

Age-Related Differences in Clinicopathological Characteristics and Overall Survival in Ovarian Serous Carcinoma: A TCGA-OV Cohort Study

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Abstract

Purpose: To evaluate age-related differences in clinicopathological characteristics and overall survival (OS) in ovarian serous carcinoma using The Cancer Genome Atlas ovarian cohort (TCGA-OV).

Methods: We performed a retrospective secondary analysis of publicly available TCGA-OV clinical data. Age was analysed continuously per 10-year increase and secondarily as <65 versus ≥65 years. OS was estimated using Kaplan-Meier methods and compared with the log-rank test. Univariable and multivariable Cox regression adjusted for stage, histologic grade, and residual disease. Proportional-hazards behaviour and sensitivity analyses were assessed.

Results: Among 595 unique primary-tumour records, 584 had age data and 578 formed the survival cohort (346 deaths). Median OS was 49.8 months for patients aged <65 years and 35.8 months for those aged ≥65 years (log-rank $p < 0.001$). Each 10-year age increase was associated with higher mortality in univariable analysis (HR 1.28, 95% CI 1.16-1.41) and after multivariable adjustment (aHR 1.31, 95% CI 1.18-1.46). Stage IV and residual disease remained adverse prognostic factors. The age effect was stronger during the first 3 years of follow-up.

Conclusion: Older age was independently associated with shorter OS in TCGA-OV, although the association varied over time. Age should be interpreted alongside stage, residual disease, and broader patient fitness rather than as an isolated treatment criterion.

Introduction

Ovarian cancer remains a major cause of cancer morbidity and mortality among women worldwide, with outcomes driven by stage at diagnosis, tumour biology, response to systemic therapy, and completeness of cytoreduction [1]. Serous carcinomas account for a substantial proportion of advanced ovarian cancers and have been extensively characterised through large-scale molecular initiatives, particularly The Cancer Genome Atlas (TCGA) [2, 3].

Chronological age is clinically relevant because ovarian cancer incidence rises with age, while older patients often present with different comorbidity profiles, functional reserve, treatment exposure, and surgical outcomes. Multiple cohorts have reported poorer survival in older women, although the independent contribution of age varies after adjustment for stage, performance status, treatment, and residual disease [4-12]. Contemporary reviews therefore caution against interpreting age alone as a surrogate for frailty or treatment eligibility [13].

Age thresholds also differ across studies. A TCGA-supported analysis of serous epithelial ovarian cancer identified 66 years as a prognostically informative cut-off [4], whereas several clinical studies have used 65 or 70 years [5-12]. Other work suggests that age-related survival differences may vary over follow-up rather than remain constant [14]. These inconsistencies support modelling age continuously while retaining clinically interpretable categorical analyses.

The present study used the publicly available TCGA-OV clinical cohort to examine age-related clinicopathological differences and OS. We hypothesised that increasing age at diagnosis would remain associated with higher mortality after adjustment for clinical stage, histologic grade, and residual disease.

Materials and Methods

Study design and data source

This retrospective secondary cohort study used publicly available de-identified clinical data from TCGA-OV, accessed through the UCSC Xena TCGA ovarian clinical matrix (https://tcga.xenahubs.net/download/TCGA.OV.sampleMap/OV_clinicalMatrix). The landmark TCGA ovarian study characterised 489 high-grade serous ovarian adenocarcinomas; the clinical matrix used here contains a broader set of records and therefore was not assumed to represent an identical analytic cohort [2]. Nonblank source histology entries were recorded as 'Serous Cystadenocarcinoma'; the full cohort was consequently described as ovarian serous carcinoma rather than being uniformly relabelled as modern high-grade serous ovarian carcinoma.

