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ORIGINAL ARTICLE

Amivantamab plus lazertinib versus osimertinib in first-line *EGFR*-mutant advanced non-small-cell lung cancer with biomarkers of high-risk disease: a secondary analysis from MARIPOSA

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Background: Amivantamab-lazertinib significantly prolonged progression-free survival (PFS) versus osimertinib in patients with epidermal growth factor receptor (*EGFR*)-mutant advanced non-small-cell lung cancer [NSCLC; hazard ratio (HR) 0.70; $P < 0.001$], including those with a history of brain metastases (HR 0.69). Patients with *TP53* co-mutations, detectable circulating tumor DNA (ctDNA), baseline liver metastases, and those without ctDNA clearance on treatment have poor prognoses. We evaluated outcomes in these high-risk subgroups.

Patients and methods: This analysis included patients with treatment-naive, *EGFR*-mutant advanced NSCLC randomized to amivantamab-lazertinib ($n = 429$) or osimertinib ($n = 429$) in MARIPOSA. Pathogenic alterations were identified by next-generation sequencing (NGS) of baseline blood ctDNA with Guardant360 CDx. Ex19del and L858R ctDNA in blood was analyzed at baseline and cycle 3 day 1 (C3D1) with Biodesix droplet digital polymerase chain reaction (ddPCR).

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Results: Baseline ctDNA for NGS of pathogenic alterations was available for 636 patients (amivantamab-lazertinib, $n = 320$; osimertinib, $n = 316$). Amivantamab-lazertinib improved median PFS (mPFS) versus osimertinib for patients with *TP53* co-mutations {18.2 versus 12.9 months; HR 0.65 [95% confidence interval (CI) 0.48-0.87]; $P = 0.003$} and for patients with wild-type *TP53* [22.1 versus 19.9 months; HR 0.75 (95% CI 0.52-1.07)]. In patients with *EGFR*-mutant, ddPCR-detectable baseline ctDNA, amivantamab-lazertinib significantly prolonged mPFS versus osimertinib [20.3 versus 14.8 months; HR 0.68 (95% CI 0.53-0.86); $P = 0.002$]. Amivantamab-lazertinib significantly improved mPFS versus osimertinib in patients without ctDNA clearance at C3D1 [16.5 versus 9.1 months; HR 0.49 (95% CI 0.27-0.87); $P = 0.015$] and with clearance [24.0 versus 16.5 months; HR 0.64 (95% CI 0.48-0.87); $P = 0.004$]. Amivantamab-lazertinib significantly prolonged mPFS versus osimertinib among randomized patients with [18.2 versus 11.0 months; HR 0.58 (95% CI 0.37-0.91); $P = 0.017$] and without baseline liver metastases [24.0 versus 18.3 months; HR 0.74 (95% CI 0.60-0.91); $P = 0.004$].

Conclusions: Amivantamab-lazertinib effectively overcomes the effect of high-risk features and represents a promising new standard of care for patients with *EGFR*-mutant advanced NSCLC.

Key words: amivantamab, lazertinib, NSCLC, biomarkers, ctDNA, *TP53*

INTRODUCTION

Various characteristics of high-risk disease, including detectable circulating tumor DNA (ctDNA) at baseline and during treatment,[1,2] baseline *TP53* co-mutations,[3] and baseline brain or liver metastases,[4-8] are common in advanced non-small-cell lung cancer (NSCLC). Overall, patients with these clinical features have poorer outcomes compared to those without high-risk features (i.e. those with wild-type *TP53*, ctDNA clearance, absence of baseline brain or liver metastases).[1-8] Because most patients with advanced NSCLC present with at least one high-risk feature, the ideal therapy should be efficacious for patients in these subgroups.

Detectable ctDNA at baseline,[9] *TP53* co-mutations,[10-12] liver metastases,[13,14] and brain metastases[15] are poor prognostic factors for patients with epidermal growth factor receptor (*EGFR)*-mutant advanced NSCLC treated with the third-generation tyrosine kinase inhibitor (TKI) osimertinib. Even patients without high-risk features will develop resistance to third-generation TKIs,[16] emphasizing the importance of using the most efficacious therapy first, particularly for patients with high-risk disease.

