

Dynamic Conditional Survival and Competing Causes of Death Among Older Adults With Nonmetastatic Small-Cell Lung Cancer Receiving Chemotherapy and First-Course Radiation: A SEER Analysis

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Research Article

Keywords: small-cell lung cancer, conditional survival, competing risks, older adults, SEER

Posted Date: October 14th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11037129/v1>

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Additional Declarations: No competing interests reported.

Abstract

Background

Prognosis estimated at diagnosis becomes less useful once a patient has survived the first year. We examined 3-year survival and competing causes of death at successive landmarks in older adults with nonmetastatic small-cell lung cancer (SCLC) whose records documented chemotherapy and first-course radiation.

Methods

We analyzed SEER Research Data (17 Registries, November 2025 Submission). Eligible 2010–2017 tumor records met prespecified age, histology, strict M0, nonsurgical, chemotherapy, and Beam-radiation criteria. Nested risk sets included records surviving to 12, 24, or 36 months. Kaplan–Meier methods estimated conditional 3-year overall survival (OS); Aalen–Johansen methods estimated cause-specific cumulative incidence functions (CIFs). Record-level bootstrap resampling compared landmarks.

Results

The cohort comprised 3422 tumor records; the landmark risk sets contained 2347, 1393, and 983 records. Conditional 3-year OS was 32.7% (95% confidence interval [CI], 30.8%–34.6%), 43.1% (40.4%–45.7%), and 47.7% (44.6%–50.8%). The L36–L12 difference was 15.1 percentage points (bootstrap 95% CI, 12.1–18.3). Across the same landmarks, SCLC-death CIF was 56.8%, 41.7%, and 30.1%, whereas other-cause-death CIF was 10.0%, 14.7%, and 21.3%. Other causes accounted for 14.9%, 26.0%, and 41.5% of deaths from known causes. Older age (per 5 years) was associated with higher other-cause mortality in exploratory models.

Conclusions

Three-year prognosis was more favorable and other causes accounted for more remaining mortality in later survivor cohorts. These estimates compare nested landmark-defined groups, not change within individuals or treatment effects.

Introduction

Small-cell lung cancer is aggressive and carries substantial early mortality. For appropriately selected patients with limited-stage disease, radiation is an established component of curative-intent treatment alongside systemic therapy [1–3]. Prognosis in older adults is also shaped by comorbidity, functional reserve, and risks unrelated to cancer [4, 5].

Survival reported from diagnosis does not directly answer a later question: what is the prognosis for someone who has already survived 1, 2, or 3 years? Conditional survival resets the starting point at a specified landmark and estimates survival over a subsequent interval [6, 7]. In population-based lung cancer studies, both conditional prognosis and the distribution of causes of death have differed among longer-term survivors [8]. SCLC studies have examined conditional survival after surgery and across broad metastatic patterns [9, 10].

SCLC death and death from another cause are mutually exclusive first events, so their absolute risks require competing-risk methods. Previous SCLC studies have described cause-specific mortality, competing-risk models, or treatment-pattern outcomes in broader and differently selected registry populations [11–13, 16–19]. Less is known about how a fixed 3-year prognosis and the balance of competing causes differ across survivor cohorts of older, nonoperative patients with strict M0 disease and recorded chemotherapy and first-course radiation.

We therefore estimated 3-year OS and the CIFs of SCLC and other-cause death among records surviving to 12, 24, or 36 months after diagnosis. We also calculated the relative contribution of other-cause death and explored associations of age and stage with landmark-specific outcomes. These analyses were descriptive and prognostic: they did not compare treatment strategies or estimate treatment effects. The study is reported in accordance with STROBE [20], with the completed checklist supplied separately.

Methods

Study design, data source, and reporting

This retrospective population-based registry cohort used the Incidence–SEER Research Data, 17 Registries, November 2025 Submission. A SEER tumor record, rather than an identified individual, was the unit of analysis. Because the extract did not include a patient-level identifier, separate records could not be linked across multiple primary tumors.

We included every record that met the prespecified eligibility criteria; no a priori sample-size calculation was used for this descriptive analysis.

The completed STROBE checklist accompanies the manuscript. Analyses were run in the approved local environment, and case-level SEER data were neither uploaded nor redistributed.

Cohort definition

Eligible records were diagnosed during 2010–2017, were from people aged at least 65 years, and had a primary lung or bronchus site with SCLC histology (ICD-O-3 8041–8045). Summary Stage 2000 had to be Localized or Regional.

