Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Clinicopathological Characteristics of Early-Onset Prostate Cancer in Black African Men: A Single-Centre Cohort from Johannesburg

Eesaa Cassim Docrat
University of the Witwatersrand
Ekene Emmanuel Nweke
University of the Witwatersrand
Shingai Mutambirwa
Sefako Makgatho Health Sciences University
Sean Doherty
University of the Witwatersrand
Akram A.H. Bokhari
University of Hail
Timothy R. Rebbeck
Harvard TH Chan School of Public Health and Dana-Farber Cancer Institute
Wenlong Carl Chen
University of the Witwatersrand
Ahmed Adam
ahmed.adam@wits.ac.za

University of the Witwatersrand

Research Article

Keywords: prostate cancer, early-onset, South Africa, Black African, diagnostic pathway, prostatitis

Posted Date: November 12th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11565655/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Purpose

Early-onset prostate cancer (EOPC) is poorly characterised in African populations. We describe the clinicopathological features of men aged ≤ 55 years diagnosed with prostate cancer at a Johannesburg public-sector hospital.

Methods

We retrospectively analysed men aged ≤ 55 years with histologically confirmed prostate adenocarcinoma enrolled at the Chris Hani Baragwanath Academic Hospital site of the Men of African Descent and Carcinoma of the Prostate (MADCaP) Consortium between October 2016 and August 2020. High-risk or advanced disease and clinically significant disease were study-defined composites adapted from EAU risk groups. Exploratory logistic regression assessed associated factors.

Results

Seventy-seven of 1,095 men (7.0%) were aged ≤ 55 years; median age was 53 years. High-risk or advanced disease was present in 38 men (49.4%) and clinically significant disease in 56 (72.7%). Initial PSA exceeded 20 ng/mL in 35 men (45.5%), 23 (29.9%) had ISUP Grade Group 4–5 disease, and distant metastases were documented in 8 of the 69 men with an assessable M stage (11.6%). Median initial PSA was 17.8 ng/mL. Prior prostatitis or inflammation was recorded in 63 men (81.8%), and 53 (68.8%) had been treated for urinary symptoms before the cancer diagnosis. Abnormal digital rectal examination was associated with both study endpoints after adjustment, although confidence intervals were wide.

Conclusion

Early-onset disease accounted for 7.0% of prostate cancers at this centre; half met criteria for high-risk or advanced disease and almost three-quarters for clinically significant disease. The frequency of prior prostatitis diagnoses and urinary treatment highlights the need to better understand diagnostic pathways in younger men.

Introduction

Prostate cancer is the second most commonly diagnosed cancer and the fourth leading cause of cancer death among men worldwide, according to latest GLOBOCAN estimates [1]. In South Africa, late-stage presentation, advanced or high-grade disease and high mortality remain major challenges [2–9], with prostate cancer the most commonly diagnosed cancer and the second most leading cause of cancer-related mortality amongst men [10]. These patterns highlight the need for locally relevant data,

Early-onset prostate cancer (EOPC), typically defined as prostate cancer diagnosed at age ≤ 55 years [11–14], represents a clinically important subgroup. Younger men have a longer potential period during which disease progression and treatment-related morbidity may occur. Furthermore, some studies have reported a greater frequency of adverse pathological features and inherited susceptibility in this population [11–13, 15–17].

Detailed clinicopathological characterisation of early-onset prostate cancer in African settings remains limited. The presentation and disease spectrum of early-onset prostate cancer in South African men remains poorly characterised. This study aimed to describe the sociodemographic, clinical and pathological profile of men ≤ 55 years diagnosed with prostate cancer at Chris Hani Baragwanath Academic Hospital.

Methods

Study Design, Setting and Population

This was a retrospective descriptive cohort included all 77 men aged ≤ 55 years at diagnosis with histologically confirmed prostate adenocarcinoma enrolled at the Chris Hani Baragwanath Academic Hospital (CHBAH) site of the Men of African Descent and Carcinoma of the Prostate Consortium (MADCaP), Johannesburg, between October 2016 and August 2020 [18].

Data Collection

Data were obtained from the existing de-identified MADCaP database and linked clinical records. Extracted variables included demographic, socioeconomic, clinical, pathological, staging, and survival status follow-up.

Prostate biopsies were performed under transrectal ultrasound guidance using a systematic protocol. Magnetic resonance imaging was not available for biopsy targeting during the study period. The number of cores containing adenocarcinoma were recorded.