Amivantamab is an EGFR-MET bispecific antibody with immune cell—directing activity.[17-20] In clinical studies, amivantamab has shown efficacy alone and in combination with chemotherapy in various treatment settings, including as first-line therapy for patients with *EGFR* exon 20 insertions (Ex20ins) and for patients with *EGFR* exon 19 deletions (Ex19del) or L858R mutations after disease progression on osimertinib.[21-23] Lazertinib is a third-generation EGFR TKI with central nervous system (CNS) activity.[24,25] In the phase III MARIPOSA study (ClinicalTrials.gov identifier, NCT04487080), amivantamab plus lazertinib demonstrated a statistically significant and clinically meaningful improvement in progression-free survival (PFS) versus osimertinib in patients with *EGFR*-mutant advanced NSCLC {median PFS, 23.7 versus 16.6 months; hazard ratio (HR) 0.70 [95% confidence interval (CI) 0.58-0.85]; $P < 0.001$}. Improved durability of response among confirmed responders was also seen for amivantamab-lazertinib versus osimertinib (25.8 versus 16.8 months). Additionally, in patients with a history of brain metastases, amivantamab-lazertinib prolonged PFS versus osimertinib [median PFS 18.3 versus 13.0 months; HR 0.69 (95% CI 0.53-0.92)].[26]

Here, we present efficacy results from other high-risk subgroups, including those with detectable ctDNA at baseline and cycle 3 day 1 (C3D1; i.e. patients without ctDNA clearance at C3D1), *TP53* co-mutations, and baseline liver metastases, in patients from the MARIPOSA study who received amivantamab-lazertinib versus osimertinib.

PATIENTS AND METHODS

Patients

Details of the global, phase III MARIPOSA study (ClinicalTrials.gov identifier, NCT04487080) were previously reported.[26] In brief, eligible treatment-naive adults (18 years of age or older) with Ex19del or Exon 21 L858R-mutated locally advanced or metastatic NSCLC with one or more measurable lesion according to RECIST v1.1 were enrolled. Patients with brain metastases were eligible if asymptomatic before study treatment. A total of 1074 patients were randomized 2 : 2 : 1 to receive amivantamab-lazertinib ($n = 429$), osimertinib ($n = 429$), or lazertinib ($n = 216$). The lazertinib monotherapy arm was included to evaluate the contribution of components in the amivantamab-lazertinib combination arm. The current analysis presents the results in high-risk patient subgroups who received amivantamab-lazertinib or osimertinib. A flow diagram of patient disposition among the high-risk subgroups is shown in Supplementary Figure S1, available at https://doi.org/10.1016/j.annonc.2024.05.541.

The study was conducted in accordance with the provisions of the Declaration of Helsinki, Good Clinical Practice guidelines (as defined by the International Council on Harmonisation), and applicable regulatory requirements, and approved by an independent ethics committee. All patients provided written informed consent.

