

Pathologic Nodal Upstaging and L858R-Associated Postoperative Outcomes in Resected Clinically Node-Positive EGFR-Mutant Non-Small Cell Lung Cancer

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Abstract

Background: Clinical nodal staging may underestimate mediastinal disease in patients undergoing lung cancer resection, and common sensitizing epidermal growth factor receptor (*EGFR*) mutations may not confer equivalent postoperative outcomes. We evaluated nodal upstaging and subtype-specific outcomes in resected clinically node-positive *EGFR*-mutant non-small cell lung cancer.

Methods: Patients with clinical N1–2, stage II–III *EGFR*-mutant non-small cell lung cancer who underwent resection at four Japanese institutions from 2012 to 2020 were retrospectively studied. *EGFR* exon 19 deletion (Del19) and exon 21 L858R point mutation (L858R) were compared for postoperative overall and progression-free survival. Clinical-to-pathological nodal migration and post-recurrence outcomes were also evaluated.

Results: Among 66 patients, 33 had Del19, 28 had L858R, and five had uncommon mutations. Of 42 patients with clinical N1 disease, 18 (42.9%) were upstaged to pathological N2; overall, 38 patients (57.6%) had pathological N2 disease. L858R showed numerically less favorable overall survival in both clinical and pathological analyses, whereas postoperative progression-free survival was comparable. After recurrence, progression-free survival was shorter with L858R. Brain or meningeal recurrence occurred in 37.5% of L858R cases and 17.9% of Del19 cases.

Conclusions: In resected node-positive *EGFR*-mutant non-small cell lung cancer, L858R showed a less favorable postoperative course than did Del19. Frequent clinical N1-to-pathological N2 upstaging supports careful nodal assessment and the incorporation of mutation subtype and pathological nodal burden into postoperative risk assessment.

Introduction

The epidermal growth factor receptor (*EGFR*) inhibitor osimertinib, when used as an adjuvant, improves disease-free survival and overall survival (OS) after the complete resection of *EGFR*-mutant non-small cell lung cancer (NSCLC) [1, 2]. In neoadjuvant settings, osimertinib has shown activity in resectable disease [3], and its use after chemoradiotherapy has extended genotype-directed treatment across stage III disease [4]. However, data remain limited for patients who undergo surgery for clinically node-positive stage II–III disease, a population in which occult mediastinal involvement can alter postoperative risk.

EGFR exon 19 deletion (Del19) and exon 21 L858R point mutation (L858R) are not biologically interchangeable. In advanced disease, several studies reported greater benefits and longer survival with Del19 than with L858R [5–7]. However, surgical studies produced less consistent results [8, 9].

We examined postoperative survival by *EGFR* subtype in a multicenter cohort of patients with clinical N1–2 and stage II–III disease. We also evaluated clinical-to-pathological nodal upstaging, recurrence patterns, and post-recurrence outcomes.

Patients and Methods

The Medical Ethics Committee of Nara Medical University approved this multicenter retrospective study (CRG25-040; June 23, 2025), and the relevant review boards of the participating institutions approved the protocol. This study conformed to the principles of the Declaration of Helsinki. The requirement for written informed consent was waived or addressed via an institutional opt-out process. No identifiable patient information is reported. The study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations [10].

Patients with *EGFR*-mutant NSCLC, clinical N1–2 disease, and clinical stage II–III disease who underwent lung resection at four Japanese institutions from January 2012 to December 2020 were identified from institutional databases. Of 71 patients, 5 were excluded because of insufficient clinical data ($n = 2$), absence of lung resection ($n = 1$), or unavailable postoperative time-to-event data ($n = 2$). The final cohort comprised 66 patients (Supplementary Fig. S1). Clinical and pathological stages were assigned according to the eighth edition of the TNM classification. *EGFR* mutations were categorized as Del19, L858R, or uncommon mutations. Sex was extracted from medical records as female or male; gender identity and race/ethnicity were not systematically recorded in the source databases and, therefore, were unavailable.

