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Evaluation of the Positive Response Rate to First-Generation Hormone Therapy (FGHT) in Patients with Prostate Cancer (PCa) in Kinshasa, from January 2014 to January 2025

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Abstract

Introduction: Prostate cancer (PCa) is a hormone-dependent malignancy and a major public health concern. In resource-limited settings such as the Democratic Republic of the Congo, it is frequently diagnosed at an advanced stage, making hormone therapy the cornerstone of management. **Objective:** To evaluate the rate of positive response to FGHT and to determine the time to castration resistance (CR) and associated factors. **Methods:** We conducted a retrospective observational cohort study including 258 patients with PCa treated at the Pointe-à-Pitre Clinic (CPAP) in Kinshasa between January 2014 and January 2025. **Results:** The mean age was 70.7 years (range: 53 - 94). Most patients presented with advanced, high-risk or metastatic disease (94.6%). A positive response to FGHT was observed in 82.1% of patients. An initial PSA ≥100 ng/mL, the elevated SGOT levels (≥38 U/L), the presence of metastases at diagnosis, the high ISUP grade (3 - 5), advanced tumor stage (cT3 - cT4), and high or very high risk classification according to D'Amico/NCCN/STAMPEDE were several factors significantly associated with the development of CR. The median time to CR, estimated by Kapan-Meier analysis, was 18 months. The median overall survival was 28 months. The overall mortality rate was 15.8%. **Conclusion:** In our setting, PCa predominantly affects men aged 70 - 79 years and is most often diagnosed at an advanced stage. FGHT shows sub-

stantial efficacy, with a high rate of positive response. CR develops progressively, allowing many patients to remain hormone-sensitive after five years. FGHT therefore remains a valuable therapeutic option in resource-limited settings.

Keywords

Prostate Cancer, First-Generation Hormone Therapy, Castration Resistance, Democratic Republic of the Congo

1. Introduction

Prostate cancer (PCa) is one of the most common malignancies affecting men and remains a leading cause of cancer-related mortality worldwide. In 2020, it accounted for approximately 25% of all male cancers, ranking second after lung cancer [1]. In the Democratic Republic of the Congo (DRC), PCa also occupies the second position (14%), just behind Kaposi's sarcoma (14.5%) [2].

The management of PCa is based on a multimodal approach that combines local and systemic therapies, tailored according to tumor stage, prostate-specific antigen (PSA) level, ISUP grade, patient age, life expectancy, and comorbidities. Its hormone-sensitive nature, first demonstrated by Huggins in 1949, underscores the pivotal role of androgens (testosterone and dihydrotestosterone) in tumor progression. Suppressing their production or blocking their action effectively slows disease progression.

First-generation hormone therapy (FGHT), whether used alone or in combination with anti-androgens, remains the standard treatment for advanced and metastatic disease. Its efficacy can be enhanced by the addition of next-generation hormone therapy (NGHT) or chemotherapy [3].

Approximately 80% to 90% of patients initially respond favorably to FGHT, with improvements in pain control, a PSA reduction of ≥50%, radiologic stability or regression, and an overall improvement in quality of life [4] [5]. However, this response is typically transient, lasting on average between 12 to 18 months before the onset of castration resistance (CR) [6].

In our context, limited access to healthcare and low socioeconomic conditions often result in delayed diagnosis, frequently at the advanced or metastatic stage. Consequently, FGHT remains the most commonly used therapeutic approach. This study aims to evaluate the rate of favorable response to FGHT, as well as the timeframe and factors associated with the development of CR in our context.

2. Materials and Methods

2.1. Study Design and Period

This retrospective observational study was conducted on a cohort of 258 patients diagnosed with prostate cancer (PCa) and managed at the Pointe-à-Pitre Clinic

(CPAP) in Matete/Kinshasa, over an 11 years' period from January 2014 to January 2025.

2.2. Population and Cohort

A total of 271 patient's records were initially screened. Among these: 13 records were excluded due to completely incomplete data and were not included in the main analysis; 7 records had missing data for certain variables, but these patients were included in the analysis using a complete-case analysis approach.

We included patients diagnosed with PCa who were treated with FGHT and had undergone at least one PSA check at a three-moth interval. Patients who did not meet these criteria were excludes.

Thus, the exact denominator for the FGHT analyses was 251 patients. The baseline characteristics of the cohort are summarized in (Table 1).

Table 1. Baseline characteristics of the cohort.

		Frequency	Percentage	Valid Percentage	Cumulative Percentage
Valid	Positive response	206	79.8	82.1	82.1
	CR	45	17.4	17.9	100.0
	Total	251	97.3	100.0	
Missing	System	7	2.7		
Total		258	100.0		

2.3. Follow-Up and Survival Status

Survival status was assessed as the time from admission to the date of last follow-up for patients lost to follow-up, to the date of death for deceased patients, and to the end of the study for patients who remained alive. Patients lost to follow-up were censored as alive.

2.4. Setting, and Parameters of Interest

The analyzed parameters included patient age, presenting signs and symptoms at initial consultation, digital rectal examination findings, clinical stage (cTNM), total PSA level, histological data (ISUP grade group), metastatic workup, risk stratification according to the D'Amico, NCCN, and STAMPEDE (EAU 2025) classifications, treatment modalities, response to FGHT, frequency and time to CR, overall survival, and predictors of mortality.

Androgen deprivation was achieved either surgically (orchidectomy) or medically through FGHT, with or without anti-androgens. In cases of CR, therapeutic intensification was implemented using next-generation hormone therapy (NGHT) and/or chemotherapy.

Treatment modalities included:

- **FGHT:** Goserelin 10.8 mg subcutaneous injection every 3 months ± bicalu-

tamide 50 mg/day or cyproterone acetate 100 mg three times daily.

- **NGHT:** Abiraterone acetate 500 mg (two tablets daily) taken on an empty stomach, combined with prednisone 5 mg/day.
- **Chemotherapy:** Docetaxel 75 mg/m^2 administered by intravenous infusion every 3 weeks.
- **For bone metastatic castration-resistant PCa (mCRPC):** Zoledronic acid 4 mg intravenously every 4 weeks.

2.5. Definition of Study Endpoints

A favorable response to hormone therapy was defined as a testosterone level below 0,5 ng/ml and a reduction of ≥50% in serum from baseline PSA, confirmed after treatment initiation. This response may be accompanied by linical improvement and/or regression of radiological lesions.