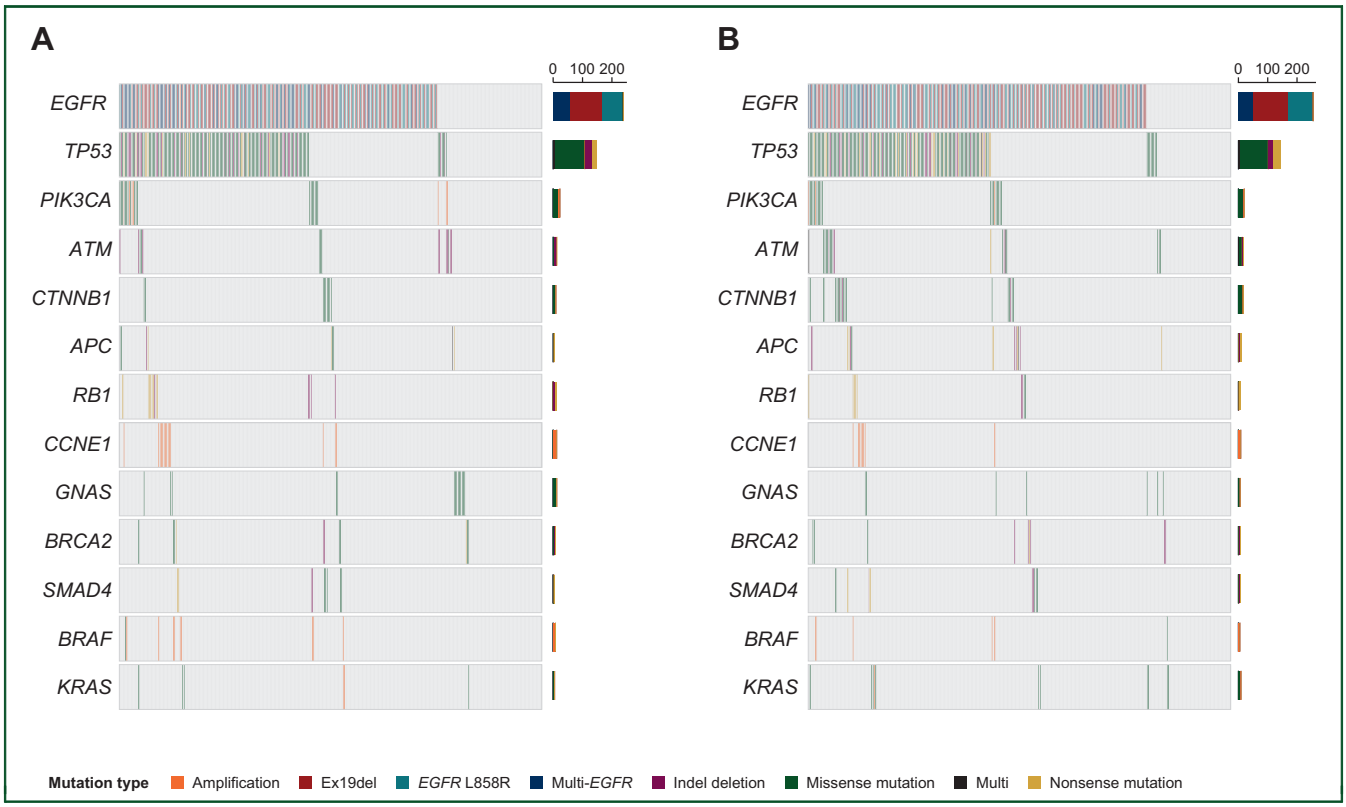

Figure 1. Baseline pathogenic mutation frequency and patterns. Shown here are the mutation variants observed in ≥2% of patients who received amivantamab-lazertinib (A) and osimertinib (B). *EGFR* amplification occurred in 20% of patients with detectable ctDNA at baseline in the amivantamab + lazertinib arm and 19% in the osimertinib arm. *MET* amplification occurred in one patient in each arm (neither had high-level amplification).
ctDNA, circulating tumor DNA; EGFR, epidermal growth factor receptor; Ex19del, exon 19 deletion; multi, multiple.

Sample collection

Patients were required to provide unstained tumor tissue (in quantity sufficient to allow for central analysis of *EGFR* mutation status) and blood [for ctDNA and droplet digital polymerase chain reaction (ddPCR) analysis] samples. When possible, the tumor tissue provided for central analysis was from the same biopsy used for local testing and identification of Ex19del or L858R and was obtained before randomization.

Assessments

Co-mutation status was analyzed using ctDNA by next-generation sequencing (NGS) of blood with Guardant360 CDx (Redwood City, CA) at baseline and within 30 days of progressive disease before next anticancer therapy. These samples were used to assess changes in the levels or types of genetic alterations observed over time and to monitor for the emergence of potential markers of resistance to study therapy. For patients with detectable ctDNA, defined as any identified pathogenic alteration, it was assumed that *TP53* co-mutations would be identified if present. Pathogenic loss-of-function *TP53* co-mutations were annotated using ClinVar (National Library of Medicine, Bethesda, MD), OncoKB (Memorial Sloan Kettering Cancer Center, New York, NY), Cancer Hotspots (Memorial Sloan Kettering Cancer Center, New York, NY), and Human Somatic Mutation Database (Qiagen, Redwood City, CA) and included for analysis in the clinical outcome association.[27-30] Benign variants or variants of unknown significance (VUS) and other functionally unknown *TP53* variants were not included in this analysis, unless otherwise specified.

Detection and clearance of Ex19del and L858R ctDNA in the blood were analyzed with Biodesix ddPCR (Louisville, CO) at baseline and C3D1.

Details of the efficacy assessments have been previously described.[26]

Statistical analysis

This exploratory analysis included all randomized patients who had one or more biomarker assessment. The association of biomarker positivity with clinical response or time-to-event endpoints was analyzed using statistical methods appropriate for each endpoint (e.g. categorical or survival models). Subgroup analyses of efficacy endpoints were carried out using statistical methods for the primary analysis of the general MARIPOSA population, which have been previously published.[26] *P* values for the subgroup analyses are all nominal.

RESULTS

Individual biomarker analysis

ctDNA analysis by Guardant360 NGS. As of the clinical cut-off of 11 August 2023, baseline ctDNA was analyzed by NGS for 636 patients (amivantamab-lazertinib, $n = 320$;