Study population and data quality control

The source clinical matrix contained 630 rows. Restriction to records labelled Primary Tumor yielded 595 records representing 595 unique patient identifiers, with no duplicate patient IDs. Age at initial pathological diagnosis was available for 584 patients. Survival eligibility required nonmissing age, an interpretable vital status, and a valid survival time defined from days to death for deceased patients or days to last follow-up for living patients. Records with unresolved survival conflicts or nonnumeric discrepancy values were excluded rather than imputed. The final OS cohort comprised 578 patients. Missing covariate values were retained as missing; no multiple imputation was performed.

Variables and outcomes

Age at diagnosis was the primary exposure and was analysed continuously per 10-year increase. A secondary comparison used <65 versus ≥ 65 years, and ≥ 66 years was examined as a sensitivity threshold because prior serous ovarian cancer work identified 66 years as a prognostically informative cut-off [4]. Clinical stage was grouped as I-II, III, and IV. Histologic grade was grouped as G1-2 and G3-4; GX, GB, and unknown values were not reassigned. Residual disease was grouped as no macroscopic disease, 1-10 mm, and >10 mm. Laterality was examined descriptively. Treatment response and recurrence were not entered into the primary baseline Cox model because they are post-baseline variables.

The primary outcome was OS. Death was the event of interest. Follow-up time was calculated from the available TCGA survival fields and expressed in months using 30.4375 days per month.

Statistical analysis

Continuous variables were summarised using mean and standard deviation or median and interquartile range as appropriate; categorical variables were summarised as counts and percentages using nonmissing denominators. Clinicopathological distributions between age groups were compared using chi-square tests, with exact methods reserved for sparse tables where required. OS was estimated using the Kaplan-Meier method and compared using the log-rank test. Univariable Cox models evaluated age, stage, grade, residual disease, and laterality. The primary multivariable Cox model included continuous age, stage, grade, and residual disease and used complete cases. Hazard ratios (HRs) and 95% confidence intervals (CIs) were reported; two-sided $p < 0.05$ was considered statistically significant.

Proportional-hazards behaviour was screened using Schoenfeld residuals. Because this diagnostic suggested non-proportionality, the standard Cox estimates were interpreted as average relative hazards over follow-up and a 3-year landmark sensitivity analysis was performed. Additional sensitivity models used age cut-offs of 65 and 66 years. Analyses were performed in Python 3.13.5 using pandas 2.2.3, SciPy 1.17.0, and statsmodels 0.14.6.

Ethical considerations

This study used publicly available de-identified TCGA data and involved no direct patient contact or access to identifiable personal information. Ethical oversight and informed consent for the original TCGA data collection were governed by participating institutions and the TCGA programme.

AI-assisted manuscript preparation

Generative AI tools were used only as supportive tools during manuscript preparation for language refinement, structural organization, formatting assistance, and consistency checks. All study data, statistical outputs, references, interpretations, and scientific statements were independently reviewed and verified by the authors using the original TCGA-OV dataset, validated statistical analyses, and primary literature sources. The authors critically revised and approved the final manuscript and take full responsibility for its accuracy, integrity, and scholarly content.

Results

Cohort characteristics

Of 595 unique primary-tumour records, 584 (98.2%) had age data: 387 patients were aged <65 years and 197 were aged ≥65 years. Mean age was 53.0±7.2 years and 72.9±5.5 years in the two groups, respectively. Stage III disease predominated in both groups. Stage, grade, laterality, and residual-disease distributions did not differ significantly at the conventional 0.05 threshold, although residual disease showed a borderline difference (p=0.065). Baseline clinicopathological characteristics are summarised in Table 1.