OS was measured from surgery until death from any cause. Progression-free survival (PFS) was measured from the date of surgery to the date of recurrence, progression, or death. Primary comparisons evaluated Del19 versus L858R in the clinical cohort and after pathological classification. Exploratory outcomes included nodal upstaging, recurrence site, first treatment after recurrence, and survival after recurrence. Analyses involving uncommon mutations were exploratory and contextual.

Categorical variables are reported as numbers (%). Survival was estimated using the Kaplan-Meier method and compared using log-rank tests. Cox proportional hazards models were used to estimate hazard ratios (HRs) with 95% confidence intervals (CIs). Multivariable models included age, sex, performance status, smoking history, and *EGFR* subtype; age was modeled per 10-y increase. No adjustments were made for multiple comparisons. The analyses were performed using R version 4.5.2 in RStudio version 2025.09.2 + 418 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Patient characteristics

The cohort had a median age of 69 y (range, 43–86 y), and 28 (42.4%) were male. Among them, 56 (84.8%) had adenocarcinoma, 33 (50.0%) harbored Del19, 28 (42.4%) harbored L858R, and 5 (7.6%) had uncommon mutations (Table 1). Initial management consisted of upfront surgery in 52 patients (78.8%) or preoperative therapy followed by surgery, including chemoradiotherapy in 7, *EGFR* tyrosine kinase inhibitor in 5, and chemotherapy in 2. Lobectomy was performed in 54 patients (81.8%), and complete (R0) resection was achieved in 48 patients (72.7%).

Clinical-to-pathological nodal upstaging

Among 42 patients with clinical N1 disease, 10 (23.8%) were downstaged to pathological N0, 14 (33.3%) remained N1, and 18 (42.9%) were upstaged to N2 (Fig. 1; Table 2). Among 24 patients with clinical N2 disease, 2 (8.3%) were downstaged to N0, 1 (4.2%) to N1, 20 (83.3%) remained N2, and 1 (4.2%) was upstaged to N3. Pathological N2 disease was observed in 38 patients (57.6%). Clinical N1-to-pathological N2 upstaging was not concentrated at a single institution but was observed across the participating institutions.

Survival in the total and clinical common-mutation cohorts

The OS analysis of 65 patients (39 deaths) yielded a median OS of 69.2 mo, and the PFS analysis of all 66 patients (53 events) yielded a median PFS of 16.7 mo (Supplementary Fig. S2). In the common-mutation cohort, L858R showed numerically less favorable OS than did Del19 ($n = 60$; $P = 0.09$), whereas PFS was comparable ($n = 61$; $P = 0.60$; Figs. 2A and B). OS showed a trend toward favoring Del19 in clinical N2, stage III disease ($P = 0.08$), while PFS was numerically more favorable with Del19 ($P = 0.22$; Figs. 2C and D). In clinical N1, stage II disease, OS and PFS curves were largely overlapping between Del19 and L858R ($P = 0.51$ and $P = 0.89$; Figs. 2E and F).

Pathological common-mutation cohort

Analyses based on pathological nodal and stage classification showed a similar direction of effect. In the pathological N1-2, stage II-III common-mutation cohort, L858R showed less favorable OS ($P = 0.08$) and numerically shorter PFS ($P = 0.21$; Fig. 3). For OS, the HR for L858R versus Del19 was 2.05 (95% CI, 0.90–4.64; $P = 0.09$) in univariable analysis and 1.95 (95% CI, 0.85–4.48; $P = 0.12$) in multivariable analysis (Supplementary Table S1). The corresponding PFS HRs were 1.54 (95% CI, 0.78–3.04; $P = 0.21$) and 1.34 (95% CI, 0.66–2.72; $P = 0.42$). Taken together, both clinical- and pathological-stage analyses showed a consistent direction toward less favorable postoperative outcomes with L858R, particularly for OS.

Overall Cox models including uncommon mutations

In age-adjusted multivariable Cox models of the overall cohort (Table 3), L858R showed numerically worse OS than did Del19 (HR, 1.99; 95% CI, 0.97–4.09; $P = 0.06$). Uncommon mutations were associated with worse OS (HR, 5.93; 95% CI, 1.77–19.83; $P = 0.004$). For PFS, *EGFR* subtype was not independently associated with the outcome; the HR was 0.99 (95% CI, 0.54–1.81; $P = 0.96$) for L858R and 1.91 (95% CI, 0.62–5.87; $P = 0.26$) for uncommon mutations. The whole-cohort survival stratified by Del19, L858R, and uncommon mutations is presented in Supplementary Fig. S3.

