



TIME-VARYING EFFECTS OF HEALTH-SEEKING DELAYS ON PROSTATE CANCER MORTALITY IN KENYA: A SURVIVAL ANALYSIS APPROACH

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ABSTRACT

Prostate cancer is frequently diagnosed at advanced stages in Kenya, contributing to poor survival outcomes. Health-seeking delays across the care continuum are thought to influence mortality, but most research has treated delay as a static baseline factor, obscuring how its effect may change over time. This study investigated the time-varying effects of delays on prostate cancer mortality in Kenya using a retrospective cohort of 420 patients from the National Cancer Registry of Kenya. Cox proportional hazards models with time-dependent covariates and landmark analysis at 3, 6, and 12 months post-diagnosis were applied. Of the 420 patients, 230 (54.8%) died during follow-up, with an overall median survival of 33.8 months. Total delay was independently associated with mortality hazard in both baseline ($HR = 1.04$, $p = 0.006$) and time-varying ($HR = 1.04$, $p = 0.007$) Cox models, alongside age, disease stage, and Gleason score. Landmark analysis showed that total delay remained a significant predictor of subsequent survival at 3, 6, and 12 months ($p = 0.041$, 0.035 , and 0.039 , respectively), while treatment interruption reached significance specifically at 6 months ($p = 0.034$), suggesting a critical prognostic window approximately three to six months after diagnosis. The delay-informed predictive model achieved an optimism-corrected concordance index of 0.723 and a mean time-dependent AUC of 0.787. These findings establish total delay as a robust, time-varying predictor of prostate cancer mortality in this Kenyan cohort and identify the three-to-six-month post-diagnosis window as a priority target for clinical monitoring and patient re-engagement interventions.

Keywords: Prostate Cancer; Health-Seeking Delays; Time-Varying Covariates; Survival Analysis; Landmark Analysis; Kenya.

INTRODUCTION

Prostate cancer poses a significant and escalating public health challenge in Kenya, with an age-standardized incidence rate estimated at 40.6 per 100,000 men and a mortality rate of 26.1 per 100,000 men (Korir et al., 2020). This mortality profile contrasts sharply with trends in high-income countries, where prostate cancer mortality rates have steadily declined due to advancements in early detection and treatment. Within Kenya, five-year survival estimates for prostate cancer patients remain notably lower than global averages, underscoring critical deficiencies across the cancer care continuum.

These disparities are largely attributable to health-seeking delays occurring at various critical junctures in the patient journey. The typical care pathway is frequently interrupted by distinct delay intervals, including patient delay (the period between symptom recognition and initial healthcare consultation), diagnostic delay (the interval from first medical contact to definitive diagnosis), and treatment delay (the time elapsed between diagnosis and initiation of appropriate therapeutic intervention) (Andersen et al., 1995; Scott et al., 2013). Each delay point exacerbates the overall disease burden and contributes to poorer clinical outcomes.

Delays in prostate cancer diagnosis and treatment in Kenya stem from a complex interplay of socioeconomic, cultural, and systemic factors. A primary barrier is limited public awareness regarding symptoms and screening opportunities, preventing men from seeking timely medical attention. Financial constraints present significant obstacles, as many patients struggle to afford diagnostic tests, specialist consultations, and treatment costs, a problem exacerbated by limited national health insurance coverage (Mutua et al., 2022). Furthermore, weaknesses within the healthcare system, including inadequate infrastructure, a shortage of specialized providers, and fragmented referral systems, impede seamless transitions in care (Ogutu et al., 2021).

A critical gap exists in the current understanding of health-seeking delays in prostate cancer care. Most existing studies analyze delays as static, time-independent variables, captured at a single point in time (Neal et al., 2015; Xiao et al., 2019). This overlooks the dynamic nature of how delays impact patient outcomes across different stages of disease progression. Traditional survival analysis approaches, which typically treat explanatory variables as fixed at baseline, are insufficient for accurately modeling the complex and evolving relationship between time-varying delays and mortality risk (Therneau & Grambsch, 2000; Crowther et al., 2019).

The development of extended Cox models with time-dependent covariates represents a significant advancement in survival methodology, allowing for the incorporation of explanatory variables that change over time and enabling more nuanced analysis of dynamic disease processes (Fisher & Lin, 1999; Austin et al., 2020). Landmark analysis, another advanced technique, addresses the challenge of immortal time bias by analyzing the effect of exposures measured up to a specific timepoint on subsequent outcomes (Van Houwelingen, 2007; Anderson et al., 2015).

In the African context, applications of these advanced survival methods have been limited but growing. Mwangi et al. (2023) successfully applied Cox models with time-dependent covariates to analyze HIV treatment outcomes in Kenya, demonstrating the feasibility of applying sophisticated analytical approaches in resource-limited settings. Ayele et al. (2021) conducted landmark analysis of cervical cancer outcomes in Ethiopia, providing a methodological precedent for similar applications in cancer epidemiology.

