A 10-Year Experience of Prostate Cancer Management in Two Centers in Assiut

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Abstract:

Background: Prostate cancer is the second most common cancer worldwide and the fifth leading cause of cancer-related death 2022. In Egypt, it ranks fourth, accounting for 7% of male cancers. Primary endpoint of this study was to evaluate clinical and pathological characteristics, treatment protocol and outcomes of patients with prostate cancer in two specialized oncology centers in Assiut over 10 years, while secondary endpoints were DFS, PFS, and OS. Ethical approval was obtained from the Faculty of Medicine, Assiut University (IRB 04-2023-200109).

Methods: Retrospective review of patients from June 2013 to June 2023 was conducted, collecting demographic data, clinicopathologic features, diagnostic imaging, PSA levels, treatment lines, and follow-up information

Results: 100 were eligible. Stage III and IV disease observed in 26 and 74 patients, respectively. Treatment included radiotherapy (RT) in 68 patients, prostatectomy in 1, and chemotherapy (docetaxel) in 9. Androgen deprivation therapy was surgical in 2 and medical in 98. The median pretreatment PSA was 96.8 ng/mL, decreasing significantly post-treatment to 72.5 ng/mL. A significant positive correlation existed between PSA decline at 3 months and OS ($r=0.221$, $p=0.031$) and PFS ($r=0.791$, $p<0.001$). The median OS was 59 months; patients not receiving RT had higher mortality risk (HR=3), while greater PSA decline was associated with lower mortality (HR=0.98). Median PFS was 51 months, with similar predictors.

Conclusions: Radiotherapy offers a protective effect, and PSA decline serves as a valuable prognostic indicator in prostate cancer outcomes.

Keywords: Prostate Cancer, Upper Egypt, Prostate-specific antigen, Gleason score, Radiotherapy, Androgen Deprivation Therapy

Received: 16 July 2025
Accepted: 30 July 2025

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Introduction:

Prostate cancer (PCa) is the world's second most frequent cancer and the fifth cause of cancer death among men in 2022[1].

In Egypt, PCa is the fourth most frequent cancer constituting 7% as an incidence in men [2].

The clinical presentation of prostate cancer can vary widely, ranging from asymptomatic cases detected through routine screening to advanced disease with metastatic spread. The most frequent complaint is difficulty with urination, increased frequency, and nocturia, symptoms that may also arise from prostatic hypertrophy. More advanced stage of the disease may present with urinary retention or back pain, as axial skeleton is the most common site of bony metastatic disease [3].

The risk factors related to prostate cancer include family risk, ethnicity, age, obesity, and other environmental factors [4].

Many men with prostate cancer are diagnosed by digital rectal examination, prostate-specific antigen (PSA) testing, magnetic resonance imaging (MRI), prostate biopsy and by screening [5]. Prostate cancer can either be classified as hormone sensitive or hormone resistant, which is an indicator of testosterone stimulation and guide to the possible treatment option [6].

According to ESMO guidelines, prostate cancer is classified to localized disease (low risk, intermediate risk or high risk) & metastatic disease (Metastatic hormone sensitive MHSPC or metastatic castrate resistant MCRPC) [7].

Treatment strategies for prostate cancer have evolved significantly in recent years; Treatment options available for prostate cancer are active surveillance, chemotherapy, radiation therapy, hormonal therapy and surgery [8].

Primary endpoint of this study was to evaluate clinical and pathological characteristics, treatment protocol and outcomes of patients with prostate cancer in two specialized oncology centers in Assiut over 10 years (South Egypt Cancer Institute & Assiut Clinical Oncology Department), while secondary endpoints were DFS, PFS, and OS.

Materials and Methods:

Materials

Data Collection and Study Tools

A comprehensive review of medical records was conducted to extract relevant data. The information collected included:

Demographic data: such as age, smoking and relevant medical history.

Clinical characteristics: including presenting symptoms and comorbidities were recorded.

Radiological data: (for diagnosis & to monitor treatment response)

- CT chest
- MRI Pelvi-abdomen.
- Bone scan or MRI whole spine in metastatic patients.

Pathological data:

- Biopsy result
- Gleason score
- TNM staging
- PSA levels at diagnosis and follow-up
- Patients with a pathologic GS of 8–10 and a stage of pT3b or N1 were considered to have 'unfavorable' pathological findings. Intraductal carcinoma was also regarded as an unfavorable disease.
- Prostate cancer risk stratification (low, intermediate& high) according to NCCN guidelines.