Table 1. Clinicopathological characteristics according to age group

Variable	Level	<65 years	≥65 years	Global p value
Age, years	Mean ± SD	53.0 ± 7.2	72.9 ± 5.5	Not applicable*
Laterality	Unilateral	94 (25.5%)	58 (31.9%)	0.119
	Bilateral	274 (74.5%)	124 (68.1%)	
Clinical stage	I-II	33 (8.6%)	14 (7.2%)	0.062
	III	283 (73.7%)	160 (82.1%)	
	IV	68 (17.7%)	21 (10.8%)	
Histologic grade	G1-2	53 (14.1%)	22 (11.4%)	0.362
	G3-4	322 (85.9%)	171 (88.6%)	
Residual disease	No macroscopic disease	82 (24.0%)	37 (21.1%)	0.065
	1-10 mm	176 (51.5%)	78 (44.6%)	
	>10 mm	84 (24.6%)	60 (34.3%)	

*Age defined the comparison groups (<65 vs ≥65 years); therefore, a between-group p value for age itself was not calculated. Global p values compare the full distribution of each categorical variable across age groups.

Percentages use nonmissing denominators. Missing/unknown values were excluded variable-by-variable. SD, standard deviation.

Overall survival according to age

The survival-analysis cohort included 578 patients, with 346 deaths and 232 censored observations. Among these patients, 384 were aged <65 years and 194 were aged ≥65 years. Median OS was 49.8 months in the younger group compared with 35.8 months in the older group. Three-year OS was 69.9% versus 49.5%, and 5-year OS was 38.9% versus 25.0%, respectively. Kaplan-Meier curves differed significantly (log-rank $\chi^2=17.12$, $p<0.001$; Fig. 1).

Table 2. Overall survival outcomes according to age group

Age group	N	Deaths	Median OS, months	3-year OS	5-year OS
<65 years	384	211	49.8	69.9%	38.9%
≥65 years	194	135	35.8	49.5%	25.0%

Overall comparison: log-rank $\chi^2 = 17.120$; exact $p = 3.509 \times 10^{-5}$ (<0.001).

Log-rank $\chi^2=17.12$; $p=3.51 \times 10^{-5}$. OS, overall survival.

Cox regression analyses

In univariable analysis, each 10-year increase in age was associated with a 28% higher mortality hazard (HR 1.28, 95% CI 1.16-1.41; $p<0.001$). Stage III, stage IV, and both residual-disease categories were also associated with higher mortality, whereas histologic grade and laterality were not statistically significant. The complete-case multivariable model included 505 patients and 306 deaths. After adjustment, age remained independently associated with mortality (aHR 1.31 per 10-year increase, 95% CI 1.18-1.46; $p<0.001$). Stage IV was associated with higher mortality (aHR 2.55, 95% CI 1.19-5.47; $p=0.016$), as were residual disease of 1-10 mm (aHR 1.92, 95% CI 1.36-2.71; $p<0.001$) and >10 mm (aHR 2.17, 95% CI 1.49-3.16; $p<0.001$). Stage III and grade G3-4 were not independently significant (Table 3; Fig. 2).

Table 3. Univariable and multivariable Cox proportional-hazards analyses

Variable	Univariable HR (95% CI)	p value	Adjusted HR (95% CI)	p value
Age, per 10 years	1.28 (1.16-1.41)	<0.001	1.31 (1.18-1.46)	<0.001
Stage III vs I-II	2.04 (1.14-3.64)	0.016	1.79 (0.87-3.71)	0.116
Stage IV vs I-II	2.74 (1.49-5.07)	0.001	2.55 (1.19-5.47)	0.016
Grade G3-4 vs G1-2	1.22 (0.89-1.68)	0.211	1.21 (0.86-1.70)	0.275
Residual 1-10 mm vs none	2.05 (1.46-2.86)	<0.001	1.92 (1.36-2.71)	<0.001
Residual >10 mm vs none	2.68 (1.87-3.84)	<0.001	2.17 (1.49-3.16)	<0.001

Primary multivariable model: $n=505$; deaths=306. HR, hazard ratio; CI, confidence interval.