The clinical improvement was primarily defined as reduction in bone pain, while radiological response was assessed by regression or stabilization of lesions on follow-up imaging.

The clinical and biochemical evaluations (PSA and testosterone) were performed at 3, 6, 9 and 12 months, then every 6 months up to 3 years, and finally once a year during the 4th and 5th years. Radiological assessments were conducted if possible every 2 years.

The castration resistance (CR) was defined as a PSA that remained unchanged after at least 3 months of FGHT, despite a reduction in testosterone potentially reaching levels below 0.5 ng/mL, or as biochemical progression under effective castration (testosterone < 0.5 ng/mL). Biochemical progression was considered when PSA increased by at least 50% above the nadir.

2.6. Data Collection

Data was collected from medical records and institutional registries using a standardized data collection form.

2.7. Statistical Analysis

Data were entered into Microsoft Excel 2016, cleaned, and analyzed using SPSS version 25. Quantitative variables with normal distribution were expressed as means ± standard deviation (SD), non-parametric data as medians with interquartile range (IQR), and qualitative variables as proportions (%). Normality was assessed using the Kolmogorov-Smirnov or Shapiro-Wilk tests.

Comparisons of means were performed using Student's *t*-test, medians using the Mann-Whitney *U* test, and proportions using Pearson's Chi-square or Fisher's exact test as appropriate. Survival and time to CR were analyzed using Kaplan-Meier curves, with comparisons by the Log-rank test. Cox proportional hazards regression was used to identify risk factors for CR and predictors of mortality using the stepwise method; only the variables that were significant in the univariate analysis were included in the multivariate analysis. Results were expressed as adjusted Relative Risk (aRR) for incidence of CR and adjusted Hazard Ratio (aHR)

for mortality with 95% confidence intervals (CI). A p-value < 0.05 was considered statistically significant.

2.8. Ethical Considerations

The study was conducted in accordance with ethical principles, ensuring participant anonymity, data confidentiality, and full respect for medical secrecy.

3. Results

3.1. Sociodemographic Characteristics of Patients with Prostate Cancer

The most frequent age group was 70 - 79 years (50%), with a mean age of 70.7 years (range: 53 - 94) (Table 2).

Table 2. Sociodemographic characteristics of patients with PCa (n = 258).

Variables	Frequency (n = 258)	Percentage (%)
Age		
<60 years	23	8.9
60 - 69 years	81	31.4
70 - 79 years	129	50.0
≥80 years	25	9.7

3.2. Reasons for Consultation and Digital Rectal Examination Findings

Dysuria was the most common presenting symptom (64.7%). On digital rectal examination (DRE), an enlarged nodular prostate was observed in 66.7% of patients (Table 3).

Table 3. Consultation's reason and digital rectal examination findings (n = 258).

Reasons for Consultation	Frequency (n = 258)	Percentage (%)
Obstructive symptoms		
Dysuria	167	64.7
Chronic urinary retention (CUR)	53	20.5
Acute urinary retention (AUR)	69	26.7
Irritative symptoms		
Pollakiuria	113	43.8
Nocturia	122	47.3
Painful micturition	89	34.5
Urgency	86	33.3
Digital rectal examination		
Enlarged nodular prostate	172	66.7
Enlarged non-nodular prostate	76	29.5
Normal prostate	10	3.5

3.3. Paraclinical Findings

An initial total PSA level > 100 ng/mL was observed in 50.4% of cases, and PSA density > 0.15 ng/mL in 89.5%. Metastases were detected in 38.8% of patients, predominantly in the bones (22.9%) (Table 4).

Table 4. Paraclinical assessment.

Variables	N (%)	Mean ± SD	Median (IQR)
Testosterone (ng/mL)	-	-	8.0 (5.0 - 11.0)
Testosterone ≥ 10 ng/mL	88 (34.1)	-	-
PSA (ng/mL)	-	-	100 (31.0 - 100.0)
PSA ≥ 100 ng/mL	130 (50.4)	-	-
PSA density (ng/mL/cm^3)	-	-	0.79 (0.39 - 1.70)
PSA density > 0.15	231 (89.5)	-	-
SGOT (U/L)	-	-	23.0 (16.0 - 31.0)
SGOT ≥ 38 U/L	38 (14.7)	-	-
SGPT (U/L)	-	-	26.0 (17.0 - 33.9)
SGPT ≥ 40 U/L	38 (14.7)	-	-
Metastases on CT scan	n (100)	-	-
M0	33 (12.8)	-	-
M1a	18 (7.0)	-	-
M1b	59 (22.9)	-	-
M1c	23 (8.9)	-	-
Mx	124 (48.2)	-	-

3.4. Evolution of Testosterone Levels under First-Generation Hormone Therapy

This figure illustrates a rapid decline in serum testosterone levels following initiation of hormone therapy, which remained stable up to 36 months, followed by a slight subsequent increase (Figure 1).

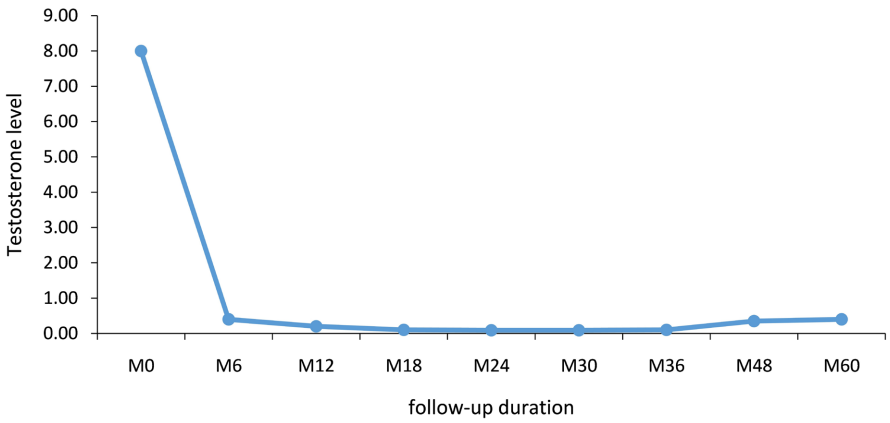

Figure 1. Evolution of serum testosterone.