Biochemical test:

- PSA levels at diagnosis and on follow-up

Treatment plans:

1) Observation or active surveillance.
2) Surgery (type and extent).
3) Radiation therapy (technique, dose, fractionation).
4) Hormone therapy (type, duration).
5) Chemotherapy (regimens, cycles).

Follow-up data:

- PSA levels.
- Imaging studies (CT or MRI).
- Response to first-line treatment assessed by Response Evaluation Criteria in Solid Tumors version 1.1
- Disease status. Type and treatment in recurrence

The study evaluated the disease-free survival, progression-free survival and overall Survival.

Methods

Study design

This research uses a retrospective cohort study design which allows for the analysis of patterns and outcomes of prostate cancer patients over a 10-year between June 2013 and June 2023.

Study Setting

The study was conducted across two specialized oncology centers in Assiut, Egypt: Radiation Oncology Department at South Egypt Cancer Institute (SECI) and Clinical Oncology and Nuclear medicine Department at AUH.

Study outcome measures

Primary end points: To evaluate clinical and pathological characteristics, treatment protocol and outcomes of patients with prostate cancer.

Secondary end points:

- Disease free survival (DFS) defined as time from the date of radical treatment to the date of survival without any signs or symptoms of that cancer.
- Progression free survival (PFS) is estimated from diagnosis until progression of cancer.
- Overall survival (OS) time estimated from diagnosis until death from any cause or lost-to-follow-up.

Study Population:

Eligible cases were as follows:

a) Inclusion criteria:

- Diagnosed as prostate cancer from (June 2013-June 2023) proved by biopsy in South Egypt Cancer Institute "SECI" & Asyut clinical oncology department
- All clinical stages diagnosed by TNM staging system of AJCC 7th edition 2010 & 8th edition 2017

b) Exclusion criteria:

- Having other types of malignancy.
- Patients with a missing data.

Statistical analysis

- Data analysis was analyzed using SPSS version 26. Categorical data were presented in the form of frequencies and percentages. Numerical data were checked for normality by Shapiro-walk test and presented by mean and standard deviation or median and range according to their distribution.
- The Wilcoxon sign test was used to compare median differences of PSA pre and post treatment. Spearman correlation was used to identify correlation between OS, PFS and % of decline of PSA.
- Survival analysis was done using the Kaplan Meir curve to calculate overall survival and progression-free survival and compared by Log rank test. Univariate Cox regression analysis was performed to identify prognostic factors associated with OS and PFS, and significant variables entered in a multivariate Cox regression analysis adjusted with age to calculate adjusted hazardous ratio. The level of significance was considered at P value < 0.05.

Results:

The study included 100 patients with prostate cancer. Their mean age was 66.24±6.00 years, 57% of them were more than 65 years and 43.0%were less than 65 years old. 15% of cases have HTN, 6% have DM and 10 % have both HTN and DM. 16% of patients studied were smokers.

Regarding presentation of studied patients, the most common presentation was urological symptoms (61.0%) followed by bone pain (39.0%).

Magnetic resonance imaging (MRI) of the pelvic abdomen was done only in 61.0%, CT Pelvic abdomen only in15.0%, bone scan only in 6.0%, and combined images was done in 37.0% of patients.

Gleason Score was calculated from prostate biopsy cases, and it was as follows; 7.9% with score ≤6, 67.1% from 7 score and 25.0% with score from 8-10. Baseline patient characteristics are presented in Table 1.

Regarding staging of studied patients, Twenty-six patients (26.0%) were stage III and seventy-four patients (74%) were stage IV. Sixty-eight patients (68% of all patients representing 92% of metastatic patients) were de novo and six patients (6% of all patients representing 8% of metastatic patients) developed metastasis after treatment.

Risk classification for locally advanced disease (n=26 cases) was as follows, 96.2% were at high risk and 3.8% were intermediate risk with (unfavorable disease).

Metastatic disease was diagnosed in 74 cases, 81.1% were metastatic hormone sensitive prostate cancer (35% were low volume and 65% were high volume) and 18.9% were metastatic castrate resistant prostate cancer (21.4% were low volume and 78.6% were high volume). Clinical data is shown in Table 2.