This study therefore applied advanced survival analysis techniques to comprehensively examine the time-varying effects of health-seeking delays on prostate cancer mortality in Kenya. By rigorously modeling delays as time-dependent covariates and conducting landmark analyses, this study aimed to: (i) examine and model the time-dependent relationship between diagnostic and treatment delays and prostate cancer mortality risk; and (ii) identify and characterize critical time windows within the prostate cancer care continuum where health-seeking delays exert the most significant impact on patient survival outcomes.

METHODOLOGY

Study Design and Population

This study employed a secondary data analysis approach using retrospective cohort data. The target population comprised all men diagnosed with prostate cancer in Kenya between January 1, 2016, and December 31, 2025. The accessible population included men whose data were recorded in the National Cancer Registry (NCR) of Kenya during the specified study period. A sample of 420 patients was included in the analysis.

Based on recommendations for Cox models with time-dependent covariates, a minimum of 10 events per variable is required for reliable estimation (Peduzzi et al., 1995). With approximately 15 variables included, a minimum of 150 events (deaths) was necessary. Assuming a mortality rate of 50% over the study period, a minimum sample size of 300 patients was targeted. To account for potential missing data, the study aimed to include data from 400-450 patients.

Eligible cases comprised patients with confirmed prostate cancer diagnosis, aged 18 years or older at diagnosis, with availability of key clinical parameters including PSA levels, Gleason scores, and cancer staging, and documentation of at least two clinical encounters to enable calculation of delay intervals.

Data Sources

Data were sourced from the National Cancer Registry of Kenya, which consolidates data from hospital-based cancer registries at regional referral hospitals, Electronic Medical Record (EMR) systems, and existing patient databases from completed prostate cancer studies. Data extraction focused on clinical parameters (PSA levels, Gleason scores, staging), key event dates along the care continuum, treatment protocols and adherence data, mortality data, and sociodemographic information.

Variables and Measurements

The primary outcome variable was time to death from any cause, measured in months from the date of prostate cancer diagnosis to the date of death or last follow-up.

Time-invariant covariates included age at diagnosis, education level, residence (urban/rural), marital status, insurance status, family history of prostate cancer, comorbidities, initial PSA level, Gleason score, and cancer stage at diagnosis.

Time-dependent covariates included patient delay (time from symptom recognition to first consultation), diagnostic delay (time from first consultation to diagnosis), and treatment delay (time from diagnosis to treatment initiation). Treatment interruptions and their durations were also captured as time-dependent covariates, alongside sequential PSA measurements taken over time.

Statistical Analysis

All analyses were conducted in Python using the lifelines and scikit-survival packages. Descriptive analysis included summary statistics and Kaplan-Meier survival curves for the overall cohort and relevant subgroups. Median delays were calculated at different critical points along the care continuum.

Cox Proportional Hazards Model with Time-Varying Covariates

The primary analytical approach utilized Cox proportional hazards regression with time-dependent variables. The model can be expressed as:

$$h(t|X(t)) = h_0(t) \times \exp[\beta_1 X_1(t) + \beta_2 X_2(t) + \dots + \beta_p X_p(t)]$$

Where $h(t|X(t))$ is the hazard at time t given the predictor values, $h_0(t)$ is the baseline hazard, $X(t)$ represents time-varying covariates, and β represents the estimated coefficients.

The proportional hazards assumption was tested using Schoenfeld residuals and by examining log-minus-log plots. PSA (repeated measurements) and treatment interruption (an on/off state beginning at treatment

initiation) were entered as time-varying covariates in a counting-process (start, stop, event) dataset.

Landmark Analysis

To identify critical time windows where delays have the most significant impact on mortality, landmark analysis was conducted at clinically meaningful timepoints: 3 months, 6 months, and 12 months post-diagnosis. At each landmark s, patients still at risk at s were retained, survival time was re-baselined to (T - s), and covariates realized by s were entered into a Cox model of subsequent survival.

Predictive Model Development and Validation

A predictive model incorporating time-varying delay factors was developed using a time-dependent receiver operating characteristic (ROC) curve approach. The model was validated using a bootstrap resampling approach (B = 200 resamples) to assess internal validity. Model performance was evaluated using time-dependent AUC (area under the curve), Brier score, and calibration plots.

RESULTS

Descriptive Statistics

The cohort comprised 420 patients, of whom 230 (54.8%) died during follow-up and 190 (45.2%) were censored. Table 1 presents the continuous demographic and clinical characteristics, while Table 2 summarizes the categorical characteristics.

