Scientific Research Publishing

Open Journal of Urology, 2021, 11, 137-157
https://www.scirp.org/journal/oju
ISSN Online: 2160-5629
ISSN Print: 2160-5440

Evolution of Androgenic Deprivation in Treatment of Prostate Cancer in Kinshasa

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How to cite this paper: Moningo, D.M., Liloku, J.K., Mafuta, A.T., Loposso, M.N., Diangienda, P.N., Maole, A.M.P., Demongawi, R.K. and Aliocha, N. (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

Received: February 28, 2021
Accepted: April 25, 2021
Published: April 28, 2021

Abstract

Context and Objective: Prostate cancer (PCa) is hormone-dependent cancer. In our area, most patients often arrive at the locally advanced stage or the metastatic stage. This justifies the choice of androgen deprivation as the mode of treatment. The objective of this study was to describe the socio-demographic characteristics of patients with PCa. Identifying the period during which the disease remains susceptible to androgen deprivation. Assessing the patient's prognosis in terms of survival. **Methods:** This is a retrospective observational study of the course of patients managed for PCa. It involved 51 cases and was conducted at the Pointe à Pitre clinic (CPAP) in Matete Township during a period of 4 years (from March 2014 to June 2018). **Results:** The mean age of patients was 69.4 ± 9.7 years (40 - 92 years); 39.2% of patients with PCa were aged between 70 - 79 years; 45.1% had consulted for dysuria and 25.5% were hypertensive. All had performed the prostate biopsy, 47.1% were diagnosed at the metastatic stage, with PSA ≥ 100 ng/ml, Gleason scores 8 - 10, and clinical-stage TNM 3 - 4. About 51% were subjected to androcure, 23.5% had been surgically cased and 3.9% had undergone radical prostatectomy. 41.1% had resisted castration within a median of 1.4 years of response to treatment. The median survival was 30 months, with a mean survival of 26.6 months. **Conclusion:** Prostate cancer involved most of the patients in the age bracket of 70 to 79 years. The diagnosis was performed lately with a high resistance rate of castration and median survival of 30 months.

Keywords

Deprivation, Prostate Cancer (PCa), Pointe à Pitre Clinic (CPAP)

1. Introduction

Prostate cancer (PCa) is the most common cancer in men in America (Afro-Americans and Afro-Caribbean) and Europe [1] [2] [3]. Patients with localized or sometimes locally advanced forms benefit from radical prostatectomy or radiotherapy. On the other hand, in patients with aggressive or metastasized forms; the treatment options may be radiotherapy but more commonly androgen deprivation [4]. To reduce the plasma level of testosterone and its derivative, dihydrotestosterone (DHT), androgen deprivation; apart from surgical castration still used by Anglo-Saxons, other practitioners prefer hormone therapy [5]. This hormone therapy has evolved with the emergence of LH-RH analogs, then treatment with non-steroidal anti-androgens, and finally, more recently, LH-RH antagonists, and Gn-RH agonists [6]. Depending on the patient's response, this hormone therapy goes from the first to the third line [7] [8]. Over 90% of patients treated respond to androgen deprivation. Unfortunately, transiently, its duration varies from a few months to a few years. The median being 12 to 18 months, then relapse is observed in 100% of cases. After the escape from this first line of hormone therapy, responses to alternative hormonal manipulations are rare, the tumor is resistant to castration [7] [8]. Once the hormone-independence stage has been reached, the tumor is resistant to castration. Median overall survival is 34 months [6]. Castration-resistant PC (CRPC) is an advanced form characterized by disease progression after surgical or pharmaceutical castration (androgen deprivation). The process by which prostate cancer cells become resistant to castration is not clear, but androgenic privations have been shown to offer a selective advantage to androgen-independent cells, which eventually grow and repopulate the tumor [9]. Compared with castration sensitive PCa, the prognosis of patients with CRPC is poor and survival is reduced. Until very recently, treatment options were mainly limited to symptomatic relief of bone metastases, which are more common in CRPC than in the castration-sensitive form [9] [10] [11] [12]. To provide a clear picture of the burden of CRPC, one must consider the prevalence of the disease, the relative time of onset versus diagnosis, patient characteristics including demographics, comorbidity, the onset of disease, metastatic form, and probable survival. There is, however, insufficiency of epidemiological evidence specifically characterizing CRPC outside of the settings of controlled trials in which patients may not represent the general population and normal disease progression. This can lead to its sub-optimal management; for example, the identification of patients with CRPC who are at risk of developing metastases is currently hampered by a poor understanding of its real epidemiology. Identifying people with CRPC may seem straightforward after androgen deprivation (drug or surgical). The characterization of the disease in epidemiological terms, eg incidence, prevalence, and survival, is however less clear. This can be attributed at least in part to the difficulty of defining, and therefore of studying, the patient population. The varying terminology—CRPC, HRPC (Hormone-refractory PCa), AIPC (Androgen-independent PCa), ERPC (Endocrine-resistant PCa)—reflects subtle differences in dentition that may hamper

research comparison. Practitioners can also use a variety of diagnostic methods: prostate-specific antigen (PSA) assays, the development of metastases, or other factors to determine if a patient is defined as CRPC. The recently published European Association of Urology (EAU) guidelines aim to standardize the diagnosis of CRPC and include a list of five defining factors of CRPC [4].

