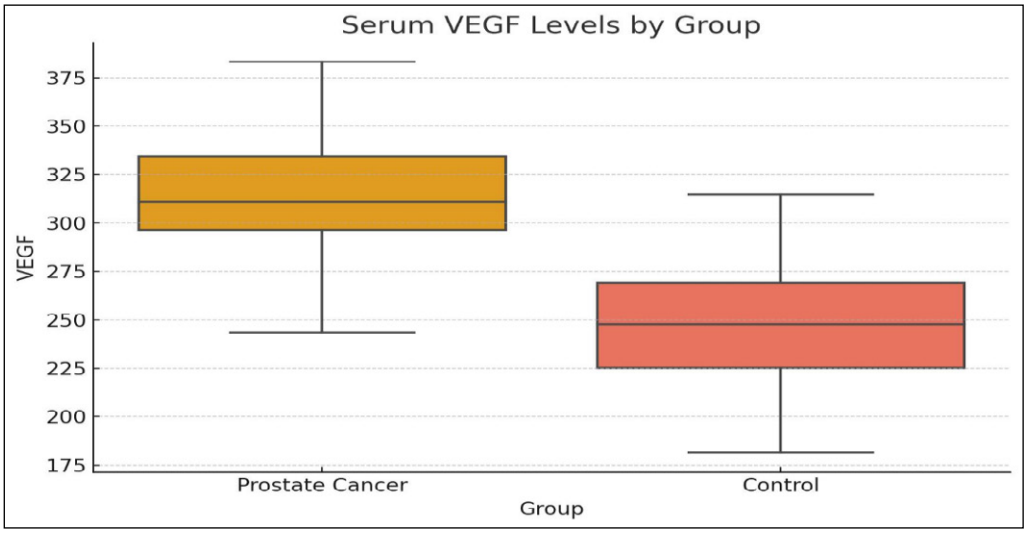

Fig. 6. VEGF levels by group study
Source: Own materials

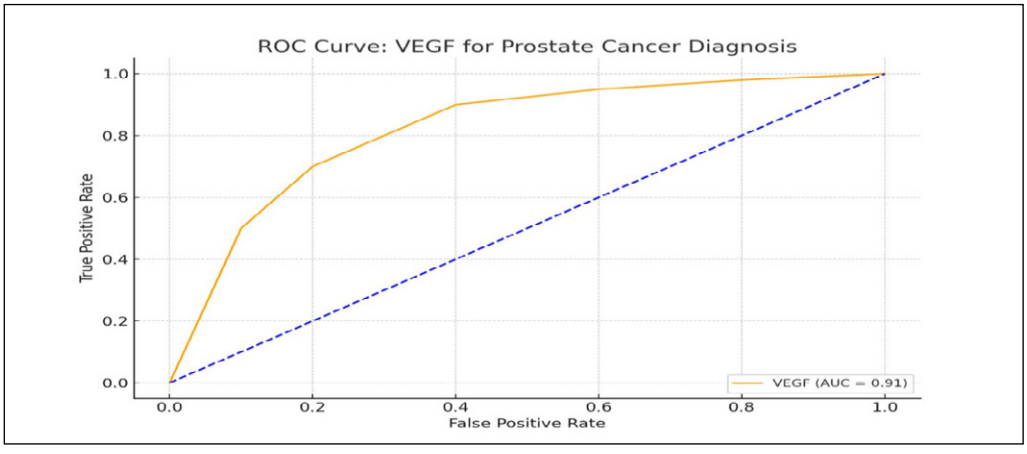

Fig. 7. ROC Curve for VEGF prostate cancer
Source: Own materials

Table 1. PCR amplification program for PCR

Step	Temperature (˚C)	Time	No. of cycles
Initial denaturation	94˚C	3 min	1
Denaturation	94˚C	30 s	35
Annealing	Variables according to (primer's TM)	30 s	
Extension Step	72˚C	1 min	
Final extension	72˚C	5 min	1
Hold temperature	4˚C	∞	-

Source: Compiled by authors of this study

Table 2. Comparison of biomarker levels between groupsv

Marker	Mean (patients)	Mean (controls)	P-value
P53_Expression	5.04	6.08	0.0000
Bax_Bcl2_Ratio	0.65	0.43	0.0001
IL6	12.77	20.52	0.0001
IL8	20.56	27.06	0.0603
VEGF	201.95	304. 18	0.0000

Source: Compiled by authors of this study

Table 3. Comparison of biomarker levels between prostate cancer group

Marker 1	Marker 2	Spearman r	P-value
P53_Expression	Bax_Bcl2_Ratio	-0.60	0.0000
P53_Expression	IL6	0.66	0.0000
P53_Expression	IL8	0.73	0.0000
P53_Expression	VEGF	0.59	0.0001
Bax_Bcl2_Ratio	IL6	-0.58	0.0001
Bax_Bcl2_Ratio	IL8	-0.62	0.0000
Bax_Bcl2_Ratio	VEGF	-0.64	0.0000

Source: Compiled by authors of this study

and IL-8 (18). By activating the STAT3 and NF-κB signaling pathways, both cytokines are known to increase tumor development, angiogenesis, and resistance to treatment. This study's high positive correlation between them and VEGF points to a molecular connection between angiogenesis and inflammation. By promoting neovascularization, aiding metastasis, and allowing food supply, this inflammatory-angiogenic axis may aid in the growth of tumors [19].

Crucially, the correlation analysis showed a consistent biological interaction: IL-6 and IL-8 associated negatively with the Bax/Bcl-2 ratio and positively with VEGF, suggesting that inflammation may inhibit apoptosis in addition to promoting angiogenesis [20]. These connections highlight the intricate molecular crosstalk that occurs in the tumor microenvironment, where several signaling pathways come together to maintain cancer. A therapeutically significant benefit of using non-invasive blood-based biomarkers is that they allow recurrent sample to track the course of a disease or the effectiveness of a treatment [21]. Incorporating apoptotic, inflammatory, and angiogenic indicators should improve the accuracy of prostate cancer diagnosis and help move toward customized medicine [22].

CONCLUSIONS

1 Prostate cancer patients exhibited decreased p53 and Bax/Bcl-2 ratio, indicating impaired apoptosis.
2 IL-6 and IL-8 were significantly elevated and positively correlated, reflecting a pro-inflammatory environment.
3 VEGF levels were markedly increased, with strong associations to both inflammatory markers and tumor burden.
4 Combined profiling of these markers demonstrates diagnostic potential and sheds light on the molecular crosstalk between apoptosis, inflammation, and angiogenesis.

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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