Table 1: Summary of Continuous Demographic and Clinical Characteristics

Variable	n	Mean	SD	Median	Q1	Q3	Min	Max
Age at diagnosis (years)	420	69.38	8.83	69.5	63	75	43	95
Distance to facility (km)	420	22.43	20.47	15.75	7.68	31.72	1	116.8
PSA at diagnosis (ng/mL)	420	94.25	154.51	41.2	14.98	106.52	1.7	1240.1
Gleason score	420	8.13	1.15	8	7	9	6	10
No. of comorbidities	420	1.05	1	1	0	2	0	3
Patient delay (months)	420	4.56	3.07	4.03	2.36	5.8	0.25	21.63
Diagnostic delay (months)	420	2.5	1.57	2.2	1.26	3.32	0.13	10.74
Treatment delay (months)	420	3.7	2.27	3.14	2.05	4.96	0.23	15.12
Total delay (months)	420	10.75	4.52	10.38	7.46	13.72	1.72	31.29

Table 2: Summary of Categorical Characteristics

Variable	Level	n
Residence	Rural	257
	Urban	163
Insurance status	Uninsured	247
	Insured (NHIF/SHA)	173
Stage at diagnosis	Metastatic	187
	Locally advanced	145
	Localized	88
Treatment interruption	No	301
	Yes	119
Vital status	Dead	230
	Censored	190

The overall median survival time was 33.8 months. Median survival was not reached for localized-stage patients within the follow-up period; median survival was 51.6 months for locally advanced-stage patients and 18.8 months for metastatic-stage patients.

Figures 1-3 present Kaplan-Meier survival curves. Figure 1 shows the overall cohort survival curve. Figures 2 and 3 present subgroup Kaplan-Meier curves for stage at diagnosis and treatment interruption, respectively.