We applied a year-specific definition of strict M0. Derived AJCC M, 7th edition, had to be M0 for diagnoses in 2010–2015; Derived SEER Combined M had to be c0, p0, M0, or 0 for diagnoses in 2016–2017. We further required no primary-site surgery, chemotherapy coded Yes, Radiation recode coded Beam radiation, usable survival months, and recorded vital status.

Chemotherapy and first-course Beam radiation were eligibility conditions, not comparison exposures. SEER first-course treatment fields do not reliably identify radiation target, dose, fractionation, sequence, or systemic regimen [14, 15]. We therefore refer only to first-course radiation and do not assume that every record represents thoracic irradiation.

Landmark design and outcomes

We defined landmarks (L) at 12, 24, and 36 months after diagnosis. A record entered a landmark risk set if its observed survival time (T) satisfied $T \geq L$. Thus, the sets were nested survivor cohorts rather than independent groups. From each landmark, follow-up continued for no more than 36 months and ended at death, last contact, or administrative truncation.

The OS estimand was the probability of surviving the 36 months after each landmark. Death from any cause was the event. For competing-risk analyses, the first event was coded as SCLC death, death from another cause, or death from an unknown cause; the last category remained a third competing event in the primary analysis.

The competing-risk estimands were the 36-month CIFs of SCLC death and other-cause death. We expressed the other-cause relative contribution as $F_{\text{other}}/(F_{\text{SCLC}} + F_{\text{other}})$, excluding unknown causes from the denominator. This derived proportion was interpreted only alongside the two absolute CIFs.

Survival months were complete. Cause of death was unknown for 32 records. These records remained in the OS analysis and constituted the third event type in the primary competing-risk analysis.

Covariates and quality variables

Prespecified covariates were age at diagnosis, sex, race, diagnosis year, Summary Stage 2000, primary site, and sequence number. Age was modeled continuously per 5 years and grouped as 65–74, 75–84, or at least 85 years for subgroup summaries. Stage was categorized as Localized or Regional.

Microscopic confirmation, completeness of survival dates, and an exact duplicate-profile flag were used for quality checks. The main cohort retained 25 records with identical values across available fields because the extract lacked a unique patient identifier. Microscopic confirmation was not an eligibility requirement and was assessed in sensitivity analysis.

Statistical analysis

We summarized the base cohort and each landmark risk set without baseline P values because the sets were nested. Kaplan–Meier methods estimated conditional 3-year OS; 95% CIs used Greenwood standard errors and a log–log transformation.

Cause-specific CIFs were estimated with the Aalen–Johansen estimator [21]. As a computational check, all-cause survival plus the three CIFs had to equal one within floating-point tolerance. We did not use one minus Kaplan–Meier for cause-specific absolute risk. Gray tests [22] were limited to mutually exclusive age or stage groups within a landmark. For each subgroup variable and cause-specific outcome, reportable Gray-test P values across landmarks were adjusted by the Benjamini–Hochberg method.

Landmark contrasts used 2000 record-level bootstrap samples drawn from the 3422-record base cohort. All three risk sets were rebuilt within every sample, preserving their dependence. We used percentile bootstrap 95% CIs for CIFs, relative contributions, and between-landmark differences; the random seed was 20260904.

Exploratory landmark-specific Cox models examined all-cause mortality, and cause-specific Cox models examined SCLC and other-cause mortality. Fine–Gray models [23] supplied complementary subdistribution hazard ratios for the two known causes. Models adjusted for age, sex, race, diagnosis year, stage, primary site, and sequence number. Treatment variables and surgery were fixed by eligibility and were not modeled.

Schoenfeld residuals were used to assess proportional hazards in Cox models. A stacked landmark OS Cox model used landmark-specific baseline hazards and robust sandwich variances clustered by `local_row_id` to account for repeated inclusion of the same record. Landmark-by-age and landmark-by-stage interactions were exploratory.

Prespecified sensitivity analyses restricted the cohort to microscopically confirmed records, excluded duplicate-profile records, imposed $T > L$, excluded unknown-cause deaths, reclassified unknown causes as other-cause deaths, required complete survival dates, or used a simplified adjustment set. Analyses used R version 4.6.1 with `survival`, `cmprsk`, `boot`, `data.table`, `ggplot2`, and related packages. Scripts, package versions, run logs, and aggregate outputs were archived locally.