These are as follows:

- Serum testosterone level.
- Three consecutive increases in Prostate Specific Antigen (PSA) 2 weeks apart resulting in two increases of 50% over the nadir.
- Anti-androgen stops for at least 4 weeks.
- Progression of PSA despite secondary hormonal manipulations.
- Progression of bone or soft tissue damage.

CRPC is a heterogeneous disease, and despite the availability of such guides for diagnosing CRPC, in practice, this can vary. Also, besides, the routes of treatment and clinical practice, particularly the stage of the disease at the onset of androgen deprivation therapy, vary widely between geographic locations and even the individual's clinic. Therefore, establishing common epidemiological estimates for the CRPC population becomes very complex and may become less relevant for individual scenarios [13].

In our environment, most affected patients consult at the advanced stage of the disease; thus, justifying androgen deprivation as a mode of treatment.

This study aimed to improve the clarity of the epidemiological evidence around CRPC, by identifying, assessing, and describing the most relevant elements that characterize the affected patient population using observational data.

Our objective was to assess the responses to hormonal deprivation, patient survival and to identify the different predictors of mortality.

2. Methods

Nature, Period, Framework and parameters of interest.

This is a retrospective observational epidemiological study that focused on the evolution of the 51 patients followed for PCa at the Pointe à Pitre Clinic during the period from March 2014 to June 2018. Pointe-à-Pitre Clinic is a non-profit organization, non-denominational and nonpolitical called "PROSTATE CANCER VIGILANCE AFRIQUE CENTRALE", PCVAC in acronym was created in Kinshasa, capital of the DRC on April 27, 2016. The head office of the association is in Kinshasa, within the Clinic cited above in the city of Kinshasa province in Matete Township, Kinzazi district, n˚ 11D in Matete Health District. During the entire study period, 1364 patients were received at the CPAP and we identified 165 cases of prostate cancer (12%) and among them, only 51 cases were retained to constitute our sample of coverage. Among the 165 patients, many were excluded because their follow-up was incomplete (PSA and Testerone). Our parameters of interest were age, profession, marital status, place of residence, complaints, cTNM, PSA, testosterone biopsy, prostate ultrasound, MRI results, Scintigraphy, radiography (pelvis and column), histological result, Gleason score,

stage of progression, treatment, patient survival, a predictor of mortality. The androgen deprivation method has been either surgical (bilateral orchidectomy) or chemical (hormone therapy). We used for hormonotherapy, Cyproterone acetate (50 - 100 mg), Gosereline (10.8 mg) Bicalutamide (50 mg), and Docetaxel for the chemotherapy. Some patients who have undergone radical prostatectomy have received complete hormone therapy or orchidectomy for PCa recurrence.

2.1. Inclusion Criteria

We considered patients' files with a PCa which was treated and operated during the period of our study.

2.2. Non Inclusion Criteria

Incomplete or absent files on during data collecting have not been taken into account.

2.3. Collection of Data

We collected data by completing an ad hoc form related to the documentary review focused on the medical records and registers of patients.

3. Statistical Analyses

The data were computerized using Excel 2010 software and were analyzed using SPSS version 17 software. Tables or graphs were used, as appropriate, for the presentation of the results. The continuous quantitative variables with Gaussian distribution were presented as mean ± standard deviation; those with non-normal distribution in the form of the median (extremes). Qualitative variables were described as relative frequency (%). Comparison of proportions, medians, and means was performed using Chi-square, Mann Whitney Wilcoxon, and Student's t-tests, respectively. Independent determinants of resistance to surgical castration and PSA ≥ 100 ng/ml were identified using logistic regression. Kaplan Meier's method estimated the probability curve of resistance to surgical castration. It also described survival between the date of diagnosis of CaP and death (complete data) and the end of the study (censored data). The Log-rank test was used to compare survival curves. Cox's regression looked for independent predictors of mortality.

A p-value < 0.05 was considered the threshold of statistical significance.

Ethical Considerations

During the collection and analysis of our data, confidentiality was strictly enforced.