Sensitivity and proportional-hazards analyses

Using a categorical age threshold, age ≥ 65 years remained associated with mortality after adjustment (aHR 1.58, 95% CI 1.24-2.00; $p<0.001$). Results were nearly identical using the literature-supported ≥ 66 -year threshold (aHR 1.58, 95% CI 1.24-2.01; $p<0.001$) [4]. Schoenfeld-residual screening suggested non-

proportional hazards for age and several covariates. In the 3-year landmark analysis, the age effect was stronger during the first 3 years (HR 1.48 per 10-year increase, 95% CI 1.27-1.71; $p < 0.001$) and was attenuated among those surviving beyond 3 years (HR 1.14, 95% CI 0.97-1.34; $p = 0.109$).

Discussion

In this secondary analysis of TCGA-OV, older age was associated with shorter OS and remained independently associated with mortality after adjustment for stage, histologic grade, and residual disease. The magnitude of the association was clinically meaningful: the adjusted mortality hazard increased by approximately 31% for each additional decade of age. A categorical ≥ 65 -year analysis produced a similar conclusion, and the result was robust to a ≥ 66 -year threshold previously identified in serous ovarian cancer using a TCGA-supported analysis [4].

These findings are consistent with several clinical and population-based studies reporting poorer survival among older women with ovarian cancer [6, 7, 9-12, 15]. Large cohorts have also documented lower use of standard treatment, lower rates of complete cytoreduction, or persistent survival gaps with advancing age [6-11]. However, the literature is not uniform. Yoshikawa et al. found that age was not independently prognostic after multivariable assessment [5], and Dumas et al. reported attenuation of the age effect after accounting for stage, performance status, and treatment [8]. Such differences are plausible because chronological age captures multiple unmeasured domains, including frailty, comorbidity, competing mortality, treatment intensity, and physiologic reserve [13, 16]. These variables were incompletely represented in the TCGA clinical matrix and therefore could not be fully separated from the observed age effect.

Residual disease was a strong independent prognostic factor in our model. Compared with no macroscopic disease, residual disease of 1-10 mm and >10 mm was associated with approximately 1.9-fold and 2.2-fold higher adjusted mortality hazards. This direction and dose-response pattern are consistent with contemporary meta-analyses showing progressively worse survival with increasing postoperative residual disease [17-19] and with longstanding evidence that maximal cytoreduction is strongly associated with survival in advanced ovarian carcinoma [20]. The finding reinforces the need to interpret age within a broader prognostic context rather than in isolation.

An important feature of the present analysis was evidence of non-proportional hazards. The association between age and mortality was stronger during the first 3 years and no longer statistically significant after the 3-year landmark. A younger-versus-older ovarian cancer study has similarly reported time-dependent differences in the apparent age effect [14]. Accordingly, the standard Cox estimates in this study should be interpreted as average relative hazards over the observed follow-up period, not as proof that the effect of age is constant over time. The early excess risk may reflect treatment tolerance, peri-treatment vulnerability, disease biology, or competing health risks, but these mechanisms cannot be distinguished using the available TCGA variables.

The distinction between chronological age and physiological vulnerability is especially important in ovarian cancer. A meta-analysis of nine cohorts found that frailty was associated with poorer overall and progression-free survival, while the EWOC-1 programme demonstrated that a geriatric vulnerability score stratified mortality risk in older women with advanced ovarian cancer [21, 22]. The G-8 screening tool has also shown prognostic value independent of maximal surgical effort [23]. Population-based data further indicate that age-related survival gaps are most pronounced among women with advanced-stage disease [24]. Together, these observations support interpreting age as a prognostic marker that may partly reflect unmeasured vulnerability rather than as a stand-alone treatment criterion.