Results

Cohort construction and characteristics

Figure 1 summarizes cohort construction. Of 3442 treatment-positive records in the starting export, 20 failed the year-specific strict M0 criterion, leaving 3422 records for analysis.

The 12-, 24-, and 36-month risk sets comprised 2347, 1393, and 983 records. Median age was 72 years in the base cohort and 71 years in each landmark set; 67.0% of base-cohort records were diagnosed at ages 65–74 years. Regional stage accounted for 84.5%, and 99.7% had microscopic confirmation (Table 1).

Conditional overall survival

Three-year OS was 32.7% (95% CI, 30.8%–34.6%) in the L12 risk set, 43.1% (95% CI, 40.4%–45.7%) in L24, and 47.7% (95% CI, 44.6%–50.8%) in L36 (Figure 2 and Table 2).

Bootstrap contrasts were 10.4 percentage points (95% CI, 8.4–12.4) for L24–L12, 4.7 points (95% CI, 2.3–7.3) for L36–L24, and 15.1 points (95% CI, 12.1–18.3) for L36–L12.

Competing causes of death

The 36-month SCLC-death CIF was 56.8% (95% CI, 54.8%–58.7%) in L12, 41.7% (95% CI, 39.1%–44.3%) in L24, and 30.1% (95% CI, 27.3%–33.1%) in L36 (Figure 3 and Table 2).

The L36–L12 SCLC-death difference was –26.7 percentage points (95% CI, –29.8 to –23.8). The other-cause-death CIF was 10.0% (95% CI, 8.7%–11.2%) in L12, 14.7% (95% CI, 12.8%–16.5%) in L24, and 21.3% (95% CI, 18.8%–23.9%) in L36; its L36–L12 difference was 11.4 points (95% CI, 8.8–13.8).

Other causes accounted for 14.9% (95% CI, 13.2%–16.7%) of deaths from known causes in L12, 26.0% (95% CI, 22.9%–29.1%) in L24, and 41.5% (95% CI, 37.1%–45.6%) in L36 (Figure 4 and Table 2). The L36–L12 difference was 26.6 percentage points (95% CI, 22.2–30.7). Unknown-cause death had a 36-month CIF of 0.6% (95% CI, 0.3%–1.0%) in L12; later estimates were suppressed because event cells were sparse.

Subgroup and regression analyses

Age-stratified estimates are shown in Table 3. Conditional 3-year OS in the 65–74-year group was 34.9% in L12 and 50.8% in L36; the corresponding values for ages 75–84 years were 27.5% and 39.0%. Results for those aged at least 85 years were suppressed where event cells were sparse.

For Localized versus Regional stage, the SCLC-death CIF was 44.1% versus 59.3% in L12 and 33.8% versus 43.6% in L24. Descriptive stage differences were less distinct at later landmarks. Table 3 reports the remaining subgroup estimates.

In exploratory adjusted models, each 5-year increase in age was associated with higher other-cause cause-specific mortality: hazard ratios were 1.22 (95% CI, 1.08–1.37), 1.25 (95% CI, 1.10–1.42), and 1.24 (95% CI, 1.09–1.42) in L12, L24, and L36. Fine–Gray estimates had the same direction. The stacked OS

model provided no clear evidence of age-by-landmark ($P=0.389$) or stage-by-landmark ($P=0.071$) interaction. Full model results are in Supplementary Table S1.

Sensitivity analyses

The direction of every reportable primary L36–L12 contrast was retained across prespecified sensitivity analyses. The largest absolute departure from the primary estimand was 1.90 percentage points under the strict $T > L$ rule. Supplementary Table S2 gives the complete estimates and simplified-model results.

Discussion

Among older SEER records meeting strict M0 and treatment criteria, the 3-year prognosis was more favorable in later survivor cohorts. The remaining mortality also differed: SCLC death was less common, whereas other-cause death made up a larger absolute and relative share. These patterns were stable in the prespecified sensitivity analyses.

Conditional survival is intended to update prognosis once a patient reaches a later time point [6, 7]. Previous population-based lung cancer work has also shown that the causes of death among long-term survivors need not mirror those observed soon after diagnosis [8]. In SCLC, published conditional-survival analyses have focused on postoperative patients or cohorts grouped by metastatic pattern [9, 10]. Our analysis addresses a narrower older, nonoperative, strict-M0 population and holds the future observation window constant at 36 months. This is a focused extension of existing evidence, not a new treatment comparison.