4. Results

4.1 General Characteristics of the Study Population

About 51 patients, the average age was 69.4 ± 9.7 years, with extremes of 40 to 92

years. The age range of 70 to 79 years was the most common (39.2%). Obstructive signs and irritative signs were respectively 72.5% and 33.3%. Dysuria was the obstructive symptom that most prompted patients to consult (45.1%) followed by nycturia (23.5%) like irritative symptoms. Hypertension was the most common comorbidity (25.5%). It appears that; 47.1% of PCa were diagnosed at the metastasis stage, followed by cancers at high risk of progression (25.5%). Only 7.8% of cancers were at low risk. The metastases were from localizations variables; bone (50%), multiples (20%), ganglion (16.7%) and testicles (12.5%). The year 2014 saw more consultations (37.2) and the highest mortality rate was observed during the year 2017 (50%). The overall mortality rate was 19.6% and overall survival was 80.4% (Table 1).

Table 1. General characteristics of the population of the study.

Variables	PSA (ng/ml) Average (extreme)	Sample (n = 51)	Pourcentage (%)	Death n (%)
Age (year) Average		**69.4 ± 9.7 (40 - 92)**		
<60 years		7	13.7	-
60 - 69 years		17	33.3	-
70 - 79 years		20	39.2	-
≥80 years		7	13.7	-
Symptoms				
Obstructive signs		37	72.5	-
Irritative signs		17	33.3	-
Haematuria		4	7.8	-
Medical history				
Arterial hypertension		13	25.5	-
Diabetes		6	11.8	-
PSA	51, 5 (34.3- ≥100, 0)	51	100	-
Stage of diagnosis				
Metastatic cancer		24	47.1	-
High-risk cancer		13	25.5	-
Medium risk cancer		10	19.6	-
Low-risk cancer		4	7.8	-
Metastasis locations		n (24)	47.1	-
Bones		12	50.0	-
Nods		4	16.7	-
Testis		3	12.5	-
Multiples		5	20.8	-
Annual frequency and death				**10 (19.6)***
2014		19	37.2	1 (5.2)
2015		6	11.7	2 (33.3)
2016		14	27.4	1 (7.1)
2017		12	23.5	6 (50)
General survival in 4 years		41	80.4	-

*Global mortality in 4 years.

4.2. Treatment and Evolution

The treatment varied depending on the case (Hormone therapy, Surgery, chemotherapy):

- Ciproterone Acetate (Androcure) 50.9%;
- The Goserelin-Bicalutamide combination in 45.0%;
- Surgical castration in 23.5%;
- Surgical castration was associated with TURP in 11.8% of cases;
- Radical prostatectomy in 3.9%.

From an evolutionary point of view; the rate of castration resistance was 43.1% within a median of 1.4 (1 - 3) years of response to treatment (Table 2).

4.3. Evaluation of Castration Resistance

Castration resistance was observed from the 5th month of treatment, especially for carrier patients with metastases.

4.4. Resistance to Castration According to Gleason Score

Patients with a Gleason score between 8 and 10 had a higher frequency of resistance compared to those with a Gleason score between 6 and 7; log-rank test ($p = 0.018$) (Figure 1).

4.5. Risk of Resistance According to the D'AMICO Classification

According to D'AMICO's classification, the risk of castration resistance was variable:

- Twice for the intermediate-risk PC [OR 2.02 95% CI (1.45 - 3.90); P = 0.021];
- Three for high-risk PC [OR 2.95 95% CI (1.36 - 4.69); P = 0.041];
- And 6 times for metastatic CaP [OR 5.88 95% CI (1.62 - 7.99); P = 0.019] (Table 3).

Table 2. Distribution of patients according to treatment mode.

Variables	Sample (n = 51)	Pourcentage (%)
Traitement		
Hormonotherapy	49	95.9
Ciprotérone acetate	26	50.9
Goselerine/Bicalutamide	23	45.0
Goselrine/Bicalutamide Chemotherapy	2	3.3
Surgery	28	54.9
Surgical Castration	12	23.5
TURP alone	8	15.7
TURP + surgical castration	6	11.8
Radical Prostatectomy	2	3.9
Evolution		
Castration resistance	22	43.1

Table 3. Distribution of resistance according to the D'AMICO classification.

Classification of D'Amico	Resistance to castration		p	OR (IC95%)
	No	Yes		
Low risk cancer	2 (6.9)	2 (9.1)	0.818	1
Intermediate risk cancer	7 (24.1)	3 (13.6)	0.021	2.02 (1.45 - 3.90)
High risk cancer	8 (27.6)	5 (22.7)	0.041	2.95 (1.36 - 4.69)
Metastatic cancer	12 (41.4)	12 (54.5)	0.019	5.88 (1.62 - 7.99)