Recurrence patterns and outcomes after recurrence

Recurrence characteristics were evaluated among 56 patients who developed postoperative recurrence or progression; patients without recurrence were excluded from the denominator. The most common recurrence sites were lung (16/56, 28.6%), lymph nodes (15/56, 26.8%), and brain or meninges (14/56, 25.0%). Brain or meningeal recurrence occurred in 9 of 24 L858R cases (37.5%) and 5 of 28 Del19 cases (17.9%; Supplementary Table S2).

Progressive disease as the best response to first treatment after recurrence was observed in 16 of 24 L858R cases (66.7%) and 10 of 28 Del19 cases (35.7%). OS after first recurrence was comparable between Del19 and L858R ($P = 0.51$), whereas PFS after recurrence was shorter with L858R ($P = 0.02$; Fig. 4).

Discussion

This multicenter retrospective study of resected clinically node-positive *EGFR*-mutant NSCLC yielded three clinically relevant findings. First, clinical-to-pathological nodal upstaging was frequent, particularly from clinical N1 to pathological N2. Second, L858R showed a consistently less favorable direction of postoperative outcome than did Del19 in both clinical- and pathological-stage analyses, especially for OS. Third, L858R was associated with poorer response to first treatment after recurrence and shorter post-recurrence PFS.

The high rate of nodal migration is central to the interpretation of the present results. In this cohort, 42.9% of the patients classified as clinical N1 were upstaged to pathological N2, and pathological N2 disease accounted for 57.6% of the final cohort. This degree of occult mediastinal nodal burden may obscure subtype-specific prognostic signals when patients are analyzed only by clinical stage. The pathological-stage analyses therefore provided a more anatomically precise framework for evaluating postoperative risk and showed a similar direction of effect for L858R compared with Del19. This finding is consistent with prior reports highlighting the risk of nodal upstaging in clinical N1 NSCLC [11–14].

These findings have implications for perioperative surgical oncology. In the current era, adjuvant *EGFR* tyrosine kinase inhibitor studies and emerging neoadjuvant osimertinib strategies have reframed the risk assessment for resectable *EGFR*-mutant NSCLC [1–4, 15]. In patients with suspected nodal involvement, careful preoperative mediastinal staging and adequate intraoperative nodal assessment remain important. The current European Society of Thoracic Surgeons (ESTS) guidelines support invasive mediastinal staging in selected patients with suspected N1 disease or other risk factors for occult mediastinal involvement, and intraoperative ESTS guidelines emphasize systematic nodal assessment for complete resection [16, 17]. Data from lobe-specific or selective dissection studies, which focused mainly on early-stage or selected populations, should not be overgeneralized to clinically node-positive or biologically high-risk cohorts [18–20]. The present study does not support a specific

lymphadenectomy strategy based on *EGFR* subtype, but it reinforces the importance of adequate nodal assessment in node-positive or high-risk patients.

The recurrence data may help explain the less favorable postoperative trajectory observed with L858R, which had a higher proportion of progressive disease as best response and shorter PFS post-recurrence. These findings are consistent with the broader observation that Del19 and L858R differ in treatment sensitivity and resistance biology [5–7, 21].

This study has several limitations. The analysis was retrospective, involved only four institutions, and included a limited number of patients, especially in the pathological subgroup analyses. The L858R findings should be interpreted with caution. In the overall clinical cohort, L858R showed a numerically higher OS hazard than did Del19 after adjustment for age and other covariates, but the association did not reach statistical significance. Thus, although the data suggest that L858R may represent a higher-risk subtype in node-positive resected *EGFR*-mutant NSCLC, they do not establish L858R as a definitive independent prognostic factor. Although uncommon mutations were retained in the overall cohort analyses for completeness, only five patients had uncommon mutations, and these mutations were biologically heterogeneous [22]. Treatment strategies before and after surgery were heterogeneous and changed over time. *EGFR* testing and recurrence-treatment selection were not centrally standardized. Some outcome denominators differed because of missing time-to-event data, and subgroup analyses were exploratory and not adjusted for multiple comparisons.