The rise in other-cause mortality was not confined to a change in proportions. Its absolute 36-month CIF was also higher in the L36 than in the L12 survivor cohort. A declining SCLC-death risk alone therefore does not account for the full pattern. The registry cannot identify its mechanism; selective survival, differences in underlying health, and measurement error are all plausible contributors.

Older age was consistently associated with other-cause mortality in the landmark-specific models. This supports attention to noncancer risks when discussing prognosis with older survivors, but it does not identify frailty, comorbidity, or a modifiable pathway because those features were not measured adequately in SEER. Nor did the interaction analyses show clear evidence that the age or stage associations varied across landmarks.

Several design choices strengthen the analysis: a year-specific strict M0 definition, a common future window at each landmark, explicit treatment of unknown cause as a competing event, and record-level bootstrap resampling that rebuilt all nested risk sets. Sensitivity analyses examined microscopic confirmation, duplicate profiles, month-boundary classification, cause assignment, survival-date completeness, and model specification.

The findings remain bounded by the registry fields. The analysis used a single secondary registry dataset comprising 3,422 eligible tumor records. The nested risk sets decreased to 2,347, 1,393, and 983 records at the 12-, 24-, and 36-month landmarks, respectively, reducing precision for later-landmark and sparse subgroup estimates.

SEER does not reliably distinguish thoracic, prophylactic cranial, and other radiation targets, and it lacks dose, fractionation, treatment timing, chemotherapy regimen, immunotherapy exposure, toxicity, recurrence, performance status, and detailed comorbidity. Treatment coding and cause-of-death assignment may be imperfect [14, 15]. Because no patient-level identifier was available, dependence among records from multiple primaries could not be assessed. Month-level survival time creates ambiguity at the landmark boundary, although results were similar under $T > L$. Although the prespecified sensitivity analyses assessed the robustness of the findings, no independent external dataset was available to validate or replicate the estimates. Their transportability beyond the SEER population therefore remains uncertain.

Most importantly, each later risk set selected records that had already survived; the landmark contrasts are neither randomized comparisons nor estimates of change within an individual.

The estimates are therefore suited to group-level prognostic updating, not individualized prediction or causal treatment decisions.

Conclusion

Among older SEER tumor records meeting strict M0 and treatment criteria, survival over the next 3 years was higher in later landmark-defined survivor cohorts. SCLC death was less common in those cohorts, while other causes accounted for a larger absolute and relative share of remaining mortality. These estimates update group-level prognosis; they do not measure the effects of chemotherapy or radiation.

Declarations

Author Contributions

Xiaolin Chen: Conceptualization, Methodology, Formal analysis, Visualization, Writing—original draft. Yuyi Lin: Data curation, Validation, Writing—review and editing. Yangchao Xie: Investigation, Methodology, Writing—review and editing. Fenpen Huang: Validation, Visualization, Writing—review and editing. Shaofeng Wang: Conceptualization, Methodology, Supervision, Project administration, Writing—review and editing. All authors read and approved the final manuscript.

Declarations

Reporting guideline

The completed STROBE checklist is provided as Supplementary Information and is cross-referenced to the accompanying author manuscript.

Data availability

The data analysed in this study were obtained from the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program, specifically the SEER*Stat database "Incidence – SEER Research Data, 17 Registries, Nov 2025 Sub (2000–2023)," released in April 2026 and based on the November 2025 submission. Dataset documentation and version details are available at <https://seer.cancer.gov/data-software/documentation/seerstat/nov2025/>. Access to case-level data requires authorization through the SEER Program and acceptance of the SEER Research Data Use Agreement. The authors are not permitted to redistribute case-level data. The present analysis used records diagnosed during 2010–2017. Analysis code and aggregate outputs are not publicly available.

Clinical trial number

Not applicable

Funding

The authors received no specific funding for this work.

Competing interests

The authors declare no competing interests.

Ethics approval and consent to participate

Not applicable

Consent for publication

Not applicable. The manuscript contains no identifiable individual information.

Use of generative AI and AI-assisted technologies

During manuscript preparation, the authors used ChatGPT (OpenAI) to assist with language editing, text organization, and drafting of analysis and figure code. All computations were run locally with prespecified scripts. The authors checked the numerical results, tables, figures, references, and final text against the archived outputs and take full responsibility for the manuscript.