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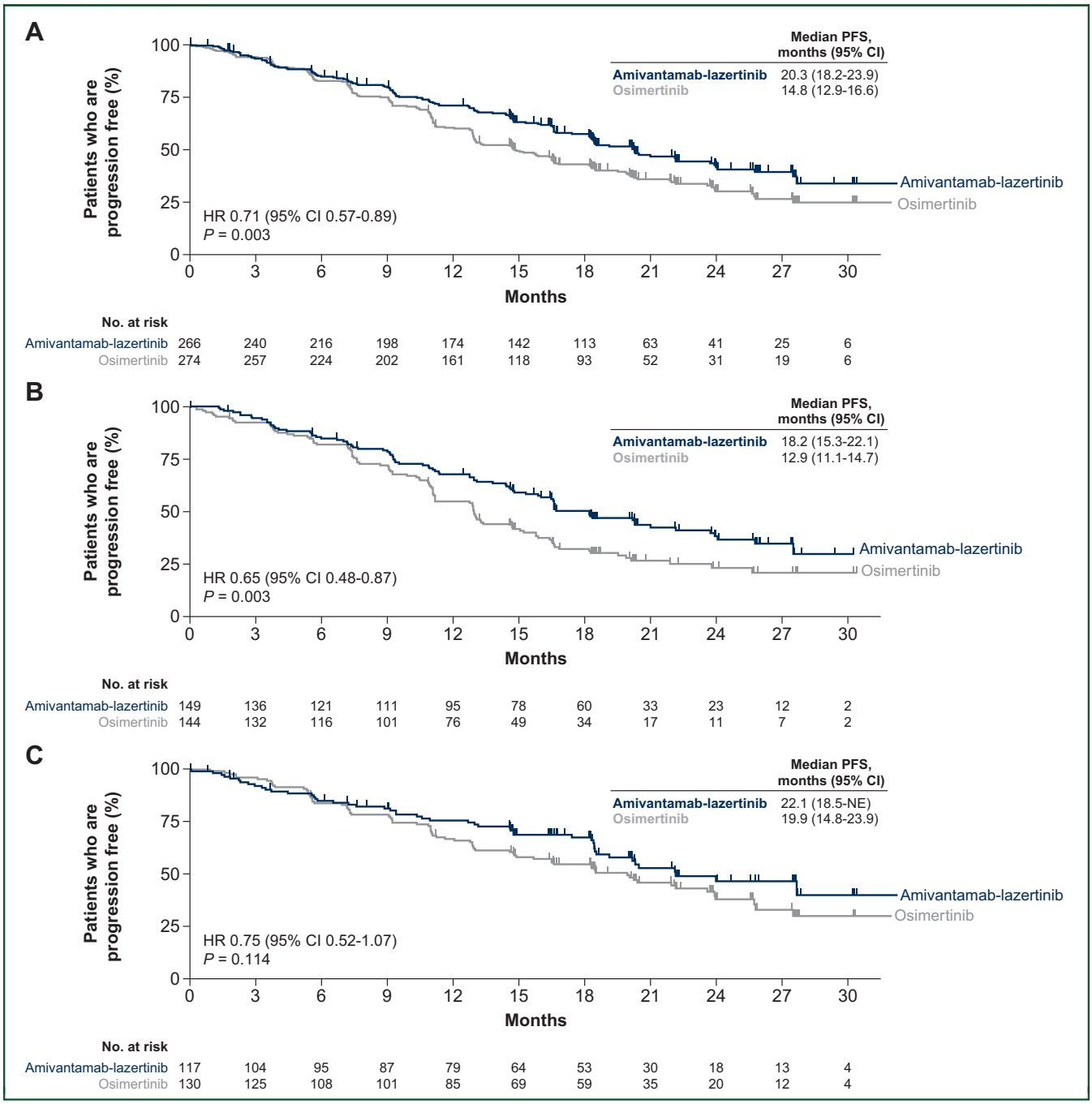

Figure 2. Progression-free survival for patient subgroups identified by NGS. Shown are Kaplan-Meier estimates of progression-free survival for subgroups of patients with detectable baseline ctDNA (A), with *TP53* co-mutations (B), and with wild-type *TP53* (C). Tick marks indicate censoring of data.
CI, confidence interval; ctDNA, circulating tumor DNA; HR, hazard ratio; NGS, next-generation sequencing; PFS, progression-free survival.

osimertinib, $n = 316$). Baseline demographics for the NGS ctDNA analysis population were well balanced (Supplementary Table S1, available at https://doi.org/10.1016/j.annonc.2024.05.541). Among patients with detectable baseline pathogenic alterations by NGS (amivantamab-lazertinib, $n = 266$; osimertinib, $n = 274$), *TP53* co-mutations were detected in 149 patients in the amivantamab-lazertinib arm and 144 patients in the osimertinib arm (Figure 1). Baseline demographics were well balanced between the treatment arms, but more patients with *TP53* co-mutations in both arms had brain and liver metastases at baseline (Supplementary Table S2, available at https://doi.org/10.1016/j.annonc.2024.05.541).