3.5. Evolution of PSA Levels under First-Generation Hormone Therapy

This figure shows a sustained therapeutic response, characterized by a rapid and marked decline in PSA levels and effective disease control for up to 60 months (Figure 2).

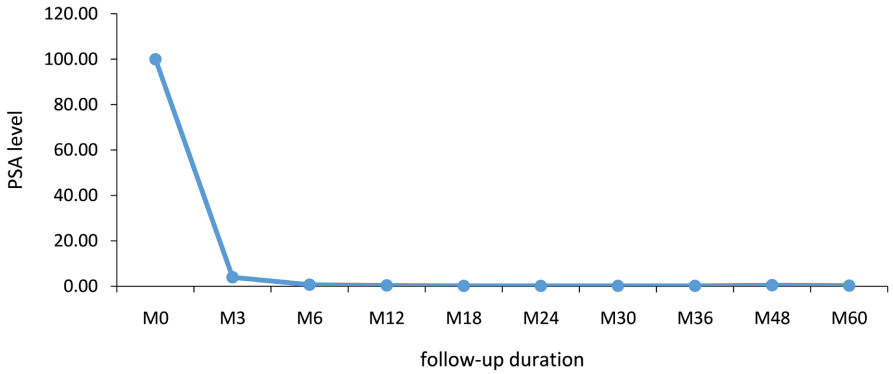

Figure 2. Evolution of PSA under first-generation hormone therapy.

3.6. Clinical Stage at Presentation and Diagnosis

Advanced stages (cT3 - cT4) were predominant (62.4%), and high ISUP grades (4 - 5) were observed in 50.8% of cases. Most patients (38.9%) presented with metastatic disease, primarily bone metastases. High-risk, locally advanced, and metastatic forms accounted for 94.6% of all cases (Table 5).

Table 5. Clinical stage and diagnostic findings (n = 258).

Variable	Frequency (n = 258)	Percentage (%)
ISUP Grade		
1	12	4.7
2	25	9.7
3	90	34.9
4	66	25.6
5	65	25.2
TNM Stage		
cT1 - cT2	97	37.6
cT3 - cT4	161	62.4
D'AMICO/NCCN/STAMPEDE	**157**	**61.1**
Low risk	2	0.8
Favorable intermediate	6	23
Unfavorable intermediate	6	2.3
High risk	67	26.1
Very high risk (locally advanced)	76	29.6
METASTATIC	**100**	**38.9**

Continued

Metastatic site		
M1a	18	18.0
M1b	59	59.0
M1c	23	23.0

3.7. Treatment Administered

LHRH analogues were the primary therapeutic agents used in 82.6% of patients, most often in combination with bicalutamide (72.9%). Orchidectomy was performed in 19% of cases. Abiraterone was used upon development of resistance to FGHT (58.9%). Transurethral resection of the prostate (TURP) was performed in 50% of patients with significant tumor volume, whereas prostatectomy was rarely performed (2.7%) (Table 6).

Table 6. Treatments administered (n = 258).

Variable	Frequency (n = 258)	Percentage (%)
LHRH analogue (goserelin. triptorelin) (1)	213	82.6
Goserelin + Bicalutamide	188	72.9
Goserelin + cyproterone acetate	53	20.5
Zoledronic acid	8	3.1
Second-line hormone therapy (abiraterone acetate)	152	58.9
Chemotherapy (docetaxel)	11	4.3
Orchidectomy	49	19.0
Prostatectomy	7	2.7
TURP or debulking surgery	129	50.0
Cervico-prostatic incision (CPI)	13	5.0
Transurethral resection of the bladder (TURB)	3	1.2
External radiotherapy	7	2.7

3.8. Rate of Positive Response

A total of 82.1% of patients receiving first-generation hormone therapy (FGHT) achieved a positive clinical response (Figure 3).

3.9. Overall Resistance Rate

This curve illustrates the cumulative risk of developing castration resistance (CR) among patients with prostate cancer (PCa). The median time to CR onset was 18.0 months (range: 6.0 - 30.0) (Figure 4).

Arithmetic mean	21.0504
Median	18.0000

The cumulative incidence of CR was 8.5% at 1 month, 11.6% at 6 months, 14.0% at 12 months, 14.7% at 18 months, 15.5% at 28 months, 16.3% at 36 months, 17.4% at 60 months, and 17.4% at 122 months (Figure 4).

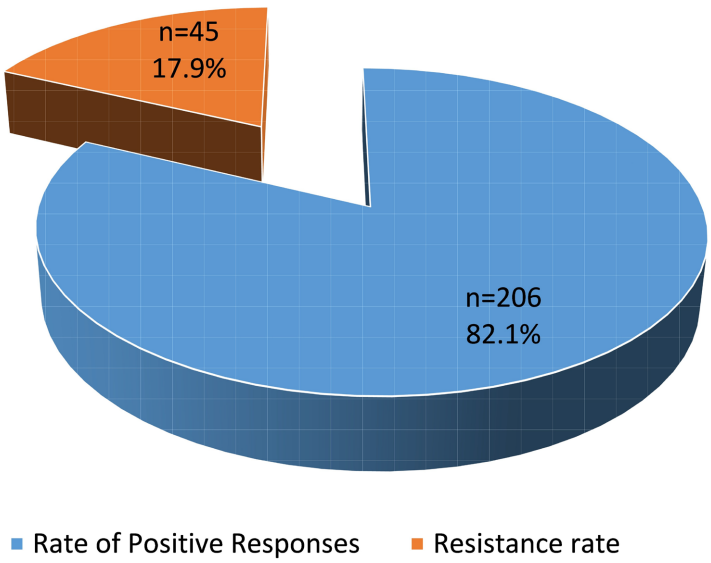

Figure 3. Rate of positive responses.