Metastatic staging was based on bone scan with additional PSMA PET, CT, or radiography where indicated, following the standard approach at this centre [19]. Eight men had no recorded M stage and were classified as having no documented metastatic disease for the descriptive analysis, so the reported metastatic prevalence is a minimum estimate. Nodal staging was based on clinical and imaging records and was incompletely characterised; one patient had documented regional nodal involvement. Clinical T stage was derived from digital rectal examination alone [20]. Disease burden is therefore reported principally by ISUP Grade Group and PSA.

Follow-up for survival was ascertained by three-monthly telephone calls and, when necessary, a national administrative database; follow-up ended at database closure [20].

Definitions and Derived Variables

Early-Onset Prostate Cancer was defined as prostate adenocarcinoma diagnosed at age ≤ 55 years, consistent with commonly used epidemiological and clinical definitions [11–13, 15].

Initial PSA was defined as the higher of the referral PSA and diagnostic or biopsy PSA.

High-risk or advanced disease was a study-defined composite endpoint based on recognised EAU high-risk and advanced disease features [21]. It was defined as the presence of one or more of the following:

- Initial PSA > 20 ng/mL;
- ISUP Grade Group 4–5 (Gleason 8–10);
- clinical stage ≥cT3;
- regional nodal involvement;
- or distant metastatic disease (M1) [21].

Clinically significant disease was defined as a broader study-defined endpoint encompassing high-risk or advanced disease, ISUP Grade Group 3, or Grade Group 2 with initial PSA 10–20 ng/mL. Adapted from EAU risk groups to the available variables, it was intended to capture unfavourable intermediate-risk disease or worse [21].

Statistical Analysis

Continuous variables were summarised as median (IQR) and categorical variables as n (%). Full-cohort percentages used a denominator of 77; missing or undocumented binary features were coded as absent, while subgroup analyses used relevant denominators. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Continuous variables were compared using the Mann–Whitney U test.

Exploratory multivariable logistic regression assessed associations of abnormal DRE, positive biopsy-core burden and HIV status with high-risk or advanced and clinically significant disease; variables defining an endpoint were excluded as predictors. A sensitivity analysis removed clinical stage from the high-risk or advanced endpoint because T stage was DRE-derived. No metastatic-disease model was fitted because only eight metastatic cases were documented. No formal sample-size calculation or adjustment for multiple comparisons was performed; $p < 0.05$ was used as a nominal significance threshold and regression findings were considered exploratory. Analyses were performed in StataNow 19.5 (StataCorp, College Station, TX, USA).

Language-editing tools were used in the preparation of this manuscript. All authors reviewed and approved the final content and take full responsibility for its accuracy and integrity.

Results

Cohort and Clinical Characteristics

A total of 1,095 men with prostate cancer enrolled at this site between October 2016 and August 2020; of these, 77 (7.0%) were aged 55 years or younger at diagnosis (median 53; IQR 50–54; range 37–55) and formed the study cohort. Eighteen (23.4%) were younger than 50; all self-identified as Black African, and 57 (74.0%) were referred from primary healthcare.

Among 60 men with income data, 39 (65.0%) reported household income ≤R1,850. Nine (11.7%) had a first-degree family history and 34 (44.2%) reported lower urinary tract symptoms. Median IPSS was 5 (IQR 2–11; n = 75), with 52 (69.3%) having mild symptoms (Table 1). An abnormal or suspicious digital rectal examination (DRE) was documented in 34 patients (44.2%). Baseline characteristics are summarised in Table 1.

Prior healthcare contact for urinary symptoms was common. A previous diagnosis of prostatitis or prostatic inflammation was recorded in 63 patients (81.8%), and 53 (68.8%) had received treatment for urinary symptoms prior to prostate cancer diagnosis; 46 (59.7%) reported both. Treatment continued for at least 3 months in 29 men (37.7%; 54.7% of the 53 treated) and for ≥ 5 months in 16 (20.8%; 30.2% of those treated).