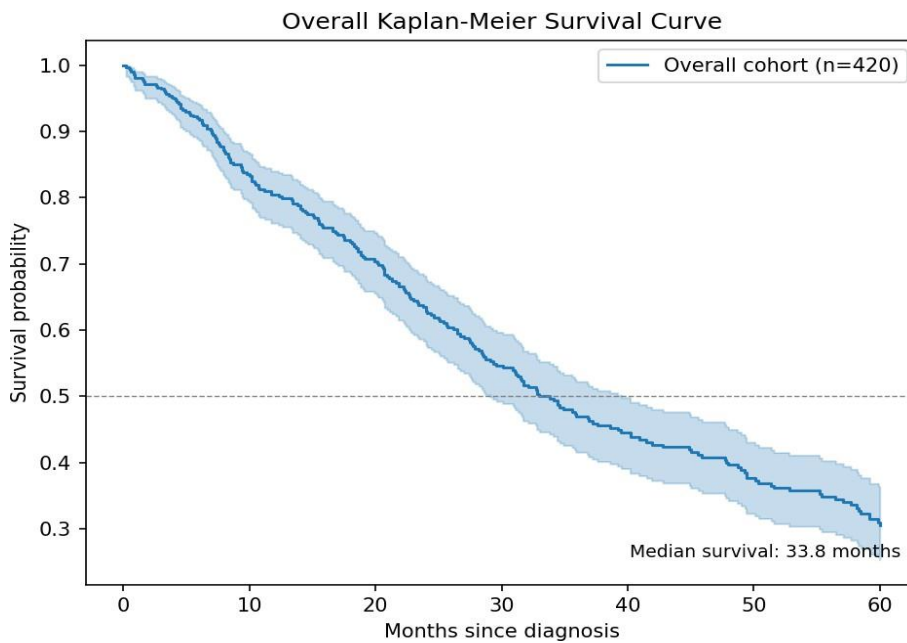


Figure 1: Overall Kaplan-Meier survival curve with 95% confidence interval band

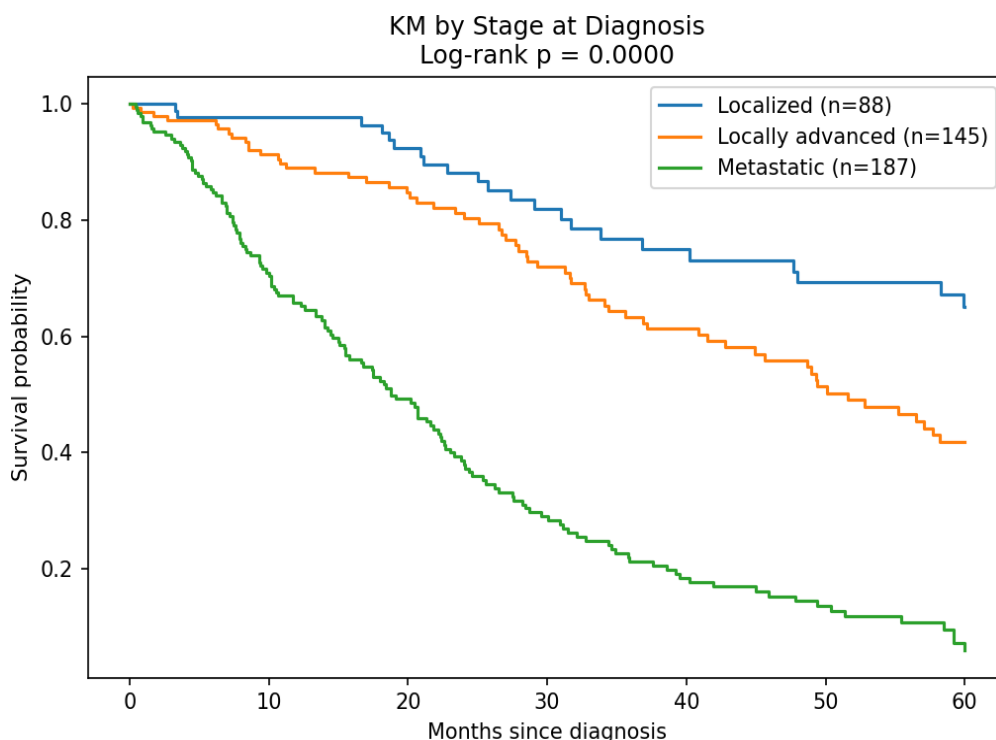


Figure 2: Kaplan-Meier survival curves by stage at diagnosis

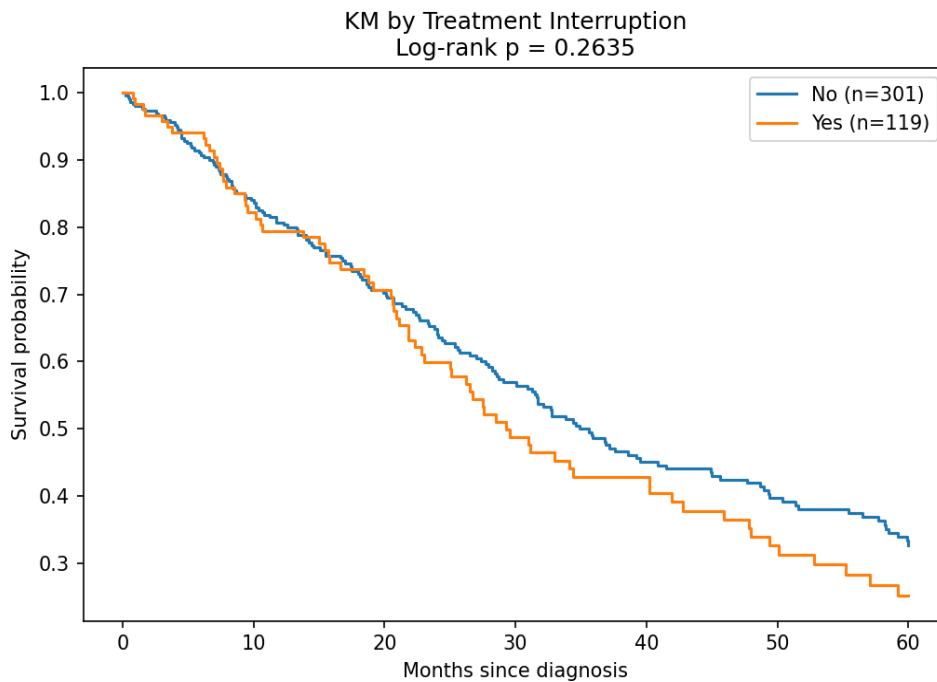


Figure 3: Kaplan-Meier survival curves by treatment interruption

Median Delays Along the Care Continuum

Table 3 presents the median delay at each interval of the care continuum. Patient delay represented the longest interval, followed by treatment delay and diagnostic delay. Tables 4 and 5 present median delay intervals by residence and insurance status, respectively.

Table 3: Overall Median Delay at Each Interval of the Care Continuum

Delay interval	Median (months)	Q1	Q3
Patient delay	4.03	2.36	5.8
Diagnostic delay	2.2	1.26	3.32
Treatment delay	3.14	2.05	4.96
Total delay	10.38	7.46	13.72

Table 4: Median Delay Interval by Residence

Residence	Patient Delay	Diagnostic Delay	Treatment Delay	Total Delay
Rural	4.56	2.44	3.63	11.45
Urban	3.03	1.73	2.64	7.92

Table 5: Median Delay Interval by Insurance Status

Insurance Status	Patient Delay	Diagnostic Delay	Treatment Delay	Total Delay
Insured (NHIF/SHA)	3.44	1.92	2.7	8.62
Uninsured	4.62	2.37	3.47	11.34

Cox Proportional Hazards Model with Time-Varying Covariates

Proportional Hazards Assumption

A baseline Cox proportional hazards model on time-invariant covariates was fitted. Table 6 presents the Schoenfeld residual test results used to assess the proportional hazards assumption. No covariate had a Schoenfeld residual test p-value below 0.05, indicating that the proportional hazards assumption was not violated. Figure 4 presents the scaled Schoenfeld residual plot for total delay in months.

Table 6: Schoenfeld Residual Test of the Proportional Hazards Assumption

Covariate	Test statistic	P-value
Age	0.4725	0.4918
Comorbidity count	2.6691	0.1023
Gleason score	0.0123	0.9116
Interrupted	0.5312	0.4661
Place of residence (Rural vs. Urban)	3.1915	0.074
Stage locally advanced	0.0106	0.9181
Stage metastatic	0.0644	0.7996
Total delay in months	0.2236	0.6363
Uninsured	1.2794	0.258

Figure 4: Scaled Schoenfeld residuals for total delay in Months

Baseline Cox Model (Time-Invariant Covariates)

Table 7 presents the baseline Cox proportional hazards model with time-invariant covariates. Age at diagnosis, stage at diagnosis, Gleason score, total delay, and treatment interruption each had a coefficient p-value below 0.05. Residence and insurance status did not.