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Tables

Table 1 Characteristics of the strict M0 cohort and nested landmark risk sets

Characteristic	Overall (N=3,422)	L12 (n=2,347)	L24 (n=1,393)	L36 (n=983)
Records, n	3,422	2,347	1,393	983
Age, years, median [IQR]	72 [68-76]	71 [68-76]	71 [68-75]	71 [68-75]
Age group, n (%)				
65-74	2,292 (67.0)	1,648 (70.2)	1,019 (73.2)	731 (74.4)
75-84	1,045 (30.5)	656 (28.0)	354 (25.4)	241 (24.5)
>=85	85 (2.5)	43 (1.8)	20 (1.4)	11 (1.1)
Sex, n (%)				
Female	1,820 (53.2)	1,297 (55.3)	785 (56.4)	565 (57.5)
Male	1,602 (46.8)	1,050 (44.7)	608 (43.6)	418 (42.5)
Race, n (%)				
White	2,994 (87.5)	2,048 (87.3)	1,203 (86.4)	850 (86.5)
Black	257 (7.5)	185 (7.9)	119 (8.5)	83 (8.4)
Other	169 (4.9)	113 (4.8)	70 (5.0)	49 (5.0)
Unknown	<11	<11	<11	<11
Diagnosis period, n (%)				
2010-2013	1,596 (46.6)	1,089 (46.4)	650 (46.7)	453 (46.1)
2014-2017	1,826 (53.4)	1,258 (53.6)	743 (53.3)	530 (53.9)
Summary stage, n (%)				
Localized	529 (15.5)	391 (16.7)	269 (19.3)	204 (20.8)
Regional	2,893 (84.5)	1,956 (83.3)	1,124 (80.7)	779 (79.2)
Primary site, n (%)				
Upper lobe	1,843 (53.9)	1,285 (54.8)	751 (53.9)	523 (53.2)
Middle lobe	195 (5.7)	125 (5.3)	77 (5.5)	56 (5.7)
Lower lobe	787 (23.0)	536 (22.8)	310 (22.3)	224 (22.8)
Main bronchus	270 (7.9)	174 (7.4)	103 (7.4)	72 (7.3)
NOS or overlapping	327 (9.6)	227 (9.7)	152 (10.9)	108 (11.0)

Data are n (%) unless otherwise indicated. Landmark cohorts comprise tumor records alive at 12, 24, or 36 months after diagnosis and are nested, not independent comparison groups. No baseline

significance tests were performed. Values of 1–10 are suppressed as <11. IQR, interquartile range; NOS, not otherwise specified.

Table 2 Conditional 3-year outcomes and contrasts across landmark risk sets

Outcome	L12, % (95% CI)	L24, % (95% CI)	L36, % (95% CI)	L36–L12 difference, percentage points (95% CI)
Conditional 3-year OS	32.7 (30.8-34.6)	43.1 (40.4-45.7)	47.7 (44.6-50.8)	15.1 (12.1 to 18.3)
SCLC death, 3-year CIF	56.8 (54.8-58.7)	41.7 (39.1-44.3)	30.1 (27.3-33.1)	-26.7 (-29.8 to -23.8)
Other-cause death, 3-year CIF	10.0 (8.7-11.2)	14.7 (12.8-16.5)	21.3 (18.8-23.9)	11.4 (8.8 to 13.8)
Other-cause contribution among known causes	14.9 (13.2-16.7)	26.0 (22.9-29.1)	41.5 (37.1-45.6)	26.6 (22.2 to 30.7)

L12, L24, and L36 denote survival to 12, 24, and 36 months from diagnosis. Values are percentages (95% CIs). OS confidence intervals use Greenwood standard errors with a log–log transformation. CIF and contrast confidence intervals use 2,000 record-level bootstrap percentile intervals. The contrast is L36 minus L12 and was calculated from unrounded estimates. Unknown-cause death was retained as a third competing event; sparse later-landmark estimates are reported only in the supplementary material. CIF, cumulative incidence function; OS, overall survival.