Among the 85% of patients with pathogenic alterations detected in ctDNA at baseline by NGS, PFS was significantly longer for patients receiving amivantamab-lazertinib versus osimertinib, with a median of 20.3 months (95% CI 18.2-23.9 months) and 14.8 months (95% CI 12.9-16.6 months), respectively [HR 0.71 (95% CI 0.57-0.89); $P = 0.003$; Figure 2A; Table 1]. Similarly, when both pathogenic

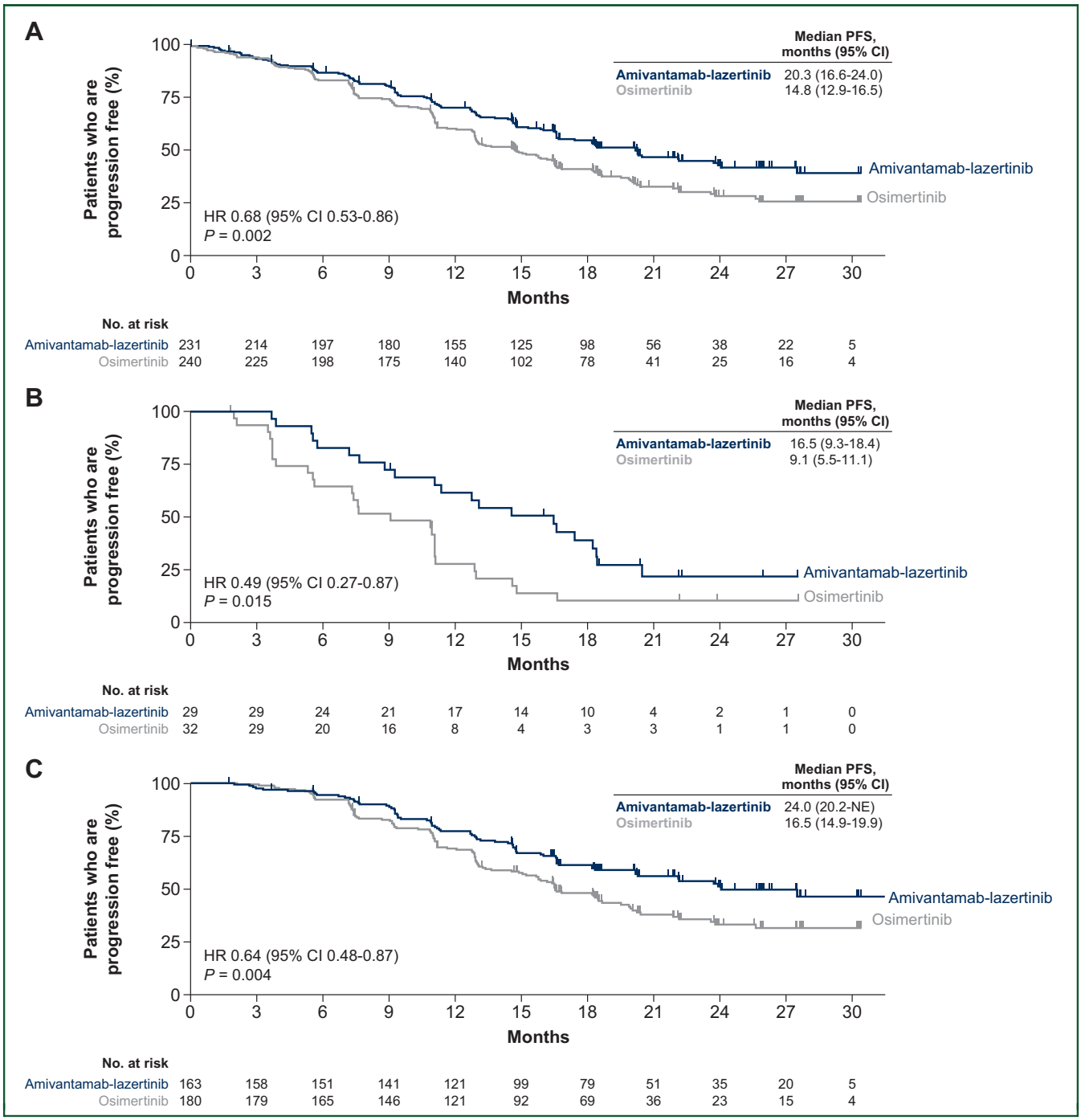

Figure 3. Progression-free survival for patient subgroups identified by ddPCR. Shown are Kaplan-Meier estimates of progression-free survival for subgroups of patients with detectable baseline ctDNA (A), without cleared ctDNA at C3D1 (B), and with cleared ctDNA at C3D1 (C). Tick marks indicate censoring of data.
C, cycle; CI, confidence interval; ctDNA, circulating tumor DNA; D, day; ddPCR, droplet digital polymerase chain reaction; HR, hazard ratio; NE, not estimable; PFS, progression-free survival.

alterations and VUS in ctDNA at baseline were considered, amivantamab-lazertinib significantly prolonged PFS versus osimertinib (Supplementary Figure S2, available at https://doi.org/10.1016/j.annonc.2024.05.541). PFS with amivantamab-lazertinib was numerically longer but did not reach statistical significance ($P < 0.05$) in patients without detectable ctDNA at baseline by NGS (15% of patients; Supplementary Figure S3A, available at https://doi.org/10.1016/j.annonc.2024.05.541).