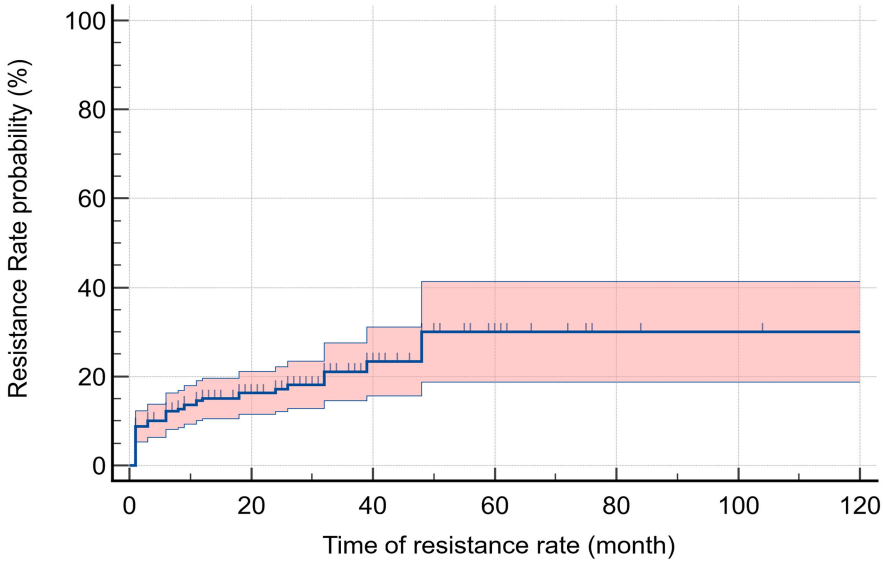

Figure 4. Overall resistance rate.

3.10. Risk Factors Associated with Hormone Resistance

Bivariate and multivariate analyses identified several factors significantly associated with the development of CR during follow-up.

An initial PSA ≥ 100 ng/mL substantially increased the risk of resistance, with an incidence of 20.5 cases per 100 person-months, compared to 7.2 cases per 100 person-months among those with PSA < 100 ng/mL. This association remained significant after adjustment (aRR = 1.76; $p = 0.019$).

Similarly, elevated SGOT levels (≥38 U/L) were also linked to a higher incidence of resistance (16.6/100 person-months) and remained an independent factor after adjustment (aRR = 1.82; $p = 0.024$).

The presence of metastases at diagnosis strongly increased the risk, showing a ninefold higher incidence in the unadjusted analysis, and remained significant in multivariate analysis (aRR = 2.83; $p = 0.014$).

Additionally, high ISUP grade (3 - 5), advanced tumor stage (cT3 - cT4), and high or very high risk classification according to D'Amico/NCCN/STAMPEDE were also independently associated with an increased risk, with adjusted relative risks of 3.28, 2.31, and up to 4.89, respectively ($p < 0.05$) (Table 7).

Table 7. Risk factors associated with hormone resistance.

Variable				Univariate analysis	Multivariate analysis	
	N	Duration	Resistance/100 P-M (95% CI)	RR (IC 95%)	RRa (IC95%)	*p*
Overall	258	5431	6.1 (4.7 - 9.7)	-	-	-
PSA (ng/mL)						
<100 ng/mL	128	3366	7.2 (5.6 - 11.1)	1	1	
≥100 ng/mL	130	2065	20.5 (18.7 - 25.4)	2.45 (1.56 - 3.85)	1.76 (1.10 - 2.82)	**0.019**
SGOT (U/L)						
<38 UI/L	153	4828	9.1 (7.9 - 13.4)	1	1	
≥38 UI/L	19	603	16.6 (14.2 - 20.8)	1.97 (1.18 - 3.29)	1.82 (1.08 - 3.05)	**0.024**
Metastases						
No	33	1032	0.9 (0.5 - 1.8)	1	1	
Yes	225	4399	8.4 (6.3 - 11.7)	5.55 (1.75 - 7.61)	2.83 (1.84 - 5.4	**0.014**
ISUP grade						
1 - 2	37	1051	1.3 (1.1 - 1.9)	1	1	
3 - 5	221	4380	8.4 (6.7 - 10.8)	3.36 (1.36 - 8.30)	3.28 (1.48 - 5.42)	**0.021**
TNM stage						
cT1 - cT2	97	2725	7.2 (5.3 - 10.2)	1	1	
cT3 - cT4	161	2706	15.4 (11.8 - 19.5)	3.00 (1.79 - 5.03)	2.31 (1.73 - 5.63)	**0.013**
D'AMICO/NCCN /STAMPEDE						
Low & favorable intermediate	7	351	0.0 (0.0 - 0.3)	1	1	
Unfavorable intermediate	7	174	0.8 (0.3 - 1.3)	1.20 (0.31 - 1.34)	1.18 (0.56 - 1.35)	0.912

Continued

High risk	70	1774	12.1 (9.2 - 13.9)	3.28 (2.39 - 5.18)	3.19 (2.11 - 6.71)	**<0.001**
Very High risk	174	3132	12.8 (10.2 - 14.6)	3.42 (2.24 - 7.30)	4.89 (2.45 - 7.53)	**<0.001**

3.11. Overall Survival Rate

As shown in the figure above, at the end of follow-up, 52.1% of patients were lost to follow-up, 32.0% were alive, and 15.8% had died (Figure 5).

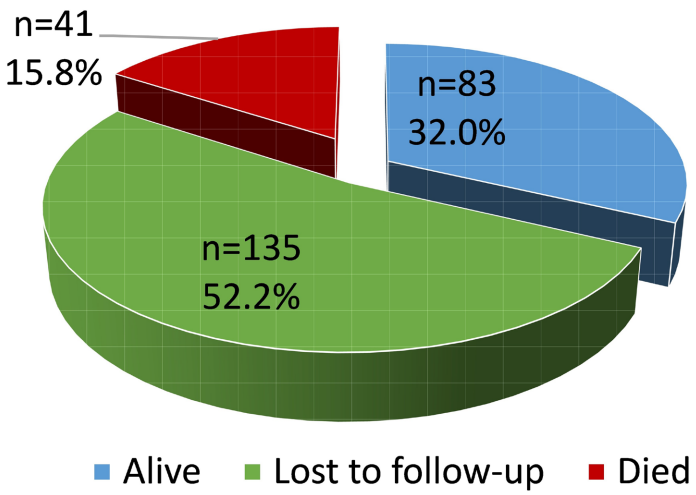

Figure 5. Overall survival rate at the end of follow-up.

3.12. Overall Survival Over Time

This figure illustrates the overall survival (OS) of patients included in the study. The median survival time was 28.0 months (IQR: 13.0 - 60.5) (Figure 6).

Arithmetic mean	44.9685
Median	28.0000

OS rates were 97.3% at 3 months, 95.7% at 9 months, 93.4% at 1 year, 91.1% at 18 months, 89.1% at 2 years, 87.2% at 3 years, 84.5% at 5 years, and 84.1% at the end of follow-up (Figure 6).