Table 1
Baseline sociodemographic and clinical characteristics of EOPC cases

Characteristic	Result
Total clinical cohort	77
Age, median (IQR), years	53 (50–54)
Age < 50 years	18 (23.4%)
Foreign-born	9 (11.7%)
Referred from primary healthcare clinics	57 (74.0%)
Currently employed	39 (50.6%)
Monthly household income ≤R1,850, n/N (%)	39/60 (65.0%)
BMI, median (IQR), kg/m²	26.3 (22.5–30.5)
Hypertension	29 (37.7%)
Documented HIV infection	26 (33.8%)
Diabetes mellitus	8 (10.4%)
Previous tuberculosis	4 (5.2%)
First-degree family history of prostate cancer	9 (11.7%)
Current smoker	31 (40.3%)
Ever smoker	51 (66.2%)
Current alcohol use	35 (45.5%)
Lower urinary tract symptoms	34 (44.2%)
IPSS, median (IQR)	5 (2–11)
Erectile dysfunction	27 (35.1%)
Abnormal or suspicious DRE	34 (44.2%)
Prior prostatitis or prostatic inflammation	63 (81.8%)
Prior urinary treatment	53 (68.8%)

All variables were abstracted from medical records at study enrolment. Lower urinary tract symptoms and IPSS refer to symptoms at presentation, whereas prior prostatitis and prior urinary treatment refer to the period before the prostate cancer diagnosis. Values are n (%) unless otherwise indicated. Binary clinical features are reported as documented presence using the full cohort denominator of 77 patients.

Variables requiring an observed measurement or response use the available denominator shown. The income denominator excludes 17 patients who chose not to answer the income question.

PSA, Histopathology and Staging

Median initial PSA was 17.8 ng/mL (IQR 9.1–73.1), with 35 patients (45.5%) exceeding 20 ng/mL. The median number of positive biopsy cores was 6/12 (IQR 3–7). Gleason score 7 predominated (40/77; 51.9%), while 23 patients (29.9%) had high-grade disease (ISUP Grade Group 4–5). Most had cT1c disease (43/77; 55.8%). Distant metastases were documented in 8/69 men with assessable M stage (11.6%; 95% CI 6.0–21.2%); eight lacked recorded M stage. Detailed tumour characteristics are shown in Table 2.

Table 2
PSA, histopathology, stage, and disease-risk features

Characteristic	Result	95% CI[a]
Initial PSA, median (IQR), ng/mL	17.8 (9.1–73.1)	-
Initial PSA > 20 ng/mL	35 (45.5%)	34.8 to 56.5
Initial PSA > 50 ng/mL	24 (31.2%)	21.9 to 42.2
Initial PSA > 100 ng/mL	16 (20.8%)	13.2 to 31.1
Positive biopsy cores (of 12), median (IQR)	6 (3–7)	-
ISUP GG1 (Gleason 3 + 3=6)	14 (18.2%)	11.2 to 28.2
ISUP GG2 (Gleason 3 + 4=7)	27 (35.1%)	25.3 to 46.2
ISUP GG3 (Gleason 4 + 3=7)	13 (16.9%)	10.1 to 26.8
ISUP GG4 (Gleason 8)	6 (7.8%)	3.6 to 16.0
ISUP GG5 (Gleason 9–10)	17 (22.1%)	14.3 to 32.5
ISUP GG4–5 (Gleason ≥ 8)	23 (29.9%)	20.8 to 40.8
Clinical stage T1c	43 (55.8%)	44.7 to 66.4
Clinical stage T2a	15 (19.5%)	12.2 to 29.7
Clinical stage T2b	10 (13.0%)	7.2 to 22.3
Clinical stage T2c	7 (9.1%)	4.5 to 17.6
Clinical stage T3/T3a	2 (2.6%)	0.7 to 9.0
Documented regional nodal involvement	1 (1.3%)	0.2 to 7.0
Documented metastatic disease	8 (10.4%)	5.4 to 19.2
High-risk or advanced disease	38 (49.4%)	38.5 to 60.3
Clinically significant disease	56 (72.7%)	61.9 to 81.4

[a] *Confidence intervals for proportions were calculated using the Wilson method.*

Initial PSA was defined as the higher of the referral PSA and diagnostic/biopsy PSA. High-risk or advanced and clinically significant disease were study-defined composite endpoints. Documented metastatic disease refers to recorded M1 disease; patients without documented M1 disease were classified as having no documented metastasis for the primary descriptive analysis.

Disease endpoints and associated factors

High-risk or advanced disease was present in 38/77 patients (49.4%). Study-defined clinically significant disease was present in 56/77 patients (72.7%), comprising 38 patients with high-risk or advanced disease and an additional 18 with unfavourable intermediate-risk disease. Among the 56 patients with clinically significant disease, 21 (37.5%) had an initial PSA ≤ 20 ng/mL.