Among patients with *TP53* co-mutations detected in analyzable baseline ctDNA by NGS (54%), PFS was significantly improved for patients receiving amivantamab-lazertinib versus osimertinib, with a median of 18.2 months (95% CI 15.3-22.1 months) and 12.9 months (95% CI 11.1-14.7 months), respectively [HR 0.65 (95% CI 0.48-0.87); $P = 0.003$; Figure 2B; Table 1]. Among patients with wild-type *TP53* (46%), PFS was numerically longer but did not reach statistical significance ($P < 0.05$) for patients

Table 1. Progression-free survival for biomarker subgroups identified by NGS and ddPCR

Subgroups	Amivantamab-lazertinib, *n*	Osimertinib, *n*	Amivantamab-lazertinib, mPFS (95% CI), months	Osimertinib, mPFS (95% CI), months	HR (95% CI)	*P* value
Detectable baseline ctDNA by NGS	266	274	20.3 (18.2-23.9)	14.8 (12.9-16.6)	0.71 (0.57-0.89)	0.003
TP53 co-mutation	149	144	18.2 (15.3-22.1)	12.9 (11.1-14.7)	0.65 (0.48-0.87)	0.003
TP53 wild-type	117	130	22.1 (18.5-NE)	19.9 (14.8-23.9)	0.75 (0.52-1.07)	0.114
Detectable baseline *EGFR*-mutant ctDNA by ddPCR[a]	231	240	20.3 (16.6-24.0)	14.8 (12.9-16.5)	0.68 (0.53-0.86)	0.002
Not cleared at C3D1	29	32	16.5 (9.3-18.4)	9.1 (5.5-11.1)	0.49 (0.27-0.87)	0.015
Cleared at C3D1	163	180	24.0 (20.2-NE)	16.5 (14.9-19.9)	0.64 (0.48-0.87)	0.004
Liver metastases at baseline						
Present	64	72	18.2 (13.1-NE)	11.0 (7.4-12.8)	0.58 (0.37-0.91)	0.017
Absent	365	357	24.0 (20.3-NE)	18.3 (16.5-20.1)	0.74 (0.60-0.91)	0.004

C, cycle; CI, confidence interval; ctDNA, circulating tumor DNA; D, day; ddPCR, droplet digital polymerase chain reaction; EGFR, epidermal growth factor receptor; HR, hazard ratio; mPFS, median progression-free survival; NE, not estimable; NGS, next-generation sequencing; PFS, progression-free survival.
[a]Of the 231 patients in the amivantamab-lazertinib arm and the 240 patients in the osimertinib arm, 192 and 212 patients, respectively, had matched samples at baseline and C3D1.

receiving amivantamab-lazertinib versus osimertinib, with a median of 22.1 months [95% CI 18.5 months-not estimable (NE)] and 19.9 months (95% CI 14.8-23.9 months), respectively [HR 0.75 (95% CI 0.52-1.07); $P = 0.114$; Figure 2C].

ctDNA analysis by Biodesix ddPCR. At baseline, 471 patients (amivantamab-lazertinib, $n = 231$; osimertinib, $n = 240$) had detectable *EGFR*-mutant ctDNA by ddPCR. Baseline demographics were well balanced between the treatment arms, but more patients with detectable ctDNA at baseline in both arms had brain and liver metastases at baseline versus those without detectable ctDNA at baseline (Supplementary Table S3, available at https://doi.org/10.1016/j.annonc.2024.05.541). Among patients with detectable *EGFR*-mutant ctDNA by ddPCR, amivantamab-lazertinib significantly prolonged PFS versus osimertinib, with a median of 20.3 months (95% CI 16.6-24.0 months) versus 14.8 months (95% CI 12.9-16.5 months), respectively (Figure 3A; Table 1). The HR for disease progression or death was 0.68 (95% CI 0.53-0.86) with a *P* value of 0.002. PFS was numerically longer but did not reach statistical significance with amivantamab-lazertinib for patients without detectable ctDNA at baseline by ddPCR (30% of patients; Supplementary Figure S3B, available at https://doi.org/10.1016/j.annonc.2024.05.541).