3.13. Predictive Factors of Mortality

Univariate analysis identified several factors significantly associated with an increased risk of mortality.

Hormone-resistant cancer (HR = 4.46; $p < 0.001$), PSA ≥ 100 ng/mL (HR = 3.10; $p = 0.002$), elevated SGOT (≥38 U/L; HR = 2.28; $p = 0.017$), presence of metastases (HR = 6.25; $p = 0.003$), high ISUP grade (3 - 5; HR = 3.21; $p = 0.011$), and advanced TNM stage (cT3 - cT4; HR = 4.90; $p = 0.001$) were all significant mortality predictors.

After multivariate adjustment, five variables remained independently associated with mortality: Hormone-resistant cancer (HR = 2.79; $p = 0.013$), PSA ≥ 100 ng/mL (HR = 2.70; $p = 0.018$), Metastases (HR = 3.20; $p = 0.005$), ISUP grade 3 - 5 (HR = 3.60; $p = 0.015$), TNM stage cT3 - cT4 (HR = 2.83; $p = 0.006$). These findings highlight the prognostic importance of both clinical and biological parameters in cancer-related mortality (Table 8).

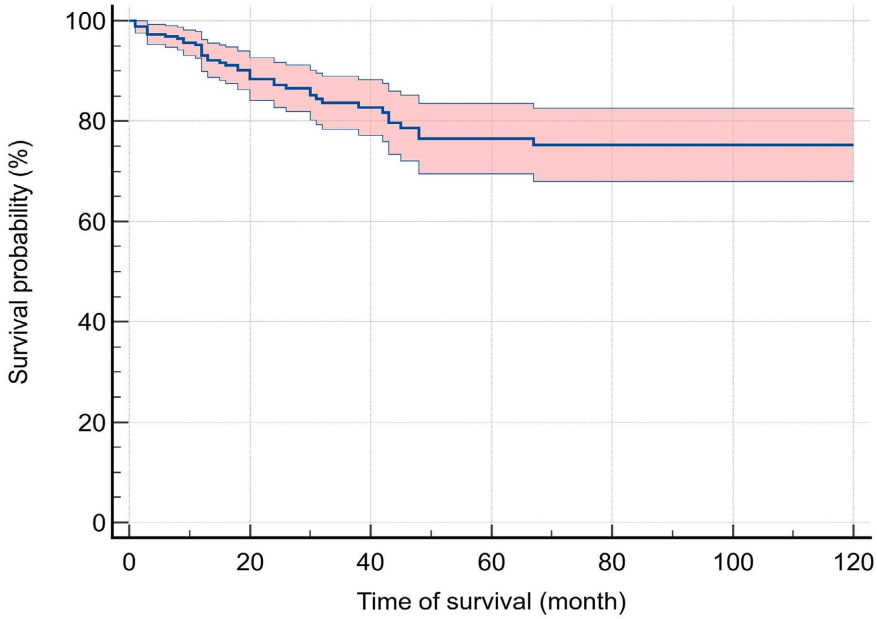

Figure 6. Overall survival of study patients over time.

Table 8. Predictive factors of mortality.

Variable	Univariate Analysis		Multivariate Analysis	
	p	HR (IC 95%)	P	HR (IC 95%)
Hormone-resistant cancer				
Yes		1		1
No	**<0.001**	4.46 (2.09 - 9.50)	**0.013**	2.79 (1.24 - 6.26)
PSA (ng/mL)				
<100 ng/mL		1		1
≥100 ng/mL	**0.002**	3.10 (1.52 - 6.32)	**0.018**	2.70 (1.78 - 3.70)
Metastases				
Yes		1		1
No	**0.003**	6.25 (3.86 - 10.49)	**0.005**	3.20 (2.26 - 8.57)
ISUP grade				
1 - 2		1		1
3 - 5	**0.011**	3.21 (1.77 - 5.36)	**0.015**	3.60 (2.31 - 8.29)
TNM stage				
cT1 - cT2		1		1
cT3 - cT4	**0.001**	4.90 (1.92 - 6.50)	**0.006**	2.83 (1.97 - 8.25)

4. Discussion

This study is the first in our context to evaluate the positive response rate to first-generation hormone therapy (FGHT1) in 258 patients with prostate cancer (PCa), as well as the factors and timing associated with castration resistance (CR). Diagnosis predominantly of PCa occurred in patients in their fifties, with the highest frequency in the 70 - 79 age group (50%), a mean age of 70.7 years, and extremes ranging from 53 to 94 years. These results align with several studies reporting a mean age between the sixth and seventh decades of life [7]-[12]. In our context, Moningo observed a similar predominance in the 70 - 79 age group (39.4%), with a mean age of 69.4 years [7], whereas Ngandu TJ in Mbuji-Mayi reported a younger mean age of 59.1 years, ranging from 57 to 60 years [8] (**Table 9**).

Table 9. Mean age of CaP according to authors.

Authors	Country	Mean Age	Year
D. M. Moningo *et al.* [7]	DRC/Kinshasa	69.4 (40 - 90)	2020
Ngandu TJ *et al.* [8]	DRC/Mbuji Mayi	59.1 (57 - 60)	2015
Moreira *et al.* [9]	USA	70.0 (67 - 81)	2016
Rozet *et al.* [10]	France	70.0	2016
Laurent Brureau *et al.* [11]	Antilles	68.0 (53 - 82)	2009
D. Moningo *et al.* [12]	DRC/Kinshasa	68.9 (43 - 88)	2018
Notre étude	DRC/Kinshasa	70.7 (53 - 90)	2025

Dysuria (64.7%), followed by nocturia (47.3%) and pollakiuria (43.8%), were the most frequent reasons for consultation. These findings are consistent with the literature but show some variation. Moningo *et al.* reported obstructive symptoms in 72.5% of cases and irritative symptoms in 33.5% [7]. In Algeria, Belabed Zaket A. observed a high frequency of pollakiuria (45%) but a lower rate of dysuria (31%) [13]. While in Kinshasa, Punga reported nocturia as the dominant symptom in 52.4% of cases [14].