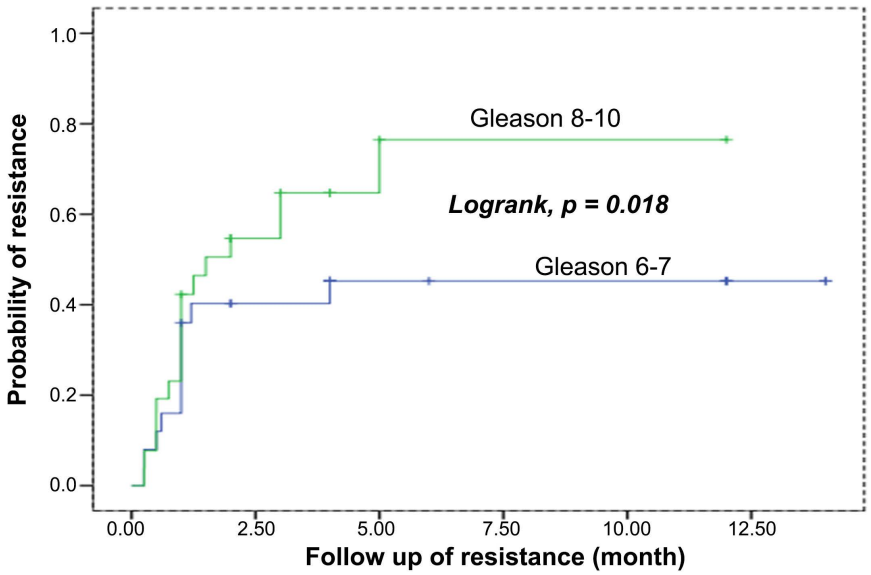

Figure 1. Gleason score and castration resistance.

4.6. Resistance to Castration According to Clinical Signs

Dysuria came first in 39.1%, followed by pollakiuria in 30.4%, nocturia in 26.1%, and bone pain in 21.1% of cases (Table 4).

4.7. Assessment of Prostate Specific Antigen Rate and Testosterone (ng/ml)

During treatment, the PSA level tended to decrease for all patients. Its increase has been observed in some patients from the 4th dosage. The mean PSA was 51.5 (0.3 - 2528.7) ng/ml for un median of 7.75 ng/ml. However, the testosterone level which reached the castration rate still tended to cancel out. Its average was 2.8 ng/ml (0.5 - 8.15).

4.8. Prostate Specifin Antigen Evaluation According to Age, cTNM, and Gleason Score

The PSA level was not statistically significant (p 0.779) compared to the ages of the patients. The increase in PSA level was influenced by clinical stage cT3 - cT4 [OR 15.0 95% CI (2.02 - 17.11); p = 0.006], with a statistically significant difference in cT1 - cT2 (p 0.006) and score Gleason [OR 6.07 95% CI (1.49 - 24.76)] without any statistically significant difference in score 6 - 7 (p = 0.011) (Table 5).

Table 4. Distribution of clinical signs in patients resistant to castration.

Paramètres	Sample (n = 23)	Pourcentage (%)
Dysuria	9	39.1
Pollakiuria	7	30.4
Nycturia	6	26.1
Bone pain	5	21.7
Urinary incontinence	4	17.4
Mictalgia	3	13.0
Drop by drop urination	3	13.0
LLE	2	8.7

Table 5. Distribution of Prostate Specific Antigen levels by age, cTNM, and Gleason score.

Variables	PSA (ng/ml)			p	OR (IC95%)
	All	<100	≥100		
Age				0.779	
<60 years	7 (13.7)	3 (11.5)	4 (16.0)		1
60 - 69 years	17 (33.3)	8 (30.8)	9 (36.0)		0.86 (0.13 - 5.82)
70 - 79 years	20 (39.2)	13 (50.0)	7 (28.0)		0.54 (0.09 - 3.41)
≥80 years	7 (13.7)	2 (7.7)	5 (20.0)		1.50 (0.14 - 16.54)
cTNM				0.006	
cT1 - cT2	14 (76.9)	7 (53.8)	7 (58.3)		1
cT3 - cT4	11 (23.1)	6 (46.2)	9 (41.7)		15.0 (2.02 - 17.11)
Gleason				0.011	
6 - 7	25 (49.0)	13 (50.0)	12 (48.8)		1
8 - 10	26 (51.0)	13 (50.0)	13 (52.2)		6.07 (1.49 - 24.76)

4.9. Assessment of Patients' Survival

The probability of patient survival was 92.2% at 10 months, 84.3% at 15 months, 82.4% at 20 months, and 80.4% at 48 months, respectively. The median patient survival was 30 (24 - 30) months and the mean survival was 26.6 months.

4.10. Assessment of Patients' Survival versus Castration Resistance

Patients resistant to castration had significantly reduced survival compared to those who did not (p = 0.029) (**Figure 2**).

4.11. Assessment of Survival about Prostate Specific Antigen Level

The survival of patients with a PSA level ≥ of 100 ng/ml (p = 0.006) was significantly lower compared to the others (**Figure 3**).