Table 3 Selected age- and stage-stratified estimates

Subgroup outcome	Level	L12, % (95% CI)	L24, % (95% CI)	L36, % (95% CI)
Age at diagnosis: Conditional 3-year OS				
	65-74	34.9 (32.6-37.2)	45.2 (42.1-48.3)	50.8 (47.1-54.3)
	75-84	27.5 (24.2-31.0)	37.9 (32.8-42.9)	39.0 (32.9-45.2)
Age at diagnosis: Other-cause death, 3-year CIF				
	65-74	9.2 (7.8-10.6)	13.6 (11.5-15.7)	19.9 (17.0-22.8)
	75-84	11.9 (9.4-14.4)	18.2 (14.2-22.2)	25.5 (20.0-31.0)
Summary stage: SCLC death, 3-year CIF. Gray P (BH-adjusted): L12 <0.001; L24 0.003; L36 0.110				
	Localized	44.1 (39.2-49.0)	33.8 (28.2-39.5)	26.0 (20.0-32.0)
	Regional	59.3 (57.1-61.5)	43.6 (40.7-46.5)	31.2 (27.9-34.4)

Values are percentages (95% CIs). Landmark denominators for ages 65–74 years were 1648, 1019, and 731 at L12, L24, and L36; those for ages 75–84 years were 656, 354, and 241. Denominators for

Localized stage were 391, 269, and 204, and those for Regional stage were 1956, 1124, and 779. Age 85 years or older is omitted here because several event cells were sparse; complete prespecified subgroup results are in Supplementary Table S3. Gray tests were used only for independent subgroups within a landmark, not for OS, and were adjusted by the Benjamini–Hochberg method. CIF, cumulative incidence function; OS, overall survival.

Supplementary Table S1 Landmark-specific Cox, cause-specific Cox, Fine-Gray, and stacked landmark OS Cox models. Supplementary Table S2 Prespecified sensitivity analyses and simplified adjustment models. Supplementary Table S3 Detailed subgroup results. Supplementary Table S4 Histology and data-quality characteristics.