Regarding tumor characteristics, stages cT3 - cT4 (62.4%) and ISUP grades 4 - 5 (50.8%) was the most frequent. Metastatic disease was present in 38.9% of patients, predominantly affecting the bone, followed by locally advanced forms (29.6%) and high-risk forms (26.1%). Low- or intermediate-risk tumors were less common with high-risk, locally advanced, and metastatic prostate cancers collectively accounted for 94.6% of cases. These results are in line with Moningo *et al.*, who reported a predominance of metastatic CaP (47.1%) and high-risk CaP (25.5%) [7]. Other authors report similar findings [9] [11] [13]. The high frequency of bone metastases is consistent with the natural osteotropism of prostate cancer.

Therapeutically, LHRH analogues (82.6%), most often combined with bicalutamide (72.9%), were the main treatment modality. Orchidectomy was performed in 19% of patients. While abiraterone (58.9%) was reserved for patient's resistant to FGHT. TURP was performed in 50% of patients for significant prostatic ob-

struction, while radical prostatectomy (RP) was rare (2.7%). These observations align with Moningo *et al.*, who reported high use of LHRH analogues (95%) and orchidectomy in 23% of cases [7]. Overall, androgen deprivation therapy, whether medical or surgical, remains the cornerstone of treatment in our context, reflecting the often advanced stage at diagnosis [15] [16].

Following initiation of first-generation hormone therapy (FGHT), 82.1% of patients in our cohort demonstrated a positive response, defined as a ≥50% reduction in PSA, often accompanied by clinical or radiological improvement. These results are consistent with the literature. For instance, Seront E., at the Saint-Luc University Clinics in Brussels, reported a response rate of 80% - 90% for ADT, with improvement in pain, PSA reduction, radiological stabilization, and better quality of life [4]. Similarly, Namiki M. in Japan emphasized that hormone therapy remains the treatment of choice for advanced PCa, achieving efficacy exceeding 90% [16]. In the United States, Raghavan D. documented an initial response rate of 60% - 70% for surgical or medical castration in hormone-sensitive cases [17].

A PSA reduction of 50% or more (PSA50) after starting HTG1 is a reliable biological indicator of favorable response [18] [19]. Clinical trials such as SWOG 9346, CHAARTED, and LATITUDE demonstrated a significant association between this response and better overall survival [20]-[22]. Although the 2023-2024 European guidelines do not consider PSA50 as the sole criterion, for response evaluation, its clinical and prognostic significance is well recognized [23].

The median time to CR was 18 months (range 6 - 30 months). Resistance can emerge within the first months of therapy, with the incidence increasing from 8.5% at 1 month to 17.4% at 5 years. Similar observations have been reported elsewhere: Hellerstedt B.A. *et al.* in Michigan and Diaz M. *et al.* in Florida documented median progression-free survival (PFS) of 12 - 30 months following treatment initiation [24] [25]. In Kinshasa, Moningo reported a CR rate of 43.1% with a median PFS of 17 months [7]. Our relatively low early resistance rate (8.5%) is consistent with estimates of primary resistance, which range from 10% to 30% (**Table 10**).

Table 10. Castration resistance rates over time.

Clinical context	Typical timeline to CRPC	Remarks	Source
Metastatic disease at baseline (mHSPC)	Median ≈ 18 - 36 months (many series report 2 - 3 years)	Faster progression with high tumor burden or elevated PSA	[26] [27]
Localized/ non-metastatic disease (treated)	Small proportion progress rapidly to CR; 10 - 20% at 5 years in some global estimates	Strongly dependent on initial risk (Gleason, PSA, stage)	[27] [28]
The novo mHSPC (recent series)	~23% - 35% progress to mCRPC at 5 years (depending on cohort)	Varies with population and intensification treatments	[29] [30]
General ADT patient population (mixed)	10% - 20% become CR within 5 years (reported range)	Rough estimate: depends on mix (localized vs metastatic)	[27] [28]

The probability of CR was significantly higher in patients with baseline PSA ≥ 100 ng/mL, SGOT ≥ 38 UI/L, and advanced tumor stage cT3 - cT4. Both bivariate and multivariate analyses confirmed these variables as independent risk factors. Patients with high initial PSA levels showed a resistance incidence of 20.5 cases per 100 person-months, compared to 7.2 for those with low PSA. Similarly, elevated SGOT was associated with a higher risk of CR (RRa = 1.82; $p = 0.024$).

The presence of metastases at diagnosis increased crude risk nearly ninefold and remained significant after adjustment (RRa = 2.83; $p = 0.014$). High ISUP score (3 - 5), cT3 - cT4 stage, and high or very high risk according to D'Amico/NCCN/Stampede classification also increased resistance risk. These clinical, biological, and radiological factors are therefore essential for assessing the likelihood of CR in PCa treatment.

Moningo *et al.* used similar parameters to evaluate CR, demonstrating that elevated PSA levels, Gleason 8 - 10 (ISUP 4 - 5), high progression risk, presence of metastases, and advanced clinical stage (cT3 - cT4) are strongly associated with increased risk of CR [7], consistent with other studies [9] [31].

In our cohort, the median overall survival (OS) was 28 months (range: 13 to 60.5 months), comparable to that reported by Moningo in Kinshasa (30 months) and Kurt İnci in Türkiye (30.44 months) [7] [32]. In contrast, Morgan in the United Kingdom observed a longer OS of 48.2 months, whereas Hellerstedt in the United States reported a shorter OS ranging from 8 to 16 months [24] [33]. B. Sine at Aristide Le Dantec Hospital of Dakar, found a median survival of 14 months [34].

Survival rates in our study were relatively high: 97.3% at 3 months, 93.4% at 1 year, and 84.5% at 5 years, closely aligning with those reported in other studies [7] [9]. Conversely, Bolla in France documented a markedly lower 5-year survival rate of 30% among patients with castration-resistant prostate cancer (CR), highlighting the poor prognosis associated with advanced disease [35].

5. Conclusion

Prostate cancer (PCa) is a major public health issue in our region. It mainly affects patients aged 70 to 79 years, often with diagnosis occurring at an advanced stage. First-generation hormonal therapy (FGHT) shows notable efficacy, with 82.1% positive responses. The clinical response evolves progressively, allowing many patients to remain hormone-naïve at five years. HTG1 therefore remains a relevant therapeutic option in our context of resource-limited settings.

Data Availability

The DAS confirms the presence of data.

Limitations of the Study

This study, the first of its kind in our context, has several limitations. Its monocentric and retrospective design led to the loss of certain relevant data, thereby limiting the generalizability of the results. Additionally, the low socio-economic status

of many patients restricted access to complementary paraclinical investigations, which may have influenced the assessment of progression-free and overall survival.