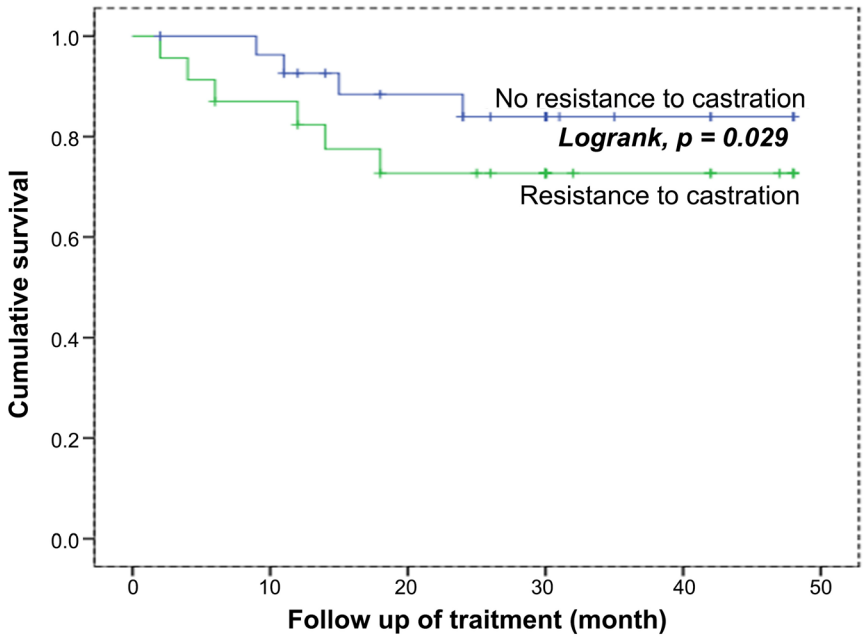

Figure 2. Patients' survival versus castration resistance.

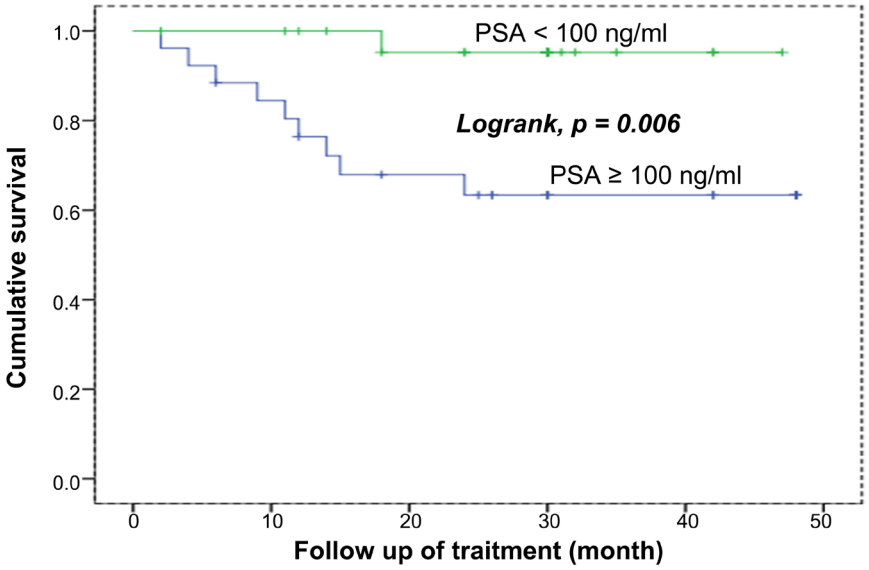

Figure 3. Survival curve as a function of the prostate specific antigen level.

4.12. Assessment of Survival about Gleason Score

The survival of patients with a Gleason score of 8 - 10 (p 0.004) was significantly lower than those with a score of 6 - 7 (**Figure 4**).

4.13. Predictors of Mortality

In univariate analysis; PSA levels ≥ 100 ng/ml [HR 10.20 95% CI (1.29 - 13.56); p = 0.001], Gleason score 8 - 10 [HR 10.97 98% CI (1.39 - 16.68); p = 0.035] castration resistance [HR 3.98 95% CI (1.56 - 7.04); p = 0.017] and metastases [HR 2.67 95% CI (1.69 - 10.35), p = 0.007] were predictors of mortality, without any