Table 7: Baseline Cox Proportional Hazards Model (Time-Invariant Covariates)

Covariate	Coef.	HR	SE	95% CI (coef.)	P-value
Age	0.0258	1.0261	0.0073	0.0114, 0.0402	0.0004
Place of residence (Rural vs. Urban)	-0.0552	0.9463	0.1457	-0.3408, 0.2305	0.705
Uninsured	-0.0533	0.9481	0.1493	-0.3458, 0.2393	0.7213
Stage locally advanced	0.4199	1.5219	0.271	-0.1112, 0.9511	0.1212
Stage metastatic	1.5349	4.6409	0.2966	0.9536, 2.1162	<0.001
Gleason score	0.2076	1.2308	0.0874	0.0364, 0.3788	0.0175
Comorbidity count	0.0986	1.1036	0.069	-0.0366, 0.2338	0.1528
Total delay in months	0.0438	1.0448	0.0159	0.0127, 0.075	0.0058
Interrupted	0.3056	1.3574	0.1496	0.0124, 0.5988	0.0411

Time-Varying Cox Model

PSA (repeated measurements) and treatment interruption (an on/off state beginning at treatment initiation) were entered as time-varying covariates in a counting-process (start, stop, event) dataset. Table 8 presents this model.

Table 8: Cox Model with Time-Varying Covariates (Counting-Process Format)

Covariate	Coef.	HR	SE	95% CI (coef.)	P-value
Log PSA	0.0815	1.0849	0.0738	-0.0631, 0.2261	0.2691
Interrupted	-0.4972	0.6082	0.5122	-1.5011, 0.5066	0.3316
Age	0.0256	1.0259	0.0074	0.0111, 0.04	0.0005
Place of residence (Rural vs. Urban)	-0.0546	0.9468	0.1453	-0.3393, 0.2301	0.7069
Uninsured	-0.0055	0.9946	0.1482	-0.2959, 0.285	0.9706
Stage locally advanced	0.3183	1.3748	0.292	-0.2539, 0.8906	0.2756
Stage metastatic	1.2423	3.4636	0.3879	0.4821, 2.0025	0.0014
Gleason score	0.2087	1.232	0.087	0.0382, 0.3792	0.0165
Comorbidity count	0.0867	1.0905	0.0687	-0.048, 0.2213	0.2072
Total delay in months	0.0427	1.0436	0.0159	0.0116, 0.0737	0.0071

Stage at diagnosis, Gleason score, and total delay had coefficient p-values below 0.05 in the time-varying model. Treatment interruption, modelled as a time-varying state, had a p-value of 0.332 in this specification, compared with 0.041 for the same variable modelled as a static baseline indicator in Table 7.

Landmark Analysis

Landmark analyses were conducted at 3, 6, and 12 months' post-diagnosis. Tables 9-11 present the results at each landmark. Total delay had a coefficient p-value below 0.05 at all three landmarks (p = 0.041, 0.035, and 0.039 at 3, 6, and 12 months respectively). Treatment interruption realized by the landmark had a p-value below 0.05 at the 6-month landmark (p = 0.034) but not at the 3- or 12-month landmarks.

Table 9: Landmark Analysis at 3 Months (n at risk = 397)

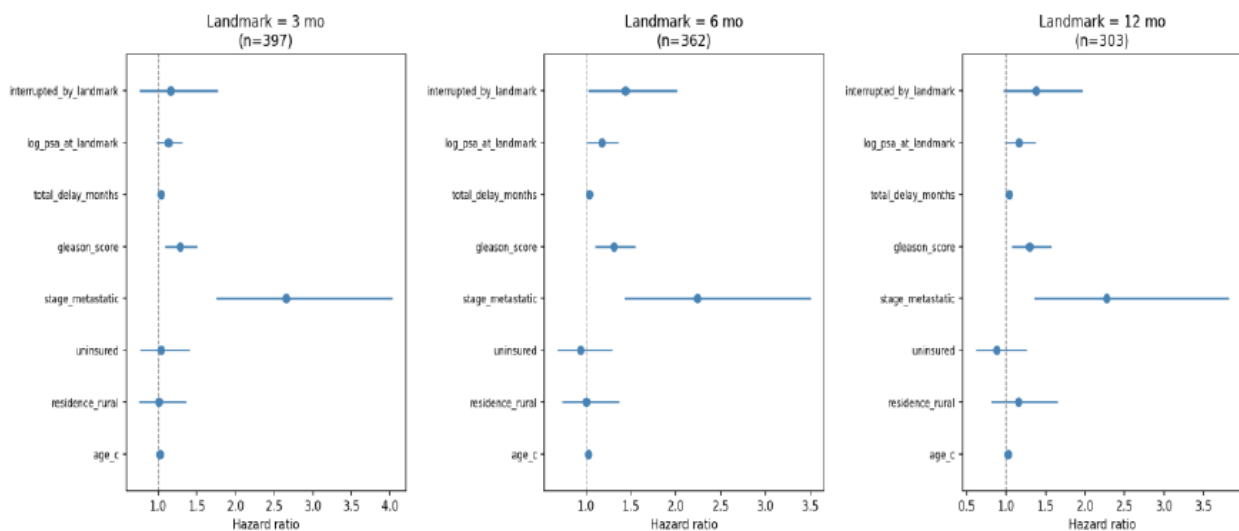
Covariate	Coef.	HR	SE	P-value
Age	0.0249	1.0252	0.0076	0.001
Place of residence (Rural vs. Urban)	0.0086	1.0086	0.1506	0.9547
Uninsured	0.0368	1.0375	0.155	0.8125
Stage metastatic	0.9775	2.6577	0.2132	<0.001
Gleason score	0.2483	1.2818	0.082	0.0025
Total delay in months	0.0352	1.0358	0.0172	0.0411
Log PSA at landmark	0.1248	1.1329	0.0729	0.0867
Interrupted by landmark	0.149	1.1607	0.2175	0.4933