All cases were on medical treatment (ADT) in the form of Goserelin (Zoladex) & Bicalutamide (Casodex) and two cases only were treated surgical (one among metastatic cases and other among non-metastatic cases). Radical prostatectomy was performed to one patient.

Radiotherapy (RT) was administered in 68% of studied patients, and the main form is palliative RT on bone (51.0%) followed by radical RT (15%) and both palliative and radical in 2.0%. More than half of the patients with non-metastatic disease (53.8%) received RT and all of them were radical. Seventy-three percent of metastatic cases received RT and the main form is palliative on bone (68.9%), RT to the primary tumor site in 1.4% and both palliative and radical in 2.7% of them.

Chemotherapy was administered to 9% of the studied patients, mainly MCRPC (8.0%) followed by MHSPC (1.0%) and all of them were metastatic,

Regarding novel hormonal agents: 7.0 % of studied patients take novel hormonal agents, and all of them were metastatic, and MCRPC. Lines of treatment are demonstrated in Table 3.

Follow up after 3 months of start of treatment and then after 6 months and after 1 year from start of treatment (patients were followed up every 6 months after this). Follow up was done by PSA and/imaging. The frequency of each follow up modality and treatment response assessment at different follow-up visits is given in Table 4.

The median level of pretreatment PSA was 96.8 ng/mL in the total studied patients. In non-metastatic cases, the median PSA level was 38.3 ng/mL and 100.0 ng/mL among metastatic cases.

At the first follow up (3 months from baseline visit) the median level of PSA was 12.4 ng/mL among total patients, 6.10 ng/mL among non-metastatic cases and 14.70 ng/mL among metastatic cases. There is a statistically significant decrease in PSA level post treatment compared to pretreatment. The percentage decline of PSA was 84.2% among total patients with prostate cancer, 80.85% among non-metastatic cases and 85.95% among metastatic cases. Prostate-specific antigen level before and after treatment is presented in Table 5.

In univariate Cox regression analysis, it was found that no statistically significant difference in hazardous ratio (HR) regarding various clinical, pathological and treatment parameters and OS. Except for patients who do not receive RT have higher HR and are associated with higher risk of mortality than patients received RT (HR=3.00, CI=1.05-8.54, p value=0.039), and patients with higher percentage of decline in PSA have lower HR and associated with lower risk of mortality (HR=0.98, CI=0.96-0.99, p value=0.006) as depicted in Table 6.

In the multivariate Cox regression: the significant variable in univariate cox regression were entered in multivariate Cox regression adjusted with age and the final model confirming data in univariate analysis with (HR=3.33, CI=1.09-10.18, p value=0.034) as for radiotherapy and (HR=0.97, CI=0.95-0.99, p value=0.003) as for percentage of decline in PSA as given in Table 6.

The only significant variable with PFS is radiotherapy, where patients receiving radiotherapy have higher PFS than patients not receiving radiotherapy (51.0 months versus 7 months respectively).

The univariate Cox regression analysis shows that patients with a higher percentage of decline in PSA have lower HR and are associated with lower risk of mortality (HR=0.96, CI=0.92-0.99, p value=0.049) and patients not receiving RT have higher HR than patients received RT (HR=12.8, CI=1.2-140.6, p value=0.037). The multivariate Cox regression confirming data in univariate analysis with (HR=0.96, CI=0.91-0.99, p value=0.049) as depicted in Table 7.

There was statistically significant positive correlation between % of decline and OS as with increase % of decline of PSA, the OS is increase (r=0.221, P- value=0.031 among total cases, r=0.242, P-value=0.042 among metastatic cases) as shown in Figure 1.

Regarding the correlation between % of decline of PSA with PFS, there was statistically significant strong positive correlation between % of decline and PFS as with increase % of decline of PSA, the PFS is increase (r=0.791, P- value<0.001) as demonstrated in Figure 2.

During the follow up, 16 died or lost follow up, 49 were alive and 35 were censored. The Kaplan-Meier estimate of OS of the studied patients with prostate cancer is shown in Figure 3.

The Kaplan-Meier estimate of PFS of the studied patients with prostate cancer is shown in Figure 4.