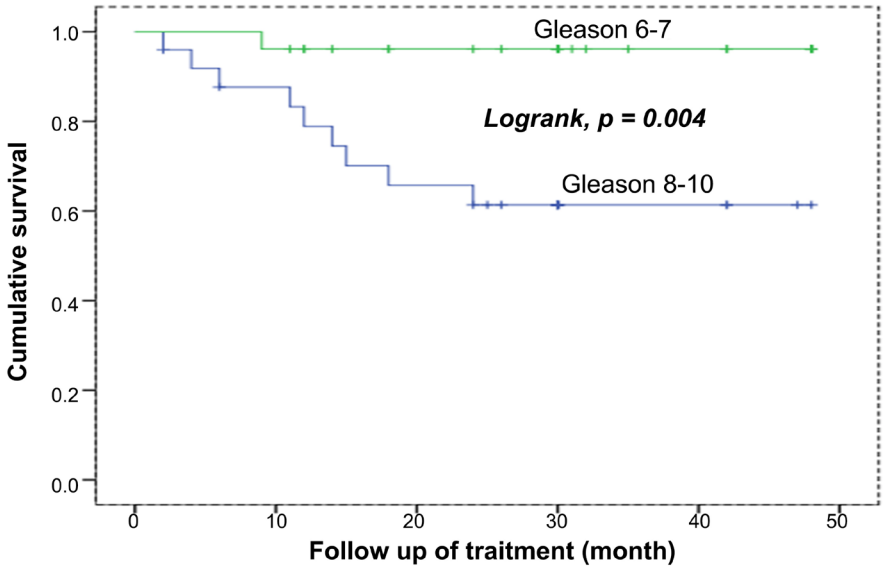

Figure 4. Survival about Gleason score.

significant difference within each group (**Table 6**). In multivariate analysis, the Gleason score 8 - 10 [HRa 10.15 95% CI (2.18 - 12.23); p = 0.035] and the PSA ≥ 100 [HRa 8.49 95% CI (2.15 - 10.56); p = 0.001] were more evident as predictors of mortality (**Table 6**).

5. Discussion

The current study is one of the few to have explored the course of androgen deprivation in 51 patients treated for PCa Apart from the response to this hormonal deprivation, the objective was to evaluate the survival of the patients and to look for the different predictors of mortality. In **Table 1**, it was from the fourth decade that PCa was diagnosed in our patients with an average age of 69.4 years (40 - 92 years). Many studies report either an average around the sixth decade [3] [14] [15] [16] [17] or around the seventh decade [18]-[24] with extremes that do not show significant differences. Only one reports an average of 59.13 years [25] (**Table 7**).

Hypertension was the most common comorbidity in 25.5% of cases. Studies have explored the association of PCa with hypertension as comorbidity and report different results. Some report hypertension as the only comorbidity; 33.3% [17] and 35.4% [26] cases. Others show an increased risk of PC [27], death [28], and an increased prevalence of PCa in hypertensive Africans [29]. Most of the patients were carriers of PCa diagnosed at the stage of metastasis followed by cases at high risk of progression and intermediate risk. Those of low risk have been rare. The same results are described in the literature, with the predominance of metastatic cancers for some authors [15] [21] [24] [29] [30]. Others report more on low-risk and intermediate-risk cancers [31] [32] [33]. Indifferent types of studies. Bone metastases predominated, followed by multiple locations and lymph nodes. PCa is first recognized as osteophytes cancer before any other

Table 6. Distribution of patients according to the predictors of mortality.

Variables	Univaried Analysis		Multivaried Analysis	
	p	HR (IC95%)	p	HRa (IC95%)
PSA (ng/ml)				
<100		1		1
≥100	0.028	10.20 (1.29 - 13.56)	0.001	8.49 (2.15 - 10.56)
Gleason				
6 - 7		1		1
8 - 10	0.023	10.97 (1.39 - 16.68)	0.035	10.15 (2.18 - 12.23)
Metastatic cancer				
No		1		1
Yes	0.015	2.67 (1.69 - 10.35)	0.007	4.77 (1.89 - 6.32)
Castration resistance				
Yes		1		1
No	0.029	3.98 (1.56 - 7.04)	0.017	3.15 (1.25 - 5.36)
cTNM				
cT1 - 2		1		1
cT3 - 4	0.035	2.16 (1.44 - 6.74)	0.736	1.35 (0.24 - 7.71)

Table 7. Char of mean age according to authors.

Authors	Country	Mean Age	Year
Carl K. *et al.* [14]	Colombia	66.0 (49 - 70)	2007
Laurent Brureau *et al.* [3]	Antilles	68.0 (46 - 95)	2009
Laurent Brureau *et al.* [15]	Guadeloupe	66.4 (46 - 95)	2016
B. Sine *et al.* [16]	Sénégal	68.5 (53 - 82)	2016
Dieudonné Moningo *et al.* [17]	DRC	68.9 (43 - 88)	2018
Hwang *et al.* [18]	USA	73.0-	2004
Smith MR *et al.* [19]	NR	75.0 (60 - 80)	2005
Henry Botto *et al.* [20]	France	70.6 ± 8.8 and 72.2 ± 7.1	2007
Daniel *et al.* [21]	USA	70.0 (67 - 81)	2016
Mohamed Ait Chtouk [22]	Morocco	75.0 (51 - 99)	2016
Rozet *et al.* [23]	France	70.0-	2016
Maha Hussain *et al.* [24]	UK	74.0 (50 - 95)	2018
Ngandu TJ *et al.* [25]	Mbujimayi (DRC)	59.1 (57 - 60)	2015
Our study	DRC	69.4 (40 - 90) (**Table 1**)	2020