In summary, in resected node-positive *EGFR*-mutant NSCLC, clinical N1-to-pathological N2 upstaging was frequent and L858R showed a less favorable postoperative and post-recurrence course than did Del19. Mutation subtype and pathological nodal burden may improve postoperative risk assessment in these patients.

Declarations

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Competing interests

The authors declare no conflicts of interest.

Ethics approval

The Medical Ethics Committee of Nara Medical University approved this study (CRG25-040; June 23, 2025), and the relevant review boards of the participating institutions approved the protocol.

Informed consent

The requirement for written informed consent was waived or addressed via an institutional opt-out process for this retrospective observational study.

Data availability statement

De-identified data supporting the findings of this study are available from the corresponding author upon reasonable request and with institutional approval.

Authors' contributions

Conceptualization and methodology: T. Manabe, M. T., F. T., and H. U.

Formal analysis: T. Manabe. Statistical advice: M. O.

Investigation: T. Manabe, M. T., K. T., Y. F., T. H., K. Y., H. M., M. M., M. K., A. T., T. Mizuno, R. M., Y. N., M. H., K. U., T. F. C.-Y., F. T., and H. U.

Writing – original draft: T. Manabe, M. T., and H. U.

Writing – review and editing: all authors.

Supervision: F. T. and H. U.

All authors read and approved the final manuscript.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the authors used OpenAI ChatGPT to assist with language editing and journal format adaptation. The authors reviewed and edited the content as needed and take full responsibility for the submitted manuscript.

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Tables

Tables 1 to 3 are available in the Supplementary Files section.