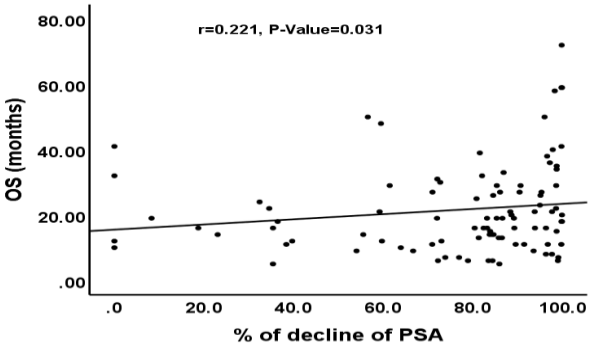

Figure 1: Scatter diagram for correlation between % of decline of prostate-specific antigen (PSA) and overall survival (OS)

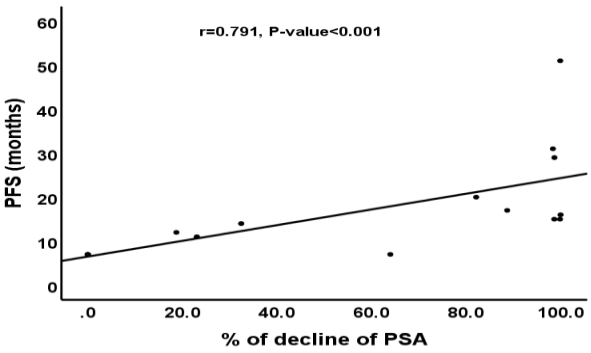

Figure 2: Scatter diagram for correlation between % of decline of prostate-specific antigen (PSA) and progression free survival (PFS)

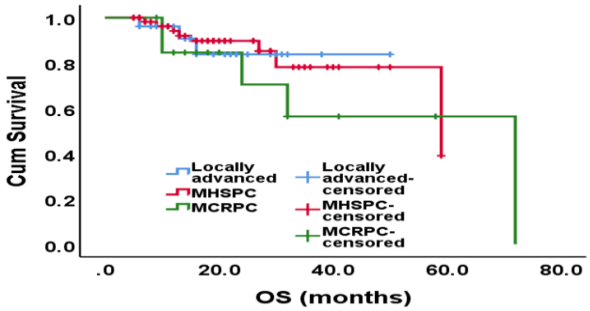

Figure 3: Kaplan-Meier estimates of the median overall survival (OS) of patients with advanced prostate cancer
Median OS for locally advanced = 43.95 months
Median OS for MHSPC = 59 months
Median OS for MCRPC = 72 months

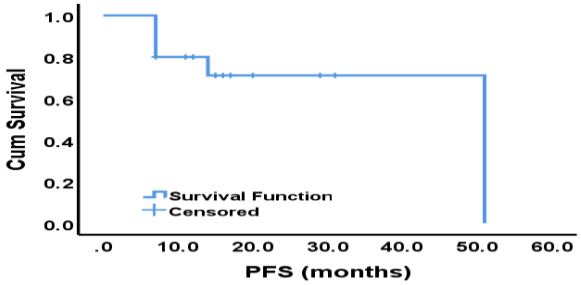

Figure 4: Kaplan Meier estimates of progression free survival (PFS) among studied patients with advanced prostate cancer (Median PFS = 51 months)

Table 1: Baseline characteristics of patients with prostate cancer

Variables	N=100	%
Age (years)		
▪ ≤65 years	43	43.0%
▪ >65 years	57	57.0%
Mean ± SD (range)	66.24±6.00 (53-81)	
Comorbidities		
▪ No	69	69.0%
▪ HTN	15	15.0%
▪ DM	6	6.0%
▪ Both HTN and DM	10	10.0%
Smoking		
▪ No	84	84.0%
▪ Yes	16	16.0%
Presentation		
Urological	61	61.0%
Bone pain	39	39.0%
Images used for diagnosis		
▪ MRI Pelvic abdomen only	42	42.0%
▪ CT Pelvic abdomen only	15	15.0%
▪ Bone scan only	6	6.0%
▪ Combined images	37	37.0%
GS score (n=76)	N=76	
▪ ≤6	6	7.9%
▪ 7	51	67.1%
▪ 8-10	19	25.0%

N: Number of patients, SD: Standard deviation, HTN: Hypertension, DM: Diabetes mellitus, MRI: Magnetic resonance imaging, CT: Computed tomography, GS: Gleason score.