NR: No reported.

localization [21] [34] [35] [36] [37] [38]. We used clinical signs, PSA and testosterone level, cTNM stage, Gleason score, and medical imaging to assess castration resistance in our patients. Everything was summed up in D'Amico's classification. Numerous studies have explored similar parameters in various ways to assess castration resistance in PCa [39] [40] [41]. Most patients had benefited from castration (hormonal or surgical), radical prostatectomy was very rare (3.9%) (**Table 2**). These results are almost like those found in the literature [20]

[42] [43] [44]. The castration-resistant cancer rate (CRPC) was 43.1% within a median of 1.4 (1 - 3 years) year (17 months) of response to treatment (**Figure 1**). Our results are different from those of other authors [11] [34] [45] [46] [47] [48]. Other studies [49] report that 10% to 20% of PCa evolve into CRPC approximately 5 years after the start of treatment (**Table 8**).

Gleason score 8 - 10 (log-rank; p = 0.018) (**Figure 1**), High-risk Cap [OR 2.95 95% CI (1.36 - 4.69)] (**Table 3**), metastatic [OR 5.88 95% CI (1.62 - 7.99)] (**Table 3**), and dysuria (**Table 4**) were providers of CPRC. These same results are reported by many researchers [11] [21] [34] [44] [49] [50] [51] [52]. The increase in PSA level was influenced by clinical stage cT3 - cT4 [OR 15.0 95% CI (2.02 - 17.11); p = 0.001], and Gleason score 8 - 10 [OR 6.07 95% CI (1.49 - 24.76); p = 0.035] (**Table 5**). These same results are repeated in many studies [53]-[58]. In terms of percentage, patient survival was 94.7%, 88.0%, 89.7%, and 80.4% at 1 year, 2 years, 3 years, and 4 years, respectively. Three groups of auteurs report report different survival percentages. The first indicates a survival at 1 year, 2 years, 3 years, and 4 years between 80% to 90% [16] [21], the second suggests a 5-year survival of 30% for CRPCs [55]. In the end, the Henry Botto team evokes a survival of 21.1%% at 8 years [20].

In terms of months or years, the median patient survival was 30 [24]-[30] months and the mean survival was 26.6 months. Most studies already published report a median survival that varies between 14 to months, with certain differences depending on the stage, grade, and comorbidity [16] [18] [19] [20] [24] [34] [53] [59] [60]. Patients resistant to castration (LogRank, p = 0.029) (**Figure 2**), those with a PSA level ≥ 100 ng/ml (LogRank, p 0.006) (**Figure 3**) or a Gleason score 8 - 10 (LogRank, p 0.004) (**Figure 4**) had significantly reduced survival compared to the others. Other researchers come to the same conclusion [11] [21] [38] [61]. Searching for predictors of mortality; in univariate analysis; PSA levels ≥ 100 ng/ml [HR 10.20 95% CI (1.29 - 13.56); p = 0.001], Gleason score 8 - 10 [HR 10.97 98% CI (1.39 - 16.68); 0.035] resistance to castration [HR 3.98 95% CI (1.56 - 7.04); p = 0.017] and metastases [HR 2.67 95% CI (1.69 - 10.35); p = 0.007] were predictors of mortality, without any significant difference within each group. In multivariate analysis, the Gleason score 8 - 10 [HRa 10.15 95% CI (2.18 - 12.23); p = 0.035] and the PSA level ≥ 100 [HRa 8.49 95% CI (2.15 - 10.56) p = 0.001] (**Table 6**), were more prominent as predictors of mortality.

Table 8. Chart of castration resistance according to the authors (34).

Reference (Autor)	Type (Study)	Country (Year)	Age (Patients)	Period (Study)	Prevalence (CRPC)
Alemayehu [45]	Retrospective	USA (2001-2007)	≥40 ans	>6 years	17.8%
Morgan [46]	Retrospective	UK (1998-2008)	≥40 ans	>10 years	11.2%
Berruti [47]	Prospective	Italy (1996-2003)	47 - 87 years	55 months	53%
Bianco [49]	Retrospective	USA (1990-1999)	-	1 - 145 months (Médiane 55 months)	19%
Our study	Retrospective	DRC (2014-2016)	40 - 92 years	4 years	43.1%

The same observation is made by many authors namely; the mortality is all the higher for high-grade, metastatic PCa, RCPC and a very high PSA rate [21] [36] [51] [61] [62] [63].