In both arms, 85% of patients cleared ctDNA at C3D1. In patients without ctDNA clearance at C3D1, PFS was significantly prolonged with amivantamab-lazertinib versus osimertinib, with a median of 16.5 months (95% CI 9.3-18.4 months) and 9.1 months (95% CI 5.5-11.1 months), respectively [HR 0.49 (95% CI 0.27-0.87); $P = 0.015$; Figure 3B]. Similarly, PFS was also significantly prolonged among patients who cleared ctDNA at C3D1 for amivantamab-lazertinib versus osimertinib, with a median of 24.0 months (95% CI 20.2 months-NE) versus 16.5 months (95% CI 14.9-19.9 months), respectively [HR 0.64 (95% CI 0.48-0.87); $P = 0.004$; Figure 3C].

Liver metastases subgroup analysis

From the intention-to-treat population, a total of 136 patients (16%) had liver metastases at baseline (amivantamab-lazertinib, $n = 64$; osimertinib, $n = 72$). Among these patients, amivantamab-lazertinib significantly prolonged PFS versus osimertinib, with a median of 18.2 months (95% CI 13.1 months-NE) versus 11.0 months (95% CI 7.4-12.8 months), respectively. The HR for disease progression or death was 0.58 (95% CI 0.37-0.91; $P = 0.017$) for amivantamab-lazertinib versus osimertinib (Figure 4A; Table 1). Among patients without baseline liver metastases, amivantamab-lazertinib significantly prolonged PFS versus osimertinib, with a median of 24.0 months (95% CI 20.3 months-NE) versus 18.3 months (95% CI 16.5-20.1 months). The HR for disease progression or death was 0.74 (95% CI 0.60-0.91; $P = 0.004$) for amivantamab-lazertinib versus osimertinib (Figure 4B).

High-risk subgroup analysis

High-risk features at baseline, including detectable ctDNA, *TP53* co-mutations, or metastases in the liver or brain, were identified in 89% of patients with baseline ctDNA available for NGS of pathogenic alterations ($n = 636$). Among patients with any of these high-risk features (amivantamab-lazertinib, $n = 280$; osimertinib, $n = 288$), PFS was significantly longer for patients receiving amivantamab-lazertinib versus osimertinib, with a median of 20.3 months (95% CI 18.2-24.0 months) and 15.0 months (95% CI 13.0-16.8 months), respectively [HR 0.72 (95% CI 0.58-0.90); $P = 0.004$; Figure 5].

In general, overall response and duration of response were numerically higher for amivantamab-lazertinib versus osimertinib across high-risk subgroups (Supplementary Table S4, Supplementary Figures S4 and S5, available at https://doi.org/10.1016/j.annonc.2024.05.541).

DISCUSSION

In the MARIPOSA study, 16% of patients had liver metastases, and 41% of patients had a history of brain metastases at baseline. Among patients with valid ctDNA NGS results, 85% of patients had detectable ctDNA at baseline and 54% had *TP53* co-mutations at baseline. Efficacy outcomes for amivantamab-lazertinib versus osimertinib based on other prognostic variables such as the