Table 2: Clinical data of patients with prostate cancer

Variables	N=100	%
▪ **Stage III**	26	26.0%
▪ **Intermediate risk**		
▪ **Favorable**	0	0.0
▪ **Unfavorable**	1	3.8%
▪ **High risk**	25	96.2%
▪ **Stage IV**	74	74.0%
A) Metastatic hormone sensitive prostate cancer	N=60	
▪ **Low volume**	21	35.0%
▪ **High volume**	39	65.0%
B) Metastatic castrate resistant prostate cancer	N=14	
▪ **Low volume**	3	21.4%
▪ **High volume**	11	78.6%

Table 3: Lines of treatment among studied patients with prostate cancer

Variables	Total (N=100)	Locally advanced (n=26)	metastatic (n=74)
(ADT)			
▪ **Surgical**	2 (2.0%)	1 (3.8%)	1 (1.4%)
▪ **Medical**	98 (98.0%)	25 (96.2%)	73 (98.6%)
Surgery			
▪ **Prostatectomy**	1 (1.0%)	1 (1.0%)	0 (0.0%)
Radiotherapy	68 (68.0%)	14 (53.8%)	54 (73.0%)
Types of radiotherapy			
▪ **Radical**	15 (15.0%)	14 (53.8%)	1 (1.4%)
▪ **Palliative on bone only**	51 (51.0%)	0 (0.0%)	51 (68.9%)
▪ **Both**	2 (2.0%)	0 (0.0%)	2 (2.7%)
Chemotherapy (Docetaxel)	9 (9.0%)	0 (0.0%)	9 (12.2%)
▪ **MHSPC**	1 (1.0%)	0 (0.0%)	1 (1.4%)
▪ **MCRPC**	8 (8.0%)	0 (0.0%)	8 (10.8%)
Novel hormonal agents (abiraterone)	7 (7.0%)	0 (0.0%)	7 (9.5%)
▪ **MCRPC**	7 (7.0%)	0 (0.0%)	7 (9.5%)

ADT: Androgen Deprivation Therapy, MHSPC: Metastatic Hormone-Sensitive Prostate Cancer, MCRPC: Metastatic Castration-Resistant Prostate Cancer

Table 4: Follow up trend among studied patients

Variables	N=100	%
First follow up (after 3 months)		
Follow up by		
▪ **PSA only**	31	31.0%
▪ **Image only**	5	5.0%
▪ **Both**	64	64.0%
Image	N=69	
▪ **PR**	60	87.0%
▪ **PD**	9	13.0%
Second follow up (after 6 months)	N=79	
Follow up by		
▪ **PSA only**	8	10.1%
▪ **Image only**	7	8.9%
▪ **Both**	64	81.0%
Image	N=71	
▪ **PR**	63	88.7%
▪ **PD**	8	11.3%
Third follow up (after 1 year)		
Follow up by	N=36	
▪ **PSA only**	7	19.4%
▪ **Image only**	1	2.8%
▪ **Both**	28	77.8%
Image	N=29	
▪ **PR**	26	89.7%
▪ **PD**	3	10.3%

PSA: prostate-specific antigen, PR: paetial response, PD: progressive disease

Table 5: PSA level pre-treatment and 1st post treatment and its correlation with OS and PFS

	Total (N=100)	Non metastatic (n=26)	metastatic (n=74)
Pre treatment PSA ng/mL			
Median ng/mL(range)	96.8 (3.80-600000.0)	38.30 (3.80-233.00)	100.0 (7.70-600000.0)
1st Post treatment PSA			
Median (range)	12.4 (0.02-7521)	6.10 (0.29-65)	14.70 (0.02-7521)
Difference (Pre treatment PSA- 1st post treatment PSA)			
Median (range)	72.50 (0.0-592479.0)	34.10 (3.0-168.0)	85.0 (0.0-592479.0)
P-Value*	<0.001	<0.001	<0.001
% of decline of PSA			
Median (range)	84.2% (0.0%-99.94%)	80.85% (34.57%-99.79%)	85.95% (0.0%-99.94%)
Correlation between % of decline of PSA with OS			
r-Value	0.221	0.124	0.242
P-Value	0.031	0.556	0.042
Correlation between % of decline of PSA with PFS			
r-Value	0.791		0.791
P-Value	<0.001		<0.001

r (Spearman correlation coefficient)

* Wilcoxon sign test was used to compare median differences of PSA pre and post treatment & was significantly different.