Figures

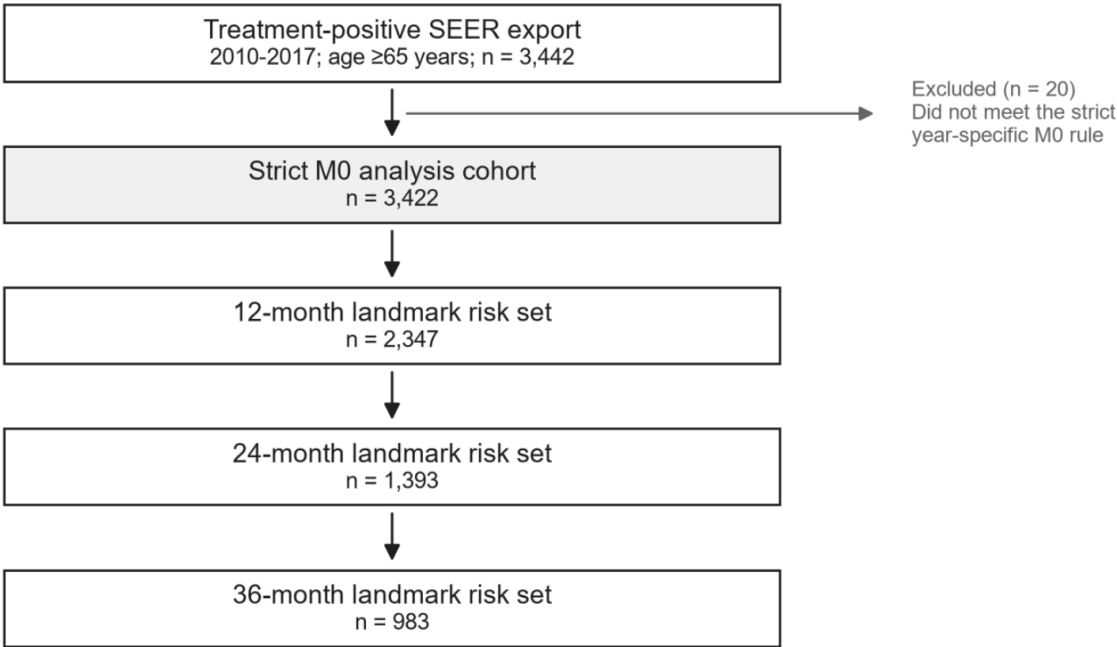


Figure 12

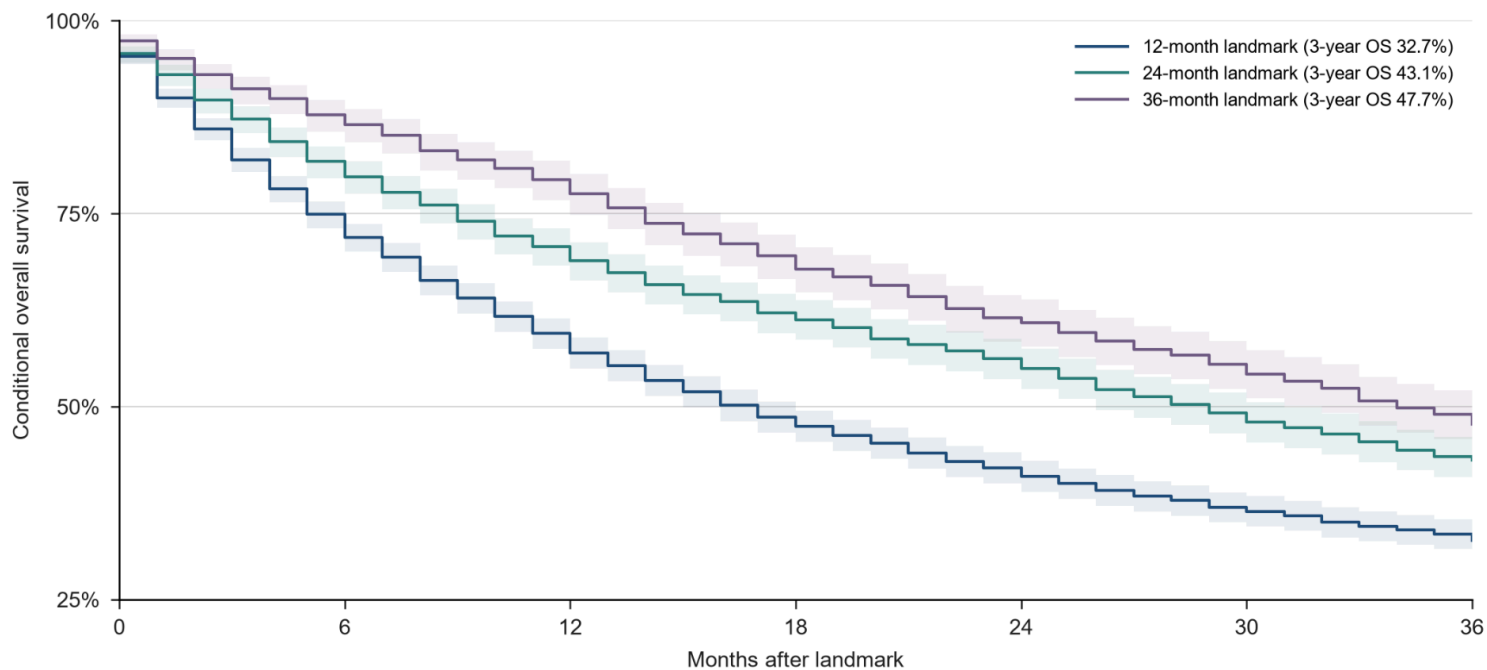


Figure 13

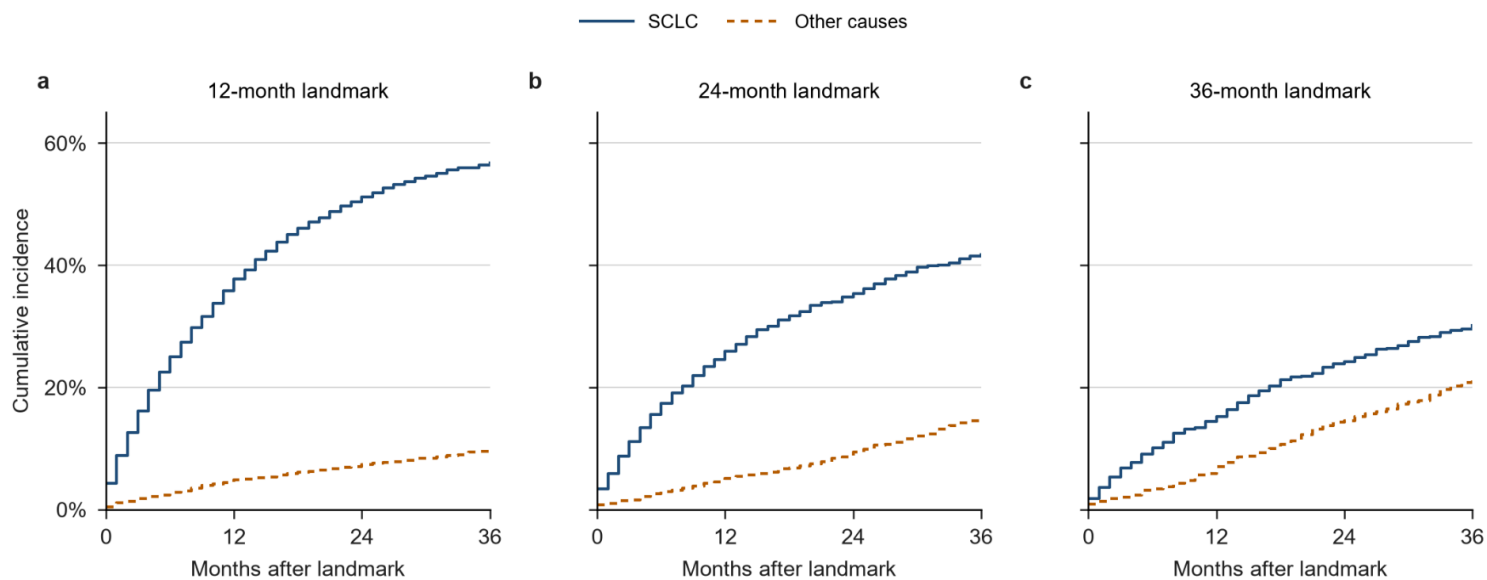