6. Conclusion

Prostate cancer is a public health challenge in our area. The average age of the patients was 69.4 years (40 - 92 years). Dysuria was the main symptom of medical consultation. Hypertension was the main comorbidity among our patients. Most of our patients have been diagnosed at metastasis stage or a high-risk stage of progression. Castration resistance was observed from the 5th month of treatment, especially for patients with metastases. The median patient survival was 30 months and the average survival was 26.6 months with a difference depending on the stage, grade, and comorbidity. The overall mortality rate was 19.6%. The patients with castration resistance had significantly reduced survival.

Limitations of the Study

This publication is considered as a pilot study which will be validated by others. The interpretation of the results should consider the limitations. The lack of randomization of the subjects studied and the limited to one center introduced a selection bias and does not allow the generalization of our results. The retrospective nature of our cohort constitutes a significant loss of some useful information. The low socio-economic level of the patients made it impossible to carry out several paraclinical examinations which have a definite influence on the survival of patients. Finally, the relatively small size of the sample could not give enough power to statistical tests to detect possible associations between the variables of interest. To get around these methodological pitfalls, a multicenter randomized longitudinal cohort study is expected.

Authors' Contribution

Dieudonné Molamba Moningo: Research design and supervision.
Junior Konga Liloku: Data collection and writing.
Richard Koseka Demongawi: French English translator.
Nkodila Aliocha: Statistical analysis.
Other authors: Corrections, remarks, and suggestions.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix

DATA COLLECTION SHEETS

Subject: "EVOLUTION OF ANDROGENIC DEPRIVATION IN TREATMENT OF PROSTATE CANCER IN KINSHASA"

Date of collection at the Pointe à Pitre/Matete Clinic ……

March 2014 to June 2018.

I. IDENTITY

Coded	……
Place and date of birth	……
Weight (kg)	……..... kg
Height (cm)	……..... cm
Marital status	Married: ☐
	Divorced: ☐
	Single: ☐
	Widower:
Age (year)	……. years
Address	C: ……...
	Q: …….....
	Av: …….....
Profession	.....
Province of origin	.....
Phone number	.....

II. MEDICAL HISTORY

1) Personal

- Smoker	Yes ☐ No ☐
- Former smoker	Yes ☐ No ☐
- How many stems/day	.....
- Allergy to a drug	Yes ☐ No ☐
- (which)?	.....
- Hypertensive	Yes ☐ No ☐
- Diabetic	Yes No
- Fracture, in the absence of major trauma	
(If yes, which fracture site)	.....
- Hyperuricemia	Yes ☐ No ☐
- Alcohol	Yes ☐ No ☐

2) Family history

- Prostate cancer	Yes ☐ No ☐
- Breast cancer	Yes ☐ No ☐

III. CLINICAL FINDING

Dominant symptom	.....
Functional signs: 1. pain:	
- Pain in RT	Yes ☐ No ☐
- Bone pain	Yes ☐ No ☐

2) Urinary disorder:

- dysuria Yes ☐ No ☐
- incontinence Yes ☐ No ☐
- mictalgia Yes ☐ No ☐
- pollakiuria Yes ☐ No ☐
- nocturia Yes ☐ No ☐
- resumes post-voiding Yes ☐ No ☐

3) Rectal touch:

- nodule Yes ☐ No ☐
- invasion Yes ☐ No ☐
- lumbar contact Yes ☐ No ☐
- Lower limb edema Yes ☐ No ☐

IV. PARACLINIC

Imaging:

- endorectal ultrasound Yes ☐ No ☐
- MRI Yes ☐ No ☐
- Abdomino-pelvic scanner Yes ☐ No ☐
- bone scan Yes ☐ No ☐

Prostate biopsy:

- Performed Yes ☐ No ☐
- Positive result Yes ☐ No ☐

Method of realization:

- echoguided Yes ☐ No ☐
- transrectal Yes ☐ No ☐

Initial stage

- localized cancer Yes ☐ No ☐
- locoregional cancer Yes ☐ No ☐
- metastatic cancer Yes ☐ No ☐

Clinical classification/Imaging, cTNM cT.....N.....M.....

Gleason score .....

Testosteroneemia (ng/ml) (1.....), (2.....), (3.....), (4.....)

PSA (ng/ml) (1.....), (2.....), (3.....), (4.....)

D'AMICO classe: Low risk (1), Intermediate risk, (2) High risk (3), Metastatic (4)

V. TREATMENT

1) Hormone therapy used R/.....

R/.....

R/.....

Start of treatment (month/year) .....

Duration .....

2) Anti-androgen therapy? Yes ☐ No ☐

Which? R/.....

R/.....

R/.....

3) Surgical castrations .....

.....

Others R/.....

Complications:

- Early .....

.....

-late .....

.....

Evolution of castration .....

.....

.....

Evolutions of testosterone .....

.....

.....

Time to onset of castration resistance after treatment is indicated

.....

.....

Death Yes ☐ No ☐

Death with cancer Yes ☐ No ☐

Age: .....

Time after treatment.....

Other causes .....