PSA: prostate-specific antigen, OS: overall survival, PFS: progression free survival

Table 6: Univariate and multivariate Cox regression analysis for prognostic factors associated with overall survival among studied patients

	OS			
	Univariate Cox regression HR (95% CI)	**P-Value***	**multivariate Cox regression HR (95% CI)**	**P-Value***
Age (years)				
▪ **≤65 years**	Reference	0.433	Reference	0.103
▪ **>65 years**	1.52 (0.53-4.36)		2.91 (0.81-10.53)	
Smoking				
▪ **No**	Reference	0.647		
▪ **Yes**	1.34 (0.37-4.85)			
Comorbidities				
▪ **No**	Reference	0.660		
▪ **Yes**	1.29 (0.41-4.07)			
Staging				
▪ **Stage III**	Reference	0.869		
▪ **Stage IV**	1.11 (0.31-4.00)			
Metastasis				
▪ **Locally advanced**	Reference	0.869		
▪ **Metastatic**	1.11 (0.31-4.00)			
Complaint				
▪ **Urological**	Reference	0.702		
▪ **Bone pain**	0.81 (0.27-2.37)			
Surgery				
▪ **No**	Reference	0.347		
▪ **Yes**	2.12 (0.44-10.21)			
Radiotherapy				
▪ **Yes**	Reference	0.039	Reference	0.034
▪ **No**	3.00 (1.05-8.54)		3.33 (1.09-10.18)	
Chemotherapy				
▪ **Yes**	Reference	0.892		
▪ **No**	0.901 (0.19-4.09)			
% of PSA decline	0.98 (0.96-0.99)	0.006	0.97 (0.95-0.99)	0.003

HR: hazardous ratio, CI: confidence interval, Cox regression analysis

Table 7: Univariate and multivariate Cox regression analysis for prognostic factors associated with PFS among studied patients

	PFS			
	Univariate cox regression HR (95% CI)	**P-Value***	**multivariate cox regression HR (95% CI)**	**P-Value***
Age (years)				
▪ **≤65 years**	Reference	0.220	Reference	0.335
▪ **>65 years**	3.51 (0.36-33.73)		3.13 (0.31-31.91)	
Smoking				
▪ **No**	Reference	0.706		
▪ **Yes**	1.55 (0.15-15.13)			
Comorbidities				
▪ **No**	Reference	0.993		
▪ **Yes**	1.09 (0.11-10.74)			
Complaint				
▪ **Urological**	Reference	0.416		
▪ **Bone pain**	0.42 (0.04-4.11)			
Radiotherapy				
▪ **Yes**	Reference	0.037	Reference	0.098
▪ **No**	12.8 (1.2-140.6)		4.58 (0.8-90.6)	
Chemotherapy				
▪ **Yes**	Reference	0.964		
▪ **No**	1.04 (0.14-7.46)			
% of PSA decline	0.96 (0.92-0.99)	0.049	0.96 (0.91-0.99)	0.049

HR: hazardous ratio, CI: confidence interval, Cox regression analysis
PFS: progression free survival, PSA: prostate-specific antigen

Discussion:

Our study aimed for evaluation of clinical and pathological characteristics and routine treatment of patients with prostate cancer with insights into staging, risk classification, and metastatic patterns. It shows that 26.0% of the studied patients were stage III, while 74% were stage IV, with 68% being de novo cases and 6% developing during treatment.

For locally advanced disease, 96.2% of cases were classified as high risk, and 3.8% as intermediate risk with unfavorable features. Among metastatic cases, 81.1% were metastatic hormone-sensitive prostate cancer (mHSPC), with 35% being low volume and 65% high volume, while 18.9% were metastatic castrate-resistant prostate cancer (mCRPC), with 21.4% being low volume and 78.6% high volume.

Ng et al investigated the characteristics and outcomes of patients with metastatic prostate cancer, focusing on hormone-sensitive and castrate-resistant disease. The study found that approximately 80% of metastatic prostate cancer cases were hormone-sensitive at diagnosis, with a significant proportion presenting with high-volume disease.