Figures

Fig. 1

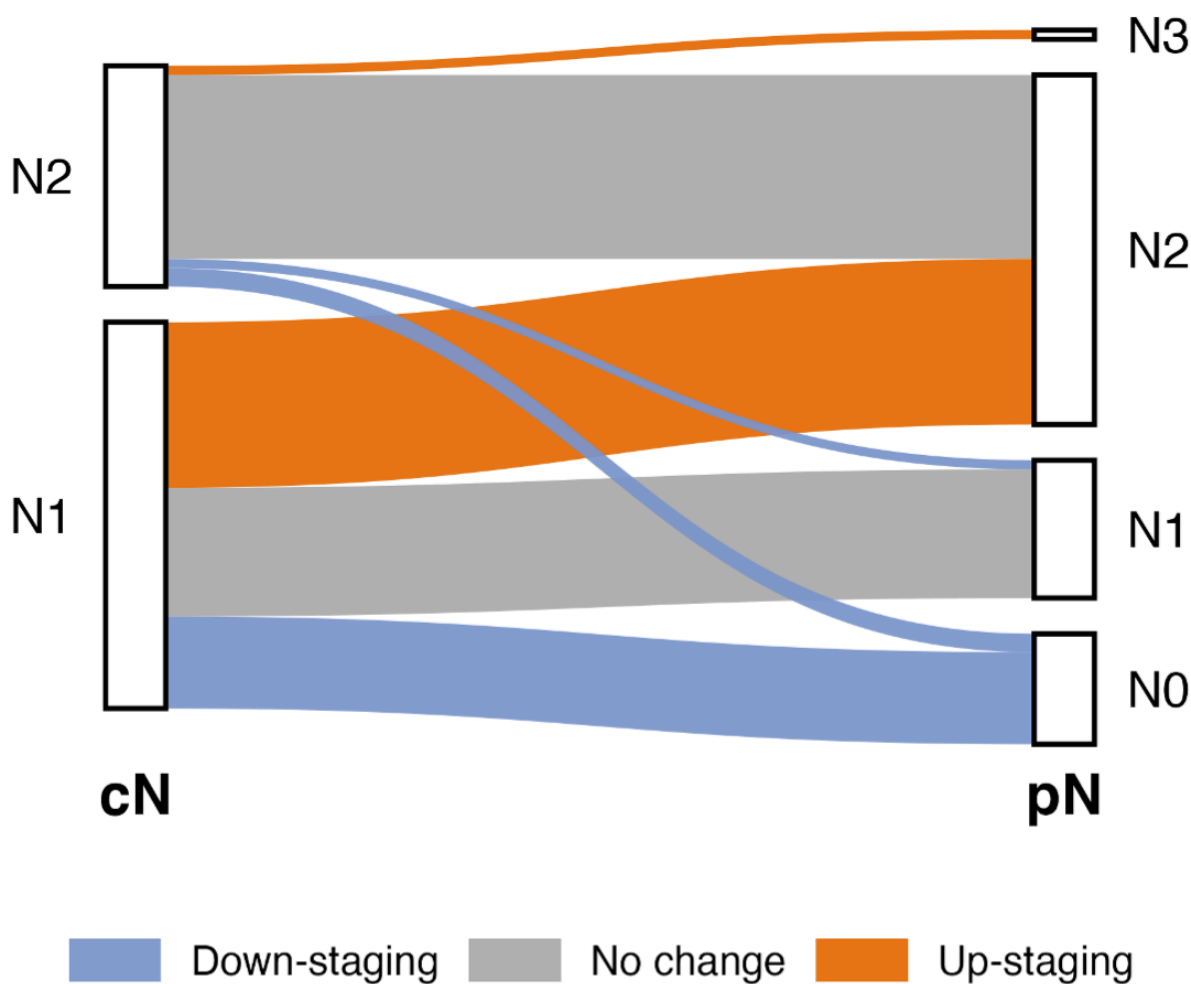


Figure 1

Nodal stage migration from clinical to pathological staging

Sankey diagram showing downstaging, no change, and upstaging from cN1–2 to pN0–3. cN, clinical nodal category; pN, pathological nodal category