Strengths of the Study

This study is the first in our region to evaluate the positive response rate to first-generation hormone therapy. It confirms the results of a previous study titled ("Evolution of androgenic deprivation in treatment of prostate cancer in Kinshasa") regarding the timing and factors associated with castration resistance. Furthermore, it provides a foundation for future research exploring the combined use of first- and new-generation hormone therapies.

Author Contributions

- **Dieudonné Molamba Moningo**: Study design and supervision.
- **Serge Mbiya Nsumba**: Research, data collection, and manuscript writing.
- **Bienvenue Massamba Lebwaze**: Pathological analysis.
- **Nkodila Aliocha**: Statistical analysis.
- **Other authors**: Critical review, comments, and suggestions.

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Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

References

[1] Sung, H., Ferlay, J., Siegel, R.L., Laversanne, M., Soerjomataram, I., Jemal, A., *et al.* (2021) Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. *CA*: *A Cancer Journal for Clinicians*, **71**, 209-249. https://doi.org/10.3322/caac.21660

[2] Mashinda, K.D., Kayembe, K.P. and Mapatano, M.A. (2012) Prévalence du cancer en République Démocratique du Congo: Données anatomopathologiques recueillies aux Cliniques Universitaires et à l'Hôpital Général de Référence de Kinshasa. *Annales Africaines de Médecine*, **5**, Article 1091.

[3] Shore, N.D., Saad, F., Cookson, M.S., George, D.J., Saltzstein, D.R., Tutrone, R., *et al.* (2020) Oral Relugolix for Androgen-Deprivation Therapy in Advanced Prostate Cancer. *New England Journal of Medicine*, **382**, 2187-2196. https://doi.org/10.1056/nejmoa2004325

[4] Seront, E. (2020) Systemic Treatment in Metastatic Prostate Cancer. Programme PGMO 2020-2021, Cliniques universitaires Saint-Luc. https://www.bordet.be/fichiers_web/PGMO_2020_2021/PGMO_2020_6/1.%20Seront%20-%20Systemic%20treatment%20for%20prostate%20cancer.pdf

[5] D'Amico, A.V., Whittington, R., Malkowicz, S.B., *et al.* (1998) Biochemical Outcome

after Radical Prostatectomy, External Beam Radiation Therapy, or Interstitial Radiation Therapy for Clinically Localized Prostate Cancer. *Journal of the American Medical Association*, **280**, 969-974. https://doi.org/10.1001/jama.280.11.969

[6] Feldman, B.J. and Feldman, D. (2001) The Development of Androgen-Independent Prostate Cancer. *Nature Reviews Cancer*, **1**, 34-45. https://doi.org/10.1038/35094009

[7] Moningo, D.M., Liloku, J.K., Mafuta, A.T., Loposso, M.N., Diangienda, P.N., Maole, A.M.P., *et al.* (2021) Evolution of Androgenic Deprivation in Treatment of Prostate Cancer in Kinshasa. *Open Journal of Urology*, **11**, 137-157. https://doi.org/10.4236/oju.2021.114014

[8] Ngandu, T.J., Kabongo, J.M., Punga, M.A., Lebwaze, M.B., Moningo, D., Munabe, K.K.P. and Roux, J.J. (2015) Valeur du PSA dans le diagnostic du cancer de la prostate à Mbujimayi. *Revue Médicale des Grands Lacs*, **6**, 195-204.

[9] Moreira, D.M., Howard, L.E., Sourbeer, K.N., Amarasekara, H.S., Chow, L.C., Cockrell, D.C., *et al.* (2016) Predictors of Time to Metastasis in Castration-Resistant Prostate Cancer. *Urology*, **96**, 171-176. https://doi.org/10.1016/j.urology.2016.06.011

[10] Rozet, F., Hennequin, C., Beauval, J.B., Beuzeboc, P., Cormier, L., *et al.* (2016) CCAFU recommandations nationales sur le cancer de la prostate 2016 2018. *Progrès en Urologie*, **27**, S95-S143. https://doi.org/10.1016/s1166-7087(16)30705-9

[11] Brureau, L., Moningo, D., Emeville, E., Ferdinand, S., Punga, A., Lufuma, S., *et al.* (2016) Polymorphisms of Estrogen Metabolism-Related Genes and Prostate Cancer Risk in Two Populations of African Ancestry. *PLOS ONE*, **11**, e0153609. https://doi.org/10.1371/journal.pone.0153609

[12] Moningo, D., Blanchet, P. and Maole, A. P. (2018) Profil épidémiologique et déterminants génétiques du cancer de la prostate à Kinshasa. Éditions universitaires européennes, 91-113.

[13] Belabed Zaket, A. (2015) Cancer de prostate (étude épidémiologique) (No. 48). https://fac.umc.edu.dz/snv/faculte/biblio/mmf/2015/14 - 2015.pdf

[14] Punga Maolea, A.M.L., Moningo, D.M., Kayembe, P.K., Tshikuela, M.L. and Kabongo, J.M. (2008) Étude de dépistage du cancer de la prostate au sein d'une population d'employés d'une entreprise de Kinshasa en république démocratique du Congo. Taux de détection et facteurs de risques nutritionnels et géographiques. *Progrès en Urologie*, **18**, 512-518. https://doi.org/10.1016/j.purol.2008.04.009

[15] Miyahira, A.K., Lang, J.M., Den, R.B., Garraway, I.P., Lotan, T.L., Ross, A.E., *et al.* (2016) Multidisciplinary Intervention of Early, Lethal Metastatic Prostate Cancer: Report from the 2015 Coffey-Holden Prostate Cancer Academy Meeting. *The Prostate*, **76**, 125-139. https://doi.org/10.1002/pros.23107

[16] Namiki, M., Ueno, S. and Kitagawa, Y. (2012) Role of Hormonal Therapy in Treatment of Advanced Prostate Cancer. *Translational Andrology and Urology*, **1**, 160-172.

[17] Raghavan, D. (2018) First Line Use of Novel Hormonal Agents in Prostate Cancer: A Critical Appraisal. *Clinical Advances in Hematology & Oncology*, **16**, 289-295.