Contemporary recommendations similarly emphasise multidimensional assessment of older patients. Position statements on geriatric ovarian-cancer care advocate integration of functional status, comorbidity, nutritional status, cognition, social support, and treatment goals into decision-making [25]. Modern first-line management of advanced high-grade serous ovarian cancer remains centred on cytoreductive surgery and platinum-based systemic therapy, increasingly supplemented by biomarker-directed maintenance strategies [26]. The 2024 ESGO-ESMO-ESP consensus also highlights the need to individualise management of older or frail patients while maintaining oncologic adequacy whenever feasible [27]. These considerations are relevant to the present analysis because TCGA-OV lacks sufficiently granular measures of frailty, treatment intensity, and contemporary maintenance therapy.

The residual-disease association observed here is also supported by higher-level evidence. A Cochrane prognostic-factor review concluded that increasing postoperative residual disease is consistently associated with worse survival after primary surgery for advanced epithelial ovarian cancer [28]. Long-term survival is dynamic rather than fixed: conditional-survival analyses in high-grade serous ovarian cancer show that prognosis improves substantially among patients who remain alive several years after diagnosis [29]. More recent surgical data in women older than 70 years likewise suggest that frailty, rather than chronological age alone, is closely related to resection completeness and long-term outcome [30]. These findings provide a plausible clinical context for the stronger age effect observed during early follow-up in the present study.

The study has several strengths. It used a defined public cohort, patient-level deduplication, explicit survival quality-control rules, continuous modelling of age as the primary exposure, clinically interpretable sensitivity thresholds, and multivariable adjustment for major clinicopathological factors. The analysis also disclosed rather than ignored the proportional-hazards concern and tested the age association across early and later follow-up.

Several limitations require emphasis. This was a retrospective secondary analysis of a historical TCGA cohort and is subject to selection bias and residual confounding. Missingness reduced the multivariable sample from 578 survival-eligible patients to 505 complete cases. Detailed comorbidity, frailty, performance status, treatment intensity, and contemporary maintenance therapy data were unavailable or incomplete. Overall rather than cancer-specific survival was analysed. Source terminology used 'Serous Cystadenocarcinoma', and the full clinical matrix should not be assumed to be identical to the

489 high-grade serous tumours in the original TCGA molecular publication [2]. Finally, the observed associations are prognostic and do not establish that age itself causes poorer survival.

Clinically, these results support incorporating age into prognostic assessment while avoiding age-only treatment decisions. Older patients should be evaluated alongside tumour stage, residual disease, fitness, comorbidity, and treatment feasibility [13, 16]. Future work should validate the time-varying age association in contemporary cohorts with detailed geriatric, treatment, molecular, and cause-specific mortality data.

Conclusion

In TCGA-OV, increasing age was associated with shorter overall survival independently of clinical stage, histologic grade, and residual disease. Residual macroscopic disease and stage IV disease were also adverse prognostic factors. The age association was strongest during the first 3 years of follow-up and attenuated thereafter, indicating that a single proportional-hazards estimate does not fully describe the temporal pattern. These findings support age-informed but multidimensional prognostic assessment in ovarian serous carcinoma.

Declarations

Ethics approval: This study was a secondary analysis of publicly available, de-identified TCGA-OV data and involved no direct participant contact or access to identifiable information. The original TCGA data collection was conducted under institutional ethical oversight and informed-consent procedures.

Consent to participate: No new consent was obtained for this secondary analysis. Consent for the original TCGA data collection was governed by the participating institutions and the TCGA programme.

Consent for publication: Not applicable; no identifiable individual participant material is reported.

Data availability: Clinical data are publicly available through TCGA and the UCSC Xena TCGA ovarian clinical matrix: https://tcga.xenahubs.net/download/TCGA.OV.sampleMap/OV_clinicalMatrix