Fig. 2

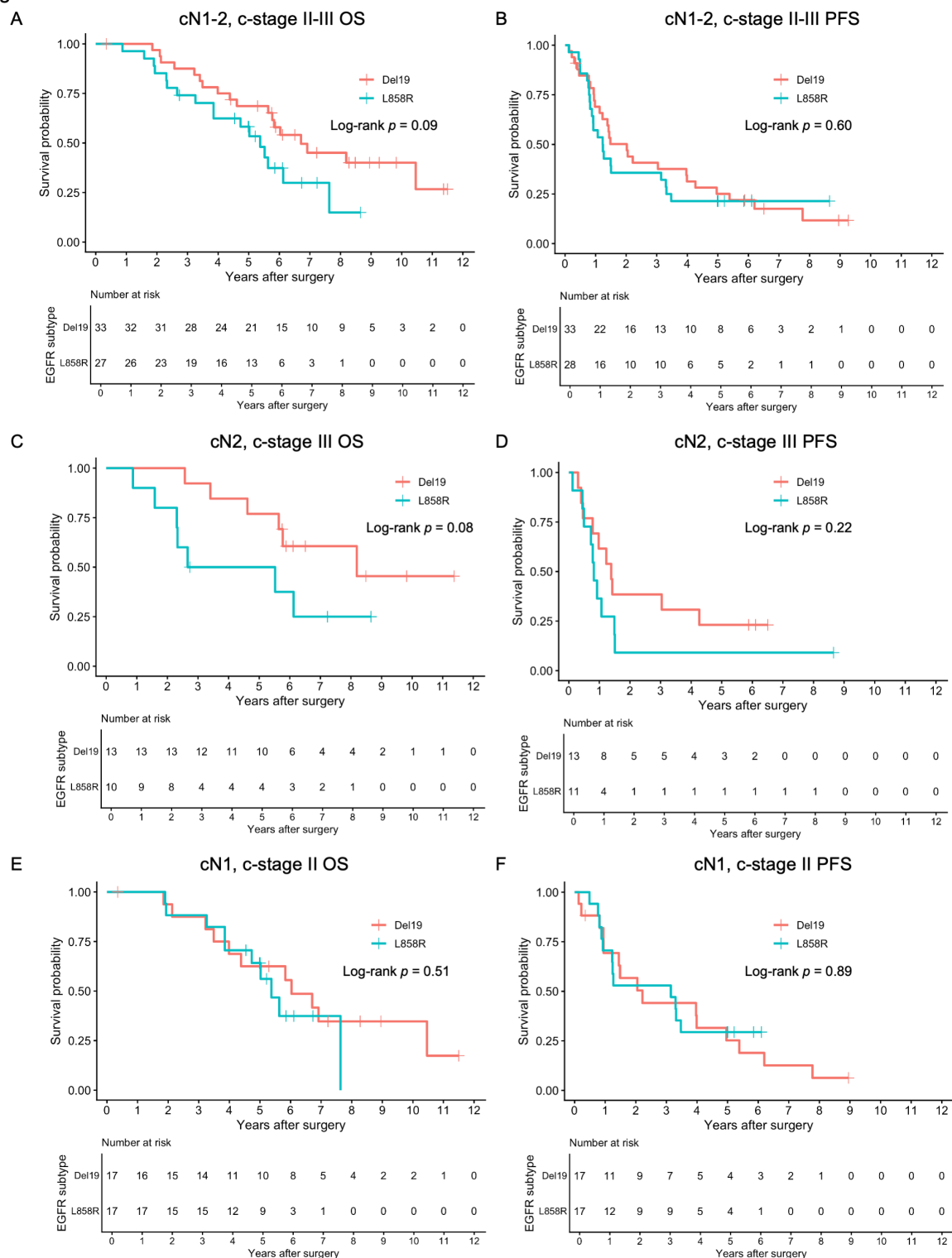


Figure 2

Overall and progression-free survival after surgery by common epidermal growth factor receptor (*EGFR*) mutation subtype in clinical cohorts

(A) Overall survival in cN1–2, clinical stage II–III disease. (B) Progression-free survival in cN1–2, clinical stage II–III disease. (C) Overall survival in cN2, clinical stage III disease. (D) Progression-free survival in

cN2, clinical stage III disease. (E) Overall survival in cN1, clinical stage II disease. (F) Progression-free survival in cN1, clinical stage II disease. *EGFR*, epidermal growth factor receptor; OS, overall survival; PFS, progression-free survival