[18] Buti, S., Bersanelli, M., Facchinetti, F., Banna, G.L. and Ciccarese, C. (2022) The Role of Fast and Deep PSA Response in Castration Sensitive Prostate Cancer. *World Journal of Urology*, **40**, 137-145.

[19] Sato, H., Narita, S., Tsuchiya, N., Koizumi, A., Nara, T., Kanda, S., *et al.* (2018) Impact of Early Changes in Serum Biomarkers Following Androgen Deprivation Therapy on Clinical Outcomes in Metastatic Hormone-Sensitive Prostate Cancer. *BMC Urology*, **18**, Article No. 32. https://doi.org/10.1186/s12894-018-0353-4

[20] Hussain, M., Goldman, B., Tangen, C., Higano, C.S., Petrylak, D.P., Wilding, G., *et*

al. (2009) Prostate-Specific Antigen Progression Predicts Overall Survival in Patients with Metastatic Prostate Cancer: Data from Southwest Oncology Group Trials 9346 (Intergroup Study 0162) and 9916. *Journal of Clinical Oncology*, **27**, 2450-2456. https://doi.org/10.1200/jco.2008.19.9810

[21] Sweeney, C.J., Chen, Y., Carducci, M., Liu, G., Jarrard, D.F., Eisenberger, M., *et al.* (2015) Chemohormonal Therapy in Metastatic Hormone-Sensitive Prostate Cancer. *New England Journal of Medicine*, **373**, 737-746. https://doi.org/10.1056/nejmoa1503747

[22] Fizazi, K., Tran, N., Fein, L., Matsubara, N., Rodriguez-Antolin, A., Alekseev, B.Y., *et al.* (2017) Abiraterone Plus Prednisone in Metastatic, Castration-Sensitive Prostate Cancer. *New England Journal of Medicine*, **377**, 352-360. https://doi.org/10.1056/nejmoa1704174

[23] Mottet, N., van den Bergh, R.C.N., Briers, E., *et al.* (2023) EAU-EANM-ESTRO-ESUR-ISUP-SIOG Guidelines on Prostate Cancer (2023 Édition). European Association of Urology. https://uroweb.org/guidelines/prostate-cancer

[24] Hellerstedt, B.A. and Pienta, K.J. (2002) The Current State of Hormonal Therapy for Prostate Cancer. *CA*: *A Cancer Journal for Clinicians*, **52**, 154-179. https://doi.org/10.3322/canjclin.52.3.154

[25] Diaz, M. and Patterson, S.G. (2004) Management of Androgen-Independent Prostate Cancer. *Cancer Control*, **11**, 364-373. https://doi.org/10.1177/107327480401100604

[26] Karantanos, T., Corn, P.G. and Thompson, T.C. (2013) Prostate Cancer Progression after Androgen Deprivation Therapy: Mechanisms of Castrate Resistance and Novel Therapeutic Approaches. *Oncogene*, **32**, 5501-5511. https://doi.org/10.1038/onc.2013.206

[27] De Nunzio, C., Amstrong, A.J., Van Oort, I. and Dorff, T. (2022) Editor' Summary: A Paradigm Shift in Castration-Resistant Prostate Cancer Management. *Prostate Cancer and Prostatic Diseases*, **25**, 601-603. https://doi.org/10.1038/s41391-022-00574-x

[28] Shore, N.D., Laliberté, F., Ionescu-Ittu, R., Yang, L., Mahendran, M., Lejeune, D., *et al.* (2021) Real-World Treatment Patterns and Overall Survival of Patients with Metastatic Castration-Resistant Prostate Cancer in the US Prior to PARP Inhibitors. *Advances in Therapy*, **38**, 4520-4540. https://doi.org/10.1007/s12325-021-01823-6

[29] Stock, S.R., Burns, M.T., Waller, J., De Hoedt, A.M., Parrish, J.A., Ghate, S., *et al.* (2024) Racial and Ethnic Differences in Prostate Cancer Epidemiology across Disease States in the VA. *JAMA Network Open*, **7**, e2445505. https://doi.org/10.1001/jamanetworkopen.2024.45505

[30] Lowentritt, B.H., Rossi, C., Du, S., Muser, E., Kinkead, F., Waters, D., *et al.* (2023) Real-World Time-to-Castration Resistance (CR) among Patients (pts) with Metastatic Castration-Sensitive Prostate Cancer (mCSPC) Initiating Apalutamide (APA), Enzalutamide (ENZ), or Abiraterone Acetate (ABI) from an Oncology Database. *Journal of Clinical Oncology*, **41**, 65. https://doi.org/10.1200/jco.2023.41.6_suppl.65

[31] Smith, M.R., Cook, R., Lee, K. and Nelson, J.B. (2011) Disease and Host Characteristics as Predictors of Time to First Bone Metastasis and Death in Men with Progressive Castration-Resistant Nonmetastatic Prostate Cancer. *Cancer*, **117**, 2077-2085. https://doi.org/10.1002/cncr.25762

[32] İnci, B.K., Gürler, F., Sütcüoğlu, O., Baştuğ, V., Yazıcı, O., Üner, A., *et al.* (2023) Prognostic Significance of Nadir PSA Value and Time to Nadir PSA in Patients with Metastatic Castration-Naive Prostate Cancer Receiving First-Line Hormonotherapy. *Journal of Cancer Research and Therapeutics*, **19**, S845-S850.

https://doi.org/10.4103/jcrt.jcrt_1527_20

[33] Morgan, C., McEwan, P., Chamberlain, G., Cabrera, C. and Parry, D. (2010) Castration Resistant Prostate Cancer (CRPC): A UK Epidemiology Study. *Value in Health*, **13**, A26.

[34] Sine, B., Bagayogo, N.A., Thiam, A., Sarr, A., Zakou, A.R., Faye, S.T., *et al.* (2016) Cancers de la prostate de score de Gleason supérieur ou égal à 8: Evaluation de la survie des patients. *African Journal of Urology*, **22**, 243-248. https://doi.org/10.1016/j.afju.2016.01.011

[35] Bolla, M., Maingon, P., Carrie, C., Villa, S., Kitsios, P., Poortmans, P.M.P., *et al.* (2016) Short Androgen Suppression and Radiation Dose Escalation for Intermediate and High-Risk Localized Prostate Cancer: Results of EORTC Trial 22991. *Journal of Clinical Oncology*, **34**, 1748-1756. https://doi.org/10.1200/jco.2015.64.8055