This is similar to our findings, where 81.1% of metastatic cases were mHSPC, and 65% of these were high volume. The study also highlighted that high-volume mHSPC is associated with poorer prognosis and requires aggressive treatment strategies; supporting our observation that high-volume disease is predominant in metastatic cases. Additionally, the study noted that a smaller proportion of patients progress to mCRPC, consistent with our finding of 18.9% mCRPC cases [9].

Van Poppel, reported that over 90% of patients with locally advanced disease were classified as high risk, which is consistent with our finding of 96.2% high-risk cases. The study also found that high-risk locally advanced prostate cancer is associated with a higher likelihood of progression to metastatic disease, underscoring the importance of aggressive treatment in this subgroup. In metastatic cases, the study observed that the majority of patients presented with high-volume mHSPC, similar to our findings of 65% high-volume mHSPC [10].

Additionally, Wenzel et al, noted that a smaller proportion of patients progress to mCRPC, consistent with our finding of 18.9% mCRPC cases. The study emphasizes the importance of early intervention in high-volume mHSPC to delay progression to castrate resistance, which is relevant to our findings on the distribution of metastatic cases [11].

Parker et al, who explored the use of RT in prostate cancer, particularly in metastatic and non-metastatic settings. The study found that palliative radiotherapy on bone was the most common form of RT in metastatic patients, consistent with our finding of 68.9% of metastatic patients. The study emphasized the role of palliative RT in managing symptoms and improving quality of life in metastatic patients, which supports our

findings on the predominance of palliative RT in this subgroup. It also highlighted that radical RT is more frequently used in non-metastatic patients, aligning with our observation that 53.8% of non-metastatic patients received radical RT [12].

Pilon et al investigated the use of chemotherapy and novel hormonal agents in metastatic prostate cancer, particularly in mCRPC. The study found that chemotherapy was primarily used in mCRPC patients, consistent with our finding that 8.0% of chemotherapy recipients had mCRPC , also noted that novel hormonal agents, such as abiraterone and enzalutamide, were predominantly used in mCRPC patients, aligning with our observation highlighting the efficacy of these agents in improving survival and delaying disease progression in mCRPC [13].

Regarding trends in follow-up patients by different methods in different interval, in the first follow-up we had 100 cases, more than 2/3 (64.0%) were monitored using both PSA and imaging, while 1/3 (31.0%) used PSA only respectively and 5% used image only. Among those followed by imaging (69 cases), 87% showed partial response (PR) and 13% had progressive disease (PD) which clarifies urge to track response of treatment & others satisfied only by PSA.

In the second follow-up (79 patients), 81.0% used both PSA and imaging, 10.1% used PSA only, and 8.9% used imaging only. Among imaging-followed cases (71 cases), 88.7% had PR and 11.3% had PD.

In the third follow-up (36 patients), 77.8% used both PSA and imaging, 19.4% used PSA only, and 2.8% used imaging only. Among imaging-followed cases (29 cases), 89.7% had PR and 10.3% had PD.

This was aligned by Scher et al. who emphasized the importance of combining PSA and imaging for accurate disease monitoring. The study found that dual monitoring (PSA and imaging) was associated with better detection of disease progression. Imaging alone was less frequently used, consistent with ours that a small proportion of patients relied solely on imaging [14].

Regarding response to treatment, Fernandes et al, evaluated the role of imaging in prostate cancer follow-up, particularly in assessing treatment response. He found that imaging was crucial for identifying PR and PD, especially when combined with PSA levels [15].

our findings on overall survival (OS) and progression-free survival (PFS) in prostate cancer patients was a median OS time of 59.0 months (95% CI: 37.11–80.88) & PFS of 51.0 months (95% CI: 27.61–50.12) respectively which was similar to what is reported by Sweeney et al., 2015 that a median OS of approximately 60 months in patients with advanced prostate cancer emphasizing that early intervention with systemic therapies, such as ADT combined with chemotherapy, significantly improved OS and PFS [16].

Regarding PFS and OS in prostate cancer patients treated with novel hormonal agents, James et al., 2017 reported a median PFS of around 50 months, consistent with our finding. This aligns with our data and suggests that incorporating advanced therapies could further enhance survival outcomes in prostate cancer patients [17].