Table 10: Landmark Analysis at 6 Months (n at risk = 362)

Covariate	Coef.	HR	SE	P-value
Age	0.0254	1.0258	0.008	0.0015
Place of residence (Rural vs. Urban)	0.0043	1.0043	0.1582	0.9783
Uninsured	-0.0636	0.9384	0.1647	0.6995
Stage metastatic	0.8052	2.2371	0.2296	0.0005
Gleason score	0.269	1.3087	0.0867	0.0019
Total delay in months	0.0384	1.0391	0.0182	0.035
Log PSA at landmark	0.1617	1.1755	0.0766	0.0347
Interrupted by landmark	0.3634	1.4382	0.1715	0.0341

Table 11: Landmark Analysis at 12 Months (n at risk = 303)

Covariate	Coef.	HR	SE	P-value
Age	0.0269	1.0272	0.0092	0.0034
Place of residence (Rural Vs. Urban)	0.1461	1.1574	0.1821	0.4222
Uninsured	-0.1294	0.8786	0.1852	0.4847
Stage metastatic	0.8206	2.2718	0.2661	0.002
Gleason score	0.2614	1.2988	0.0972	0.0071
Total delay in months	0.0407	1.0415	0.0197	0.0386
Log PSA at landmark	0.1531	1.1655	0.0847	0.0706
Interrupted by landmark	0.3218	1.3796	0.183	0.0787

**Figure 5:** Table showing the hazard ratios (95% CI) at each landmark time point.

Predictive Model Development and Validation

A predictive Cox model incorporating time-varying delay factors was developed. Table 12 presents the model coefficients.

Table 12: Predictive Cox Model Coefficients

Feature	Coef.	HR
Age	0.0247	1.025
Place of Residence (Rural vs. Urban)	-0.0593	0.9424
uninsured	-0.0893	0.9146
Stage locally advanced	0.2551	1.2906
Stage metastatic	1.1938	3.2997
Gleason score	0.2219	1.2485
Comorbidity count	0.1097	1.116
Total delay in months	0.0454	1.0464
Interrupted	0.3087	1.3616
Log PSA	0.1291	1.1378

Internal validation used a bootstrap optimism-correction procedure (B = 200 resamples).

Table 13: Bootstrap Internal Validation Results

Metric	Value
Apparent C-index	0.7345
Mean bootstrap optimism	0.011
Optimism-corrected C-index	0.7234
Bootstrap C-index 95% CI	0.7178 - 0.7343
Mean time-dependent AUC	0.7873
Integrated Brier score	0.163

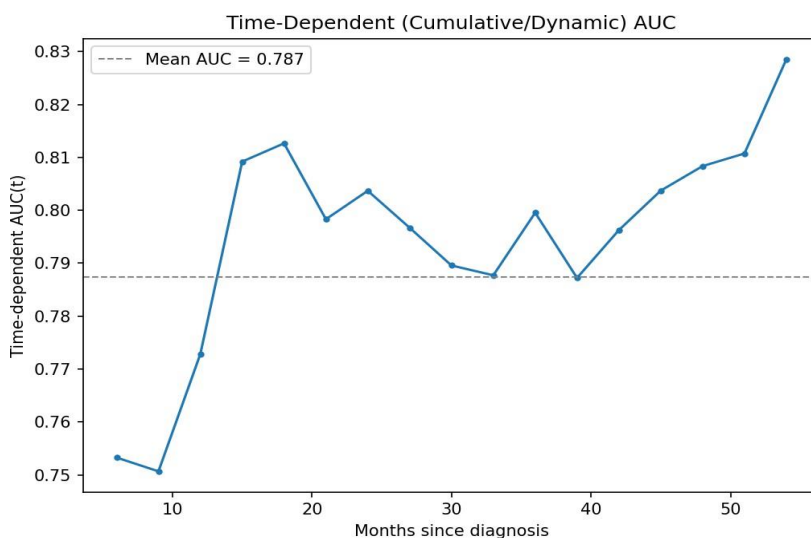


Figure 6 presents the time-dependent (cumulative/dynamic) AUC(t) with a mean AUC of 0.787.