On unadjusted analysis, abnormal DRE and greater positive biopsy core burden were associated with both endpoints. Median positive core burden was 6.0 (IQR 4–9) versus 4.5 (2–7) in men with and without high-risk or advanced disease ($p = 0.026$), and 6.0 (4–8) versus 4.0 (2–6) in men with and without clinically significant disease ($p = 0.004$). IPSS was higher in men with high-risk or advanced disease ($p = 0.020$), but did not differ significantly by clinically significant disease status. HIV status was not associated with either endpoint Table 3 presents full unadjusted comparisons.

In multivariable analysis, abnormal DRE remained associated with high-risk or advanced disease (adjusted OR 7.55; 95% CI 2.62–21.74; $p < 0.001$) and clinically significant disease (adjusted OR 12.35; 95% CI 2.50–61.01; $p = 0.002$). Positive biopsy core burden remained associated with clinically significant disease (adjusted OR 1.33 per additional core; 95% CI 1.05–1.68; $p = 0.018$), but not high-risk or advanced disease after adjustment (adjusted OR 1.15; 95% CI 0.96–1.37; $p = 0.140$). HIV infection was not independently associated with either endpoint.

Table 3
Factors associated with high-risk or advanced and clinically significant disease **A. High-Risk or Advanced disease**

Variable	**High-risk or advanced disease (n = 38)**	**No high-risk or advanced disease (n = 39)**	**p-value**
Abnormal/suspicious DRE	26/38 (68.4%)	8/39 (20.5%)	< 0.001
Positive biopsy cores, median (IQR)	6.0 (4–9)	4.5 (2–7)	0.026
IPSS, median (IQR)	6.5 (3–13)	3.0 (2–10)	0.020
Prior prostatitis/inflammation	28/38 (73.7%)	35/39 (89.7%)	0.083
Prior urinary treatment	26/38 (68.4%)	27/39 (69.2%)	1.000
Documented HIV infection	15/38 (39.5%)	11/39 (28.2%)	0.341
Current smoker	19/38 (50.0%)	12/39 (30.8%)	0.106
Low income*	24/32 (75.0%)	15/28 (53.6%)	0.107

B. Clinically significant disease

Variable	Clinically significant disease (n = 56)	Not clinically significant disease (n = 21)	p-value
Abnormal/suspicious DRE	32/56 (57.1%)	2/21 (9.5%)	< 0.001
Positive biopsy cores, median (IQR)	6.0 (4–8)	4.0 (2–6)	0.004
IPSS, median (IQR)	5.0 (2-9.5)	6.0 (2–12)	0.787
Prior prostatitis/inflammation	45/56 (80.4%)	18/21 (85.7%)	0.746
Prior urinary treatment	39/56 (69.6%)	14/21 (66.7%)	0.790
Documented HIV infection	20/56 (35.7%)	6/21 (28.6%)	0.601
Current smoker	26/56 (46.4%)	5/21 (23.8%)	0.116
Low income*	33/46 (71.7%)	6/14 (42.9%)	0.061

Values are n/N (%) or median (IQR). P-values compare patients with and without the stated outcome; categorical variables were analysed using chi-square or Fisher's exact tests and continuous variables using the Mann–Whitney U test. Positive biopsy core burden was available for 76 patients, with 1 patient missing data. *Low income was defined as monthly household income ≤R1,850; 17 patients declined to provide income data (n = 60).

Follow-Up and Outcomes

Median follow-up from diagnosis was 3.56 years (IQR 2.71 to 4.61). At last recorded follow-up, 62 men (80.5%) were alive, 7 (9.1%) had died, and 8 (10.4%) had an unknown status or were lost to follow-up. Among the seven deceased patients, 5 met the high-risk or advanced disease definition, and all seven had clinically significant disease. Cause of death was recorded as prostate cancer in three men, all of whom had metastatic Grade Group 5 disease.

Discussion

Seventy-seven of the 1,095 men enrolled at this site (7.0%) were aged ≤ 55 years at diagnosis; 49.4% had high-risk or advanced disease and 72.7% study-defined clinically significant disease. Metastases were documented in 8/69 men with assessable M stage, indicating substantial clinically important disease without documented distant spread.