The median pretreatment PSA level was 96.8 ng/mL overall. After treatment, the median PSA level decreased significantly to 12.4 ng/mL overall. The median percentage decline in PSA from pretreatment to first follow-up was 84.2% overall which was positively correlated with PFS (with P-Value <0.001) & OS (with P-Value 0.031).

A study by Chowdhury et al., examined the relationship between PSA decline and survival outcomes in prostate cancer patients, found that a greater decline in PSA levels post-treatment was associated with improved OS and PFS, highlighted that PSA decline is a reliable surrogate marker for treatment efficacy and survival, supporting our observation that a significant reduction in PSA levels correlates with better outcomes [19].

This was also reported by Armstrong et al., who reported that a significant decline in PSA levels post-treatment was strongly associated with longer PFS. This reinforces the importance of monitoring PSA kinetics as a predictor of survival in prostate cancer patients (19).

Our findings show that OS in prostate cancer patients was negatively correlated with factors such as age, smoking status, comorbidities, staging, metastasis type (mHSPC or mCRPC), presenting symptoms (bone pain or urological complaints), or treatment modalities like surgery or chemotherapy except RT which showed positive correlation with patients receiving RT showing a higher median OS of 72.0 months compared to 59.0 months in those who did not [20].

Parker et al, also emphasized that RT, especially when combined with systemic therapies, can enhance survival by controlling local disease progression and reducing symptomatic burden. This supports our observation that RT is a key factor influencing OS in prostate cancer patients [7].

Our multivariate Cox regression analysis, we identified two significant predictors of mortality in prostate cancer patients: RT and the percentage decline in PSA. Patients who did not receive RT had a higher hazard ratio (HR=3.33, 95% CI=1.09–10.18, p=0.034), indicating a significantly increased risk of mortality compared to those who received RT.

Additionally, a higher percentage decline in PSA was associated with a lower hazard ratio (HR=0.97, 95% CI=0.95–0.99, p=0.003), suggesting a reduced risk of mortality. These findings underscore the protective effect of radiotherapy, and the prognostic value of PSA decline in prostate cancer outcomes which was found also in [19].

Our findings indicated that age, smoking status, comorbidities, presenting symptoms, treatment modalities like surgery or chemotherapy did not affects PFS in prostate cancer patients. This suggests that these variables do not play a major role in determining the time to disease progression in this patient population.

Our results was similar to data presented by Ryan et al., 2013 who highlighted that disease-related factors, such as tumor biology and treatment response, were more critical determinants of PFS than patient-specific

characteristics. This aligns with our observation that the tested variables did not significantly affect PFS [21].

While in Beer et al., 2014 who investigated the impact of treatment modalities and patient characteristics on PFS in metastatic prostate cancer, reported that chemotherapy and surgery did not significantly influence PFS, which is consistent with our findings. The study emphasized that treatment response, particularly to systemic therapies like ADT and novel hormonal agents, was a stronger predictor of PFS than patient-specific factors. This supports our observation that the tested variables, including surgery and chemotherapy, did not significantly affect PFS [22].

Our findings on outcomes among prostate cancer patients indicate that 16.0% of the studied patients had died, 49.0% were alive, and 35.0% were censored. This provides a snapshot of the survival distribution and highlights the importance of further analysis to understand factors influencing mortality and survival in this population.

Similar to our data the study done by Sweeney et al., 2015 that examined survival outcomes in prostate cancer patients, particularly focusing on mortality and censoring rates. The study reported similar proportions of deaths and censored cases, with a significant percentage of patients remaining alive during follow-up [16].

Another study by James et al., 2017 also noted that censoring rates were influenced by factors such as treatment response and disease progression, which aligns with our observation of 35.0% censored cases. The study underscored the importance of analyzing censored avoid bias in survival analysis, supporting the relevance of our findings [17].

However, there are limitations to this study. Firstly, it was retrospective in nature which may have missing or incomplete data, leading to potential biases or inaccuracies in the analysis. Secondly, the study may be subject to selection bias, as it only includes patients from two specific centers, which may not be representative of the broader population. Finally, a significant proportion of patients (35%) were censored, which may introduce bias and limit the ability to draw definitive conclusions about long-term survival.

Conclusion:

The results of the current study underscore the importance of RT. It is a key factor influencing OS in prostate cancer patients. The study also highlighted that a significant decline in PSA levels post-treatment was strongly associated with longer survival.

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