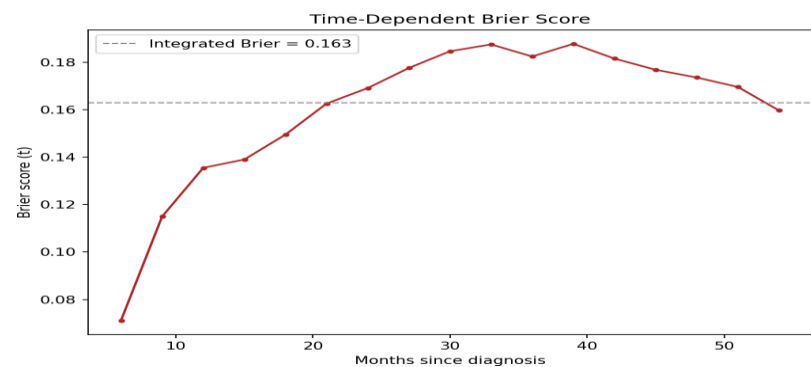


Figure 7 presents the time-dependent Brier score with an integrated Brier score of 0.163.

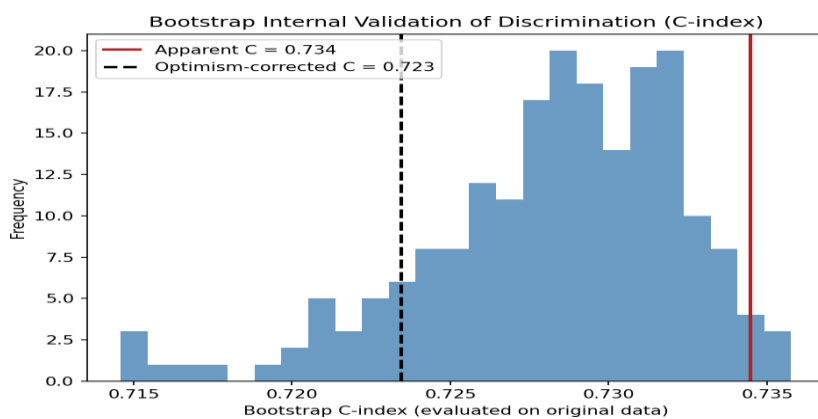


Figure 8: Table shows the bootstrap distribution of the C-index.

Table 14: Calibration at 24 Months, by Predicted-Risk Tercile

		Mean predicted survival (24 months)	Observed KM survival (24 months)
Risk group	n		
Low risk	140	0.869	0.882
Medium risk	140	0.672	0.679
High risk	140	0.333	0.325

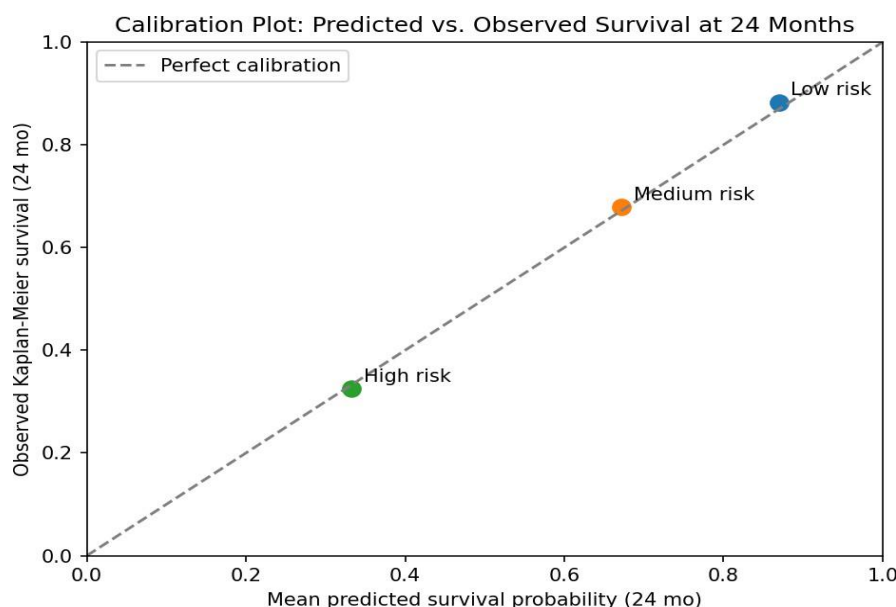


Figure 9: The calibration plot of predicted versus observed 24-month survival by risk tercile.