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Figures

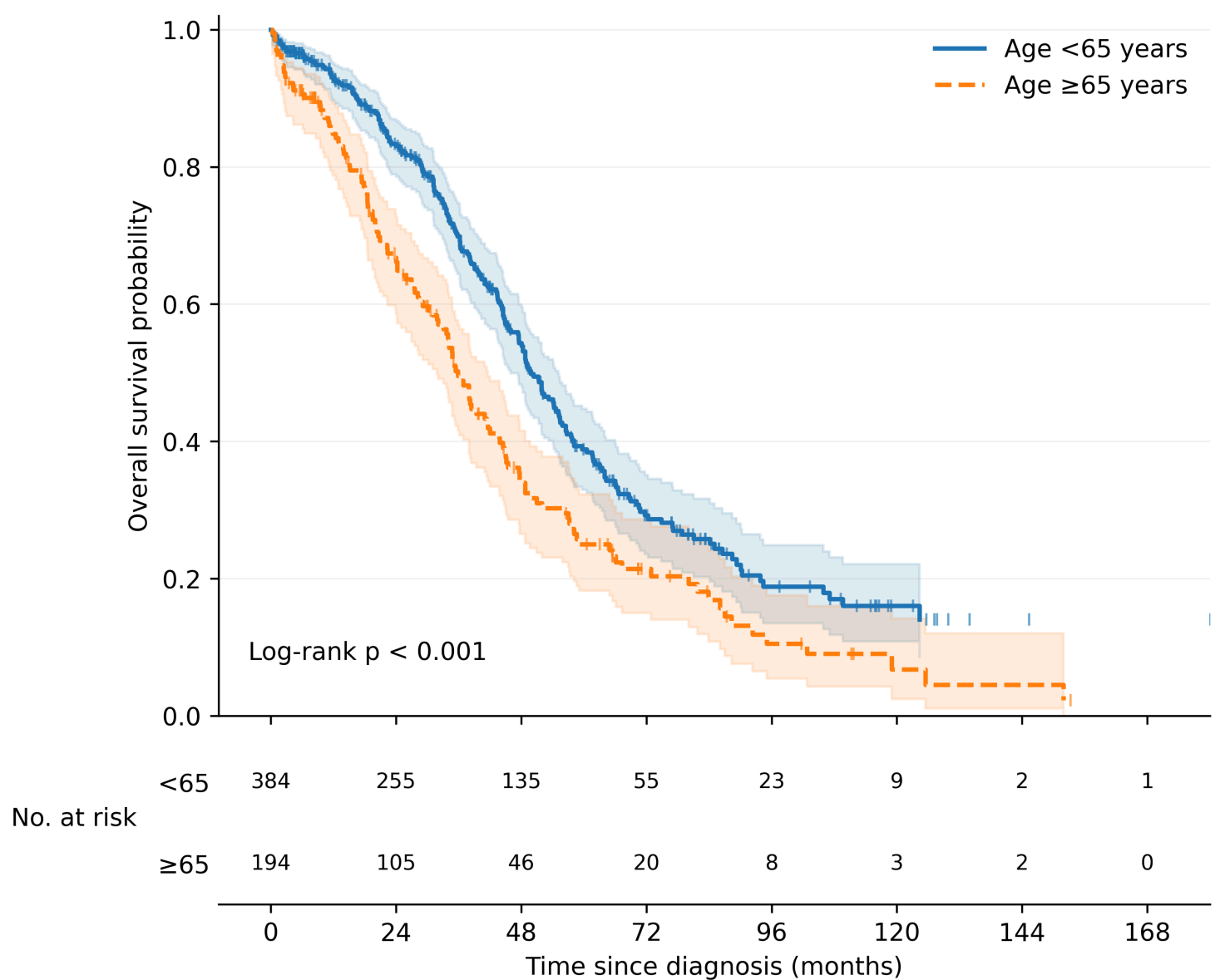


Figure 1

Kaplan-Meier overall survival according to age <65 versus ≥65 years. Shaded areas indicate 95% confidence intervals; censor marks and numbers at risk are shown. The between-group comparison used the log-rank test