Figure 14

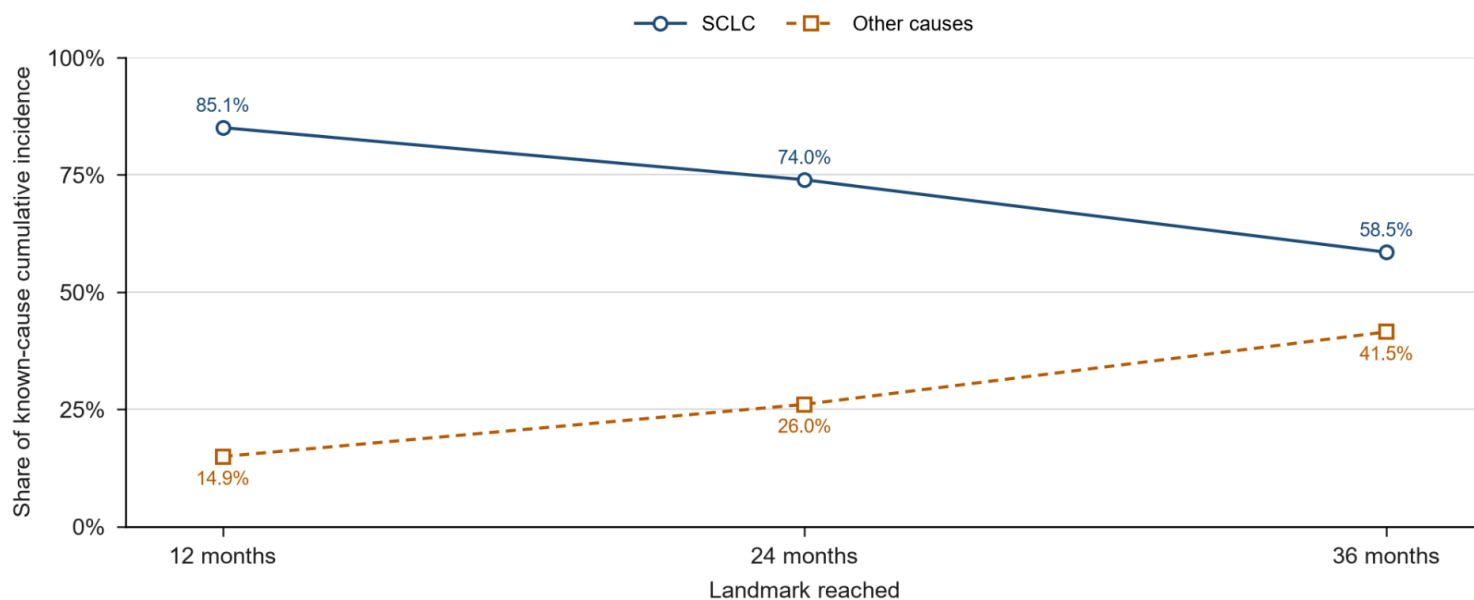


Figure 15

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [04SupplementaryTablesS1S4.docx](#)