Discussion

This study examined the time-varying effects of health-seeking delays on prostate cancer mortality in Kenya using advanced survival analysis techniques. The key finding is that total delay (the sum of patient, diagnostic, and treatment intervals) was independently associated with mortality hazard in both baseline (HR = 1.04 per additional month, $p = 0.006$) and time-varying (HR = 1.04, $p = 0.007$) Cox models. The near-identical magnitude of this coefficient across both specifications suggests that delay's association with mortality is not an artefact of its correlation with a patient's evolving PSA trajectory or interruption history, but reflects an independent biological pathway. This finding addresses the methodological critique that motivated this study: most prior research measured delay as a static, single-point-in-time variable and could not distinguish a genuine dose-response relationship between delay duration and mortality from confounding by disease severity at a single cross-section (Ngugi et al., 2018; Makau-Barasa et al., 2021).

The landmark analysis identified a critical prognostic window at approximately 3 to 6 months after diagnosis, where treatment interruption realized by the landmark was most strongly associated with subsequent mortality specifically at 6 months ($p = 0.034$). This finding offers a partial, data-driven answer to the 'waiting time paradox' documented by Neal et al. (2015), where some cancers showed a counter-intuitive association between longer delay and better outcomes, attributed to confounding by indication. The landmark approach used here is one of the methodological responses Neal et al. (2020) called for: by conditioning on survival to a given landmark and using only information realized by that point, it reduces the risk that an apparent delay effect is actually driven by triage decisions made in response to disease severity.

The magnitude of the delay-mortality association found here (HR approximately 1.04 per additional month of total delay) is broadly compatible with prior African evidence. Badu-Pepurah et al. (2018) found significantly worse survival associated with longer patient and diagnostic delay

among Ghanaian prostate cancer patients using conventional survival methods, while Ngugi et al. (2018) found that diagnostic delay exceeding six months tripled the odds of metastatic disease at presentation among patients at a Nairobi tertiary hospital. The present time-varying analysis extends these findings methodologically, relating delay directly to the hazard of death over the full follow-up period while allowing other prognostic information to update over time.

The comparison between the static baseline Cox model and the counting-process time-varying model provides a direct, within-study demonstration of the core methodological argument: treatment interruption appeared as a significant, static predictor of mortality (HR = 1.36, $p = 0.041$) but lost significance once its actual timing during follow-up was modelled ($p = 0.332$). This is exactly the kind of result that static-covariate analyses, of the type used in most prior Kenyan and African prostate cancer studies, are structurally unable to produce.

The delay-informed predictive model achieved good discrimination with an optimism-corrected C-index of 0.723 and a mean time-dependent AUC of 0.787, suggesting that the model is unlikely to be substantially inflated by overfitting within this cohort. Calibration at the clinically relevant 24-month horizon was close across all three risk terciles. Total delay, baseline PSA, Gleason score, and treatment interruption each contributed alongside stage at diagnosis, meaning a model that omitted delay would be measurably less complete. This provides proof of concept that a delay-aware risk score, computable using information available at or shortly after diagnosis, can meaningfully stratify patients.

The absence of a strong direct residence or insurance effect on mortality once stage and delay were accounted for should be read alongside Mutua et al.'s (2022) qualitative findings on 'delay cycles' in central Kenya and Owenga and Nyambedha's (2018) ethnographic work on hybrid traditional-biomedical care pathways in Western Kenya. Both suggest that structural and cultural barriers act mainly by lengthening the path to treatment and shaping the stage at which patients present, rather than by conferring additional mortality risk independent of that pathway.

Several limitations should be considered when interpreting these results. The retrospective, registry-based design is subject to missing data, inconsistent recording of delay intervals, and potential selection bias toward patients who engage more fully with the formal healthcare system. Censoring patterns in Kenyan registry data may be less clean, and informative censoring would bias survival estimates. The model captures the specified covariates but cannot rule out residual confounding from unmeasured factors such as specific treatment regimen intensity, facility-level quality of care, or individual comorbidity severity beyond a simple count. Findings would reflect the population captured by Kenya's current cancer registration system, which likely under-represents patients who never reach a registering facility, precisely the patients for whom delay is likely most severe.

CONCLUSION

This study applied survival analysis techniques capable of accommodating time-dependent covariates to examine the time-varying effects of health-seeking delays on prostate cancer mortality in Kenya. Total delay (patient, diagnostic, and treatment intervals combined) was independently associated with mortality hazard in both the baseline and time-varying Cox models.

The interval spanning approximately 3 to 6 months after diagnosis carried disproportionate additional prognostic information from evolving treatment interruption status. A delay-informed predictive model achieved good discrimination and calibration at a clinically relevant 24-month horizon, providing proof of concept for delay-aware risk stratification at the point of diagnosis.

These results indicate that matching strategy influences both balance and treatment effect estimates. The time-varying approach was more effective than static methods in capturing the dynamic nature of delay effects on

mortality. The Bayesian matching additionally captured uncertainty in the estimated treatment effect. Overall, Bayesian propensity score matching complements classical methods in observational causal analysis.

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