Compared with broader South African prostate cancer cohorts, the disease profile in these younger men appeared to contain a greater proportion of clinically important but still non-metastatic disease. Tindall et al., whose cohort was not restricted to younger men, reported PSA ≥ 20 ng/mL in 83% of Black South African patients and Gleason score > 7 in 36%, compared with 45.5% and 29.9% Grade Group 4–5

disease, respectively, in the present cohort [22]. Differences in age, referral pathways, geographic setting and period of recruitment limit direct comparison.

Metastatic disease was less frequently documented in these younger men than in the wider CHBAH cohort recruited over the same period (8/77 [10.4%] vs 233/1,076 [21.7%]) [19]. However, this comparison should be interpreted cautiously because the cohorts overlap and metastatic staging was incomplete. Nevertheless, the combination of substantial clinically significant disease with predominantly non-metastatic presentation may represent an opportunity for earlier diagnosis and curative-intent treatment.

The median initial PSA of 17.8 ng/mL may also reflect the broader diagnostic context in which these patients presented. In Southern Africa, referral delays, socioeconomic barriers, uneven specialist access and limited awareness may contribute to delayed prostate cancer diagnosis [5–7], although these factors were not assessed here.

Amongst African men, a Nigerian cancer-registry study reported prostate cancer < 55 years in 355 of 4,091 (8.7%), but provided little detail on PSA, histological grade or stage [23], while a study of 71 Afro-Caribbean men with EOPC focused primarily on germline and molecular alterations [24]. Internationally, Xu et al. reported substantial poorly differentiated and advanced disease among 106 Chinese men aged ≤ 55 [25]. A recent Romanian tertiary-centre study reported that 70 of 1,742 prostate cancer cases (4.0%) occurred in men aged ≤ 55 years [26], while US SEER data reported proportions up to 9.0% [27]. The 7.0% observed at CHBAH therefore falls within the range reported in other settings, although age definitions, populations and diagnostic practices differ.

The study-defined clinically significant endpoint captured unfavourable intermediate-risk disease or worse, accommodating discordance between PSA and histological grade. This is relevant because EAU guidelines support active surveillance for low-risk and selected favourable intermediate-risk disease, but not routinely for Grade Group 3 or higher-risk disease [21].

Abnormal DRE and a higher number of positive biopsy cores were associated with both endpoints on unadjusted analysis. After adjustment for positive core number, DRE findings and HIV status, abnormal DRE remained associated with both endpoints while positive core number remained associated only with clinically significant disease. These associations are descriptive and should be interpreted cautiously, as clinical T stage was derived from DRE, while positive biopsy-core count reflects tumour burden. The wide confidence interval for the DRE association with clinically significant disease further limits interpretation.

A previous diagnosis of prostatitis or prostatic inflammation was recorded in 63 men (81.8%) and 53 (68.8%) had received treatment for urinary symptoms before prostate cancer diagnosis, indicating prior contact with healthcare services. Prior prostatitis was not more frequent among men with high-risk or advanced disease. One possible explanation is that men with markedly elevated PSA values may have been referred directly for investigation of malignancy, whereas less marked, although still abnormal, PSA elevations may initially have prompted treatment for prostatitis or other urinary conditions.

These observations raise questions about the diagnostic pathway in younger men. Prostate cancer may not always be considered early when urinary symptoms are initially attributed to prostatitis or other benign conditions. However, these data cannot establish that diagnostic delay occurred. Symptom histories were recorded retrospectively, prostatitis was not defined by uniform criteria, and the interval between first presentation and diagnosis could not be reliably reconstructed. Prospective studies are needed to determine whether earlier PSA testing and DRE in younger men with persistent urinary symptoms may improve case detection.

HIV infection was common in the cohort, reflecting the local clinical context. However, it was not independently associated with either disease endpoint; the study was not powered specifically to assess the relationship between HIV and prostate cancer phenotype.

Strengths include detailed clinicopathological characterisation of a defined cohort of younger Black African men, a consistent biopsy protocol, prespecified study-defined endpoints and sensitivity analysis. Limitations include the single-centre design, small cohort, incomplete staging in a minority, DRE-derived clinical stage, and absence of treatment or recurrence data. The smaller outcome group in the clinically significant disease model also limited precision; regression findings should therefore be considered exploratory.

Conclusion

Early-onset prostate cancer accounted for 7.0% of men enrolled in the cohort at this centre; 49.4% met criteria for high-risk or advanced disease and 72.7% for study-defined clinically significant disease, predominantly without documented metastases. A median initial PSA of 17.8 ng/mL together with frequent prior prostatitis diagnoses and urinary treatment warrant prospective assessment of diagnostic pathways and targeted earlier evaluation in younger men.