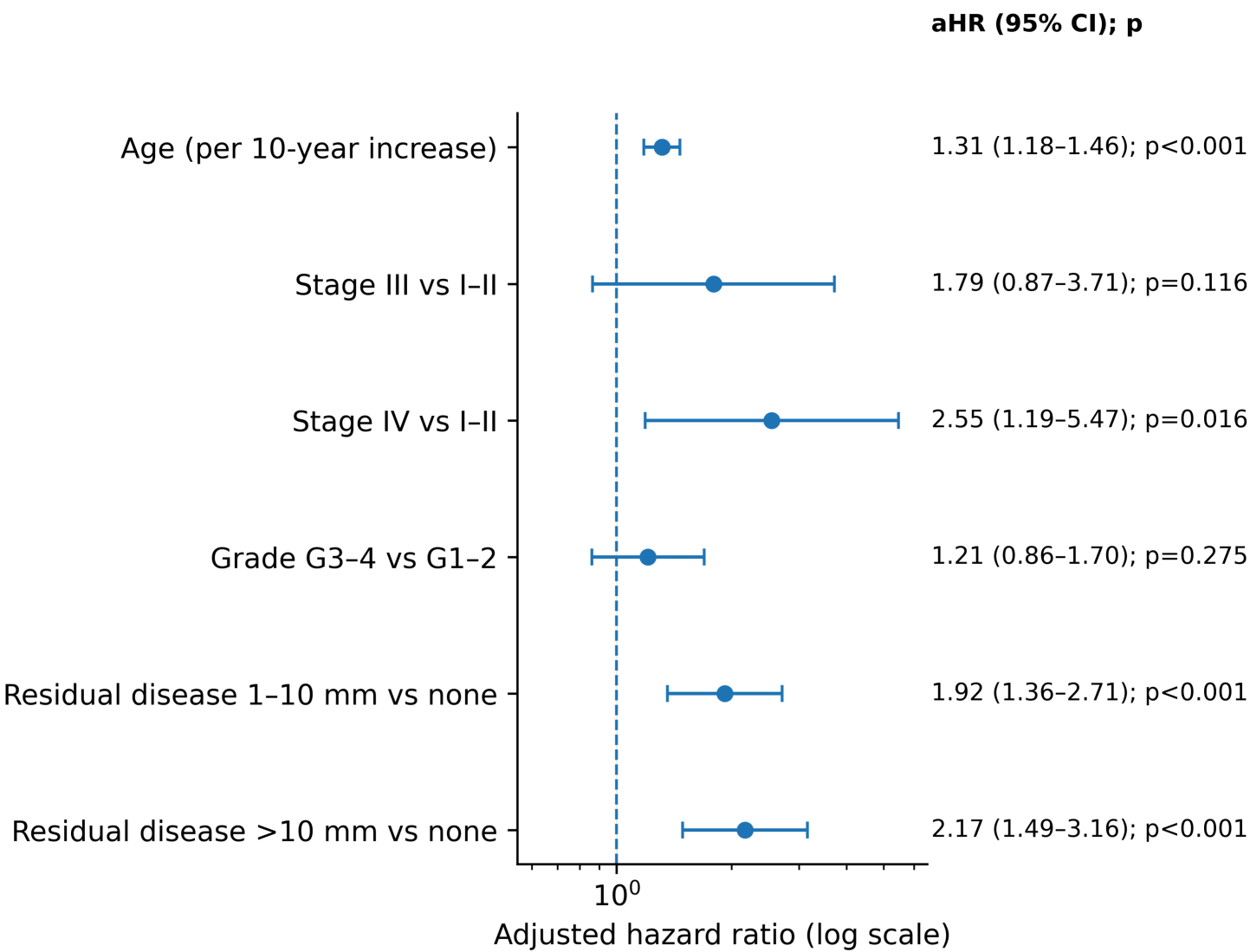


Figure 2

Adjusted Cox proportional-hazards model for overall survival. Points show adjusted hazard ratios (aHRs), horizontal bars show 95% confidence intervals, and the vertical line denotes HR=1