Fig. 3

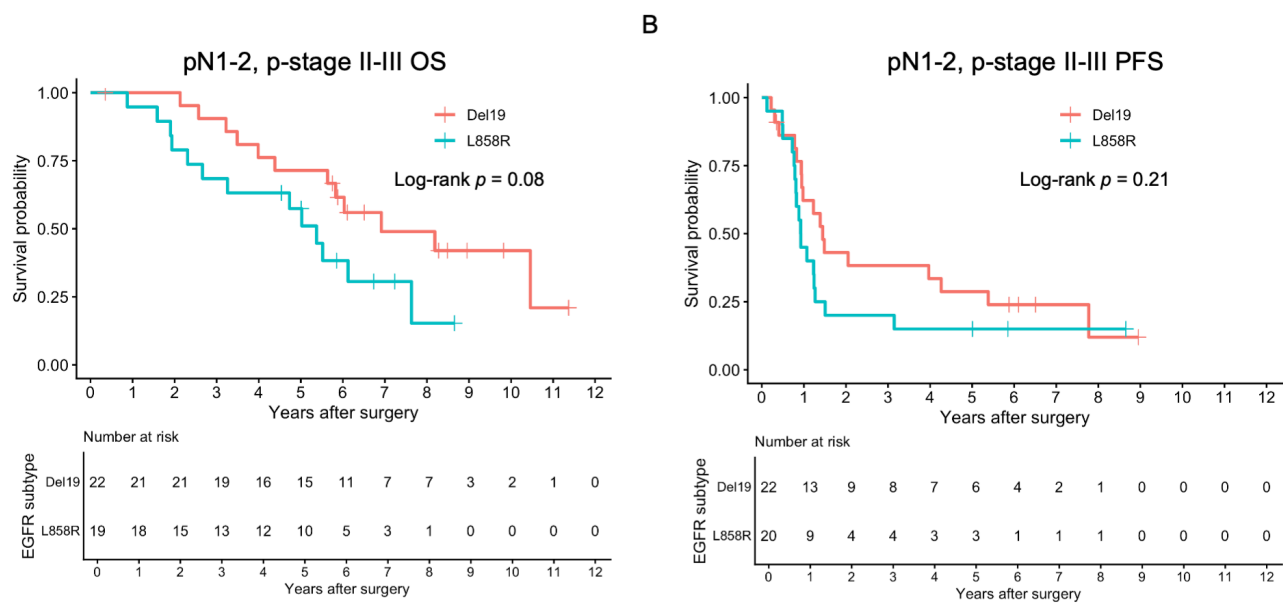


Figure 3

Overall and progression-free survival by common epidermal growth factor receptor (*EGFR*) mutation subtype after pathological classification

(A) Overall survival in pN1–2, pathological stage II–III disease. (B) Progression-free survival in pN1-2, pathological stage II–III disease. *EGFR*, epidermal growth factor receptor; OS, overall survival; PFS, progression-free survival

Fig. 4

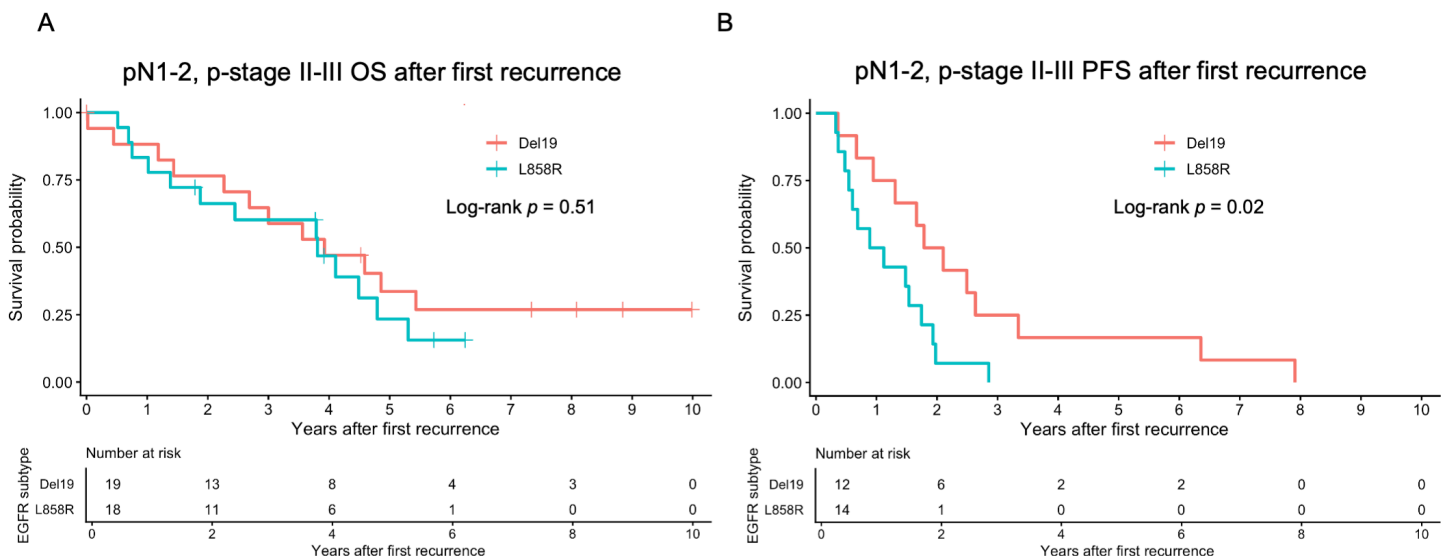


Figure 4

Overall and progression-free survival after first recurrence by common epidermal growth factor receptor (*EGFR*) mutation subtype

(A) Overall survival after first recurrence in pN1–2, pathological stage II–III disease. (B) Progression-free survival after first recurrence in pN1–2, pathological stage II–III disease. *EGFR*, epidermal growth factor receptor; OS, overall survival; PFS, progression-free survival

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