These findings identify several directions for future research and potential funding. Larger, preferably multicentre studies could combine detailed clinical phenotyping with genomic assessment, particularly in younger men with high-risk disease or a strong family history. Such work could inform region-specific approaches to early detection, genetic evaluation and treatment in African populations.

Declarations

Funding

This study was funded by National Cancer Institute grants U01CA184374 and R01CA259200 (Genetics of Prostate Cancer in Africa). The project is funded in part by the German Federal Ministry of Research, Technology and Space (01KA2220B), and in part by the Science for Africa Foundation to the Developing Excellence in Leadership, Training and Science in Africa (DELTAS Africa) programme (Del-22-008) with support from Wellcome Trust and the UK Foreign, Commonwealth and Development Office, and forms part of the EDCTP2 programme supported by the European Union.

Competing Interests

The authors have no relevant financial or non-financial interests to disclose.

Ethical Approval

Ethical approval was obtained from the University of the Witwatersrand Human Research Ethics Committee (Medical) (M22/06/73). The study was conducted in accordance with the Declaration of Helsinki.

Consent to Participate

Written informed consent for the collection and secondary analysis of clinical and genetic data was obtained from participants through the parent MADCaP study.

Data Availability

De-identified clinical data supporting the findings are available from the corresponding author on reasonable request, subject to the data-access conditions of the MADCaP Consortium.

Author Contributions

Docrat: Protocol/project development; Data collection or management; Data analysis; Manuscript writing/editing; Other: Visualization.

Nweke: Protocol/project development; Manuscript writing/editing; Other: Supervision.

Mutambirwa: Manuscript writing/editing; Other: Validation.

Doherty: Data collection or management; Manuscript writing/editing.

Bokhari: Manuscript writing/editing; Other: Validation.

Rebbeck: Manuscript writing/editing; Other: Funding acquisition.

Chen: Data collection or management; Data analysis; Manuscript writing/editing.

Adam: Protocol/project development; Manuscript writing/editing; Other: Supervision, project administration.

References

1. Sung H, Filho AM, Laversanne M et al (2026) Global cancer statistics 2024: GLOBOCAN estimates of incidence and mortality worldwide for 34 cancers in 186 countries. CA Cancer J Clin 76:e70090. https://doi.org/10.3322/caac.70090

2. Schafer EJ, Laversanne M, Sung H et al (2025) Recent Patterns and Trends in Global Prostate Cancer Incidence and Mortality: An Update. Eur Urol 87:302–313. https://doi.org/10.1016/j.eururo.2024.11.013
3. Hamdi Y, Abdeljaoued-Tej I, Zatchi AA et al (2021) Cancer in Africa: The Untold Story. Front Oncol 11:650117. https://doi.org/10.3389/fonc.2021.650117
4. Gheybi K, Mmekwa N, Lebelo MT et al (2023) Linking African ancestral substructure to prostate cancer health disparities. Sci Rep 13:20909. https://doi.org/10.1038/s41598-023-47993-x
5. Singh K, Abdel Goad EH, Ramklass SS (2015) Waiting times for prostate cancer diagnosis in KwaZulu-Natal, South Africa. S Afr Med J 105:484. https://doi.org/10.7196/SAMJ.9192
6. Marima R, Mbeje M, Hull R et al (2022) Prostate Cancer Disparities and Management in Southern Africa: Insights into Practices, Norms and Values. Cancer Manag Res 14:3567–3579. https://doi.org/10.2147/CMAR.S382903
7. Benedict MOA, Steinberg WJ, Claassen FM, Mofolo N (2023) The profile of Black South African men diagnosed with prostate cancer in the Free State, South Africa. S Afr Fam Pract (2004) 65:e1–e10. https://doi.org/10.4102/safp.v65i1.5553
8. Adeloye D, David RA, Aderemi AV et al (2016) An Estimate of the Incidence of Prostate Cancer in Africa: A Systematic Review and Meta-Analysis. PLoS ONE 11:e0153496. https://doi.org/10.1371/journal.pone.0153496
9. Marais B, Klopper G, John J (2024) Prostate cancer perspective: Africa versus the world. S Afr Med J. https://doi.org/10.7196/SAMJ.2024.v114i4.1950. 114:e1950
10. Cancer in South Africa (2008–2019). Statistics South Africa, Pretoria, South Africa
11. Gupta S, Gupta A, Saini AK et al (2017) Prostate Cancer: How Young is too Young? Curr Urol 9:212–215. https://doi.org/10.1159/000447143
12. Hussein S, Satturwar S, Van Der Kwast T (2015) Young-age prostate cancer. J Clin Pathol 68:511–515. https://doi.org/10.1136/jclinpath-2015-202993
13. Salinas CA, Tsodikov A, Ishak-Howard M, Cooney KA (2014) Prostate cancer in young men: an important clinical entity. Nat Rev Urol 11:317–323. https://doi.org/10.1038/nrurol.2014.91
14. James ND, Tannock I, N'Dow J et al (2024) The Lancet Commission on prostate cancer: planning for the surge in cases. Lancet 403:1683–1722. https://doi.org/10.1016/S0140-6736(24)00651-2
15. Bleyer A, Spreafico F, Barr R (2020) Prostate cancer in young men: An emerging young adult and older adolescent challenge. Cancer 126:46–57. https://doi.org/10.1002/cncr.32498
16. Murphy A, Cottrell-Daniels CC, Awasthi S et al (2024) Understanding and Addressing Prostate Cancer Disparities in Diagnosis, Treatment, and Outcomes Among Black Men. Cancer Control 31:10732748241275389. https://doi.org/10.1177/10732748241275389
17. Shenoy D, Packianathan S, Chen AM, Vijayakumar S (2016) Do African-American men need separate prostate cancer screening guidelines? BMC Urol 16:19. https://doi.org/10.1186/s12894-016-0137-7

18. Andrews C, Fortier B, Hayward A et al (2018) Development, Evaluation, and Implementation of a Pan-African Cancer Research Network: Men of African Descent and Carcinoma of the Prostate. J Glob Oncol 4:1–14. https://doi.org/10.1200/JGO.18.00063
19. Mapanga W, Norris SA, Craig A et al (2022) Prevalence of multimorbidity in men of African descent with and without prostate cancer in Soweto, South Africa. PLoS ONE 17:e0276050. https://doi.org/10.1371/journal.pone.0276050
20. Chikwati RP, Akokuwebe ME, Ojoniyi OO et al (2026) Factors Associated With Risk Stratification and Overall Survival of Black South African Men With Non-Metastatic Prostate Cancer. Cancer Med 15:e71628. https://doi.org/10.1002/cam4.71628
21. Cornford P, Van Den Bergh RCN, Briers E et al (2026) EAU–EANM–ESTRO–ESUR–ISUP–SIOG Guidelines on Prostate Cancer—2026 Update. Part I: Screening, Diagnosis, and Local Treatment with Curative Intent. Eur Urol 90:254–269. https://doi.org/10.1016/j.eururo.2026.04.013
22. Tindall EA, Monare LR, Petersen DC et al (2014) Clinical presentation of prostate cancer in Black South Africans. Prostate 74:880–891. https://doi.org/10.1002/pros.22806
23. Ntekim A, Folasire A, Odukoya OA (2023) The Prevalence of Prostate Cancer Among Young Men Below 55 Years of Age in Nigeria. Cancer Control 30:10732748231175255. https://doi.org/10.1177/10732748231175255
24. Loger J-S, Colomba E, Vallard A et al (2025) Germline DNA Repair and Immune-metabolic Alterations Suggest a Potentially Distinct Form of Early-onset Prostate Cancer in Men of African Descent. Eur Urol Oncol S2588–9311(25):00325–00326. https://doi.org/10.1016/j.euo.2025.11.015
25. Xu Y, Yang X, Si T et al (2016) Clinicopathological and Prognostic Factors in 106 Prostate Cancer Patients Aged ≤ 55 Years: A Single-Center Study in China. Med Sci Monit 22:3935–3942. https://doi.org/10.12659/MSM.901040
26. Dema V, Croitor A, Barna RA et al (2026) Clinicopathological Analysis of Prostate Cancer in Young Men: Experience of a Tertiary Referral Hospital in Western Romania. Medicina 62:1465. https://doi.org/10.3390/medicina62081465
27. Lin DW, Porter M, Montgomery B (2009) Treatment and survival outcomes in young men diagnosed with prostate cancer: A Population-based Cohort Study. Cancer 115:2863–2871. https://doi.org/10.1002/cncr.24324