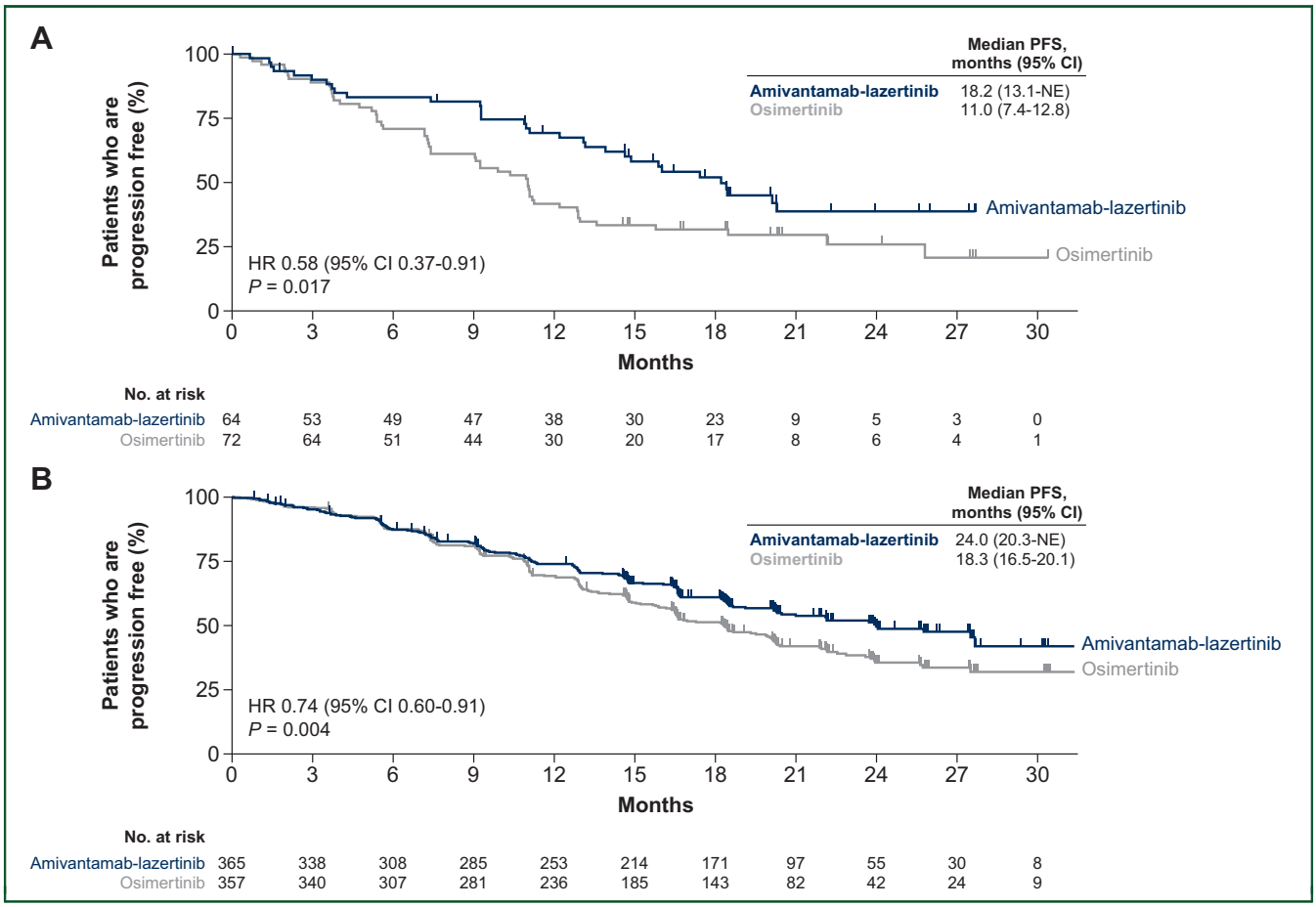

Figure 4. Progression-free survival for patients with and without baseline liver metastases. Shown are Kaplan-Meier estimates of progression-free survival for subgroups of patients with baseline liver metastases (A) and without baseline liver metastases (B). Tick marks indicate censoring of data.
CI, confidence interval; HR, hazard ratio; PFS, progression-free survival.

type of *EGFR* mutation (Ex19del versus L858R) and race (Asian versus non-Asian) have been previously reported.[26] Consistent with the previously reported prevalence of high-risk features in NSCLC, 89% of patients with analyzable ctDNA by NGS in the amivantamab-lazertinib and osimertinib arms from MARIPOSA had at least one of the high-risk features at baseline. These high-risk features are associated with poor outcomes.[3-8]

Liver metastases can affect the metabolism and efficacy of drugs, which underscores the importance of using the most efficacious regimens in patients with liver metastases.[4,6,31] Amivantamab has single-agent activity in patients with liver metastases,[32] which may be due to amivantamab's MET activity with elevated MET expression observed in the liver.[33] We observed improved efficacy versus osimertinib in patients with liver metastases (median 18.2 versus 11.0 months; HR 0.58; $P = 0.017$).

Similarly, brain metastases are a frequent outcome for patients with *EGFR*-mutated NSCLC, and are associated with decreased survival and quality of life.[5,8,34] While the CNS penetrance of third-generation EGFR TKIs, such as osimertinib and lazertinib, is excellent, patients treated with these agents can still experience intracranial disease progression.[35,36] Amivantamab-chemotherapy prolonged intracranial PFS versus chemotherapy alone (12.5 versus 8.3 months, respectively) in the MARIPOSA-2 study, which suggests that amivantamab may provide further intracranial activity. The CNS activity observed may be due to amivantamab's immune cell—directing activity or direct antitumor effects.[21] We observed improved efficacy versus osimertinib in patients with brain metastases [median 18.3 versus 13.0 months; HR 0.69 (95% CI 0.53-0.92)].[26]

TP53 co-mutations are also prevalent, occurring in up to 69% of patients with *EGFR*-mutant advanced NSCLC.[3] *TP53* is one of the most commonly mutated genes in human cancer.[3] The products of this gene play a critical role in tumor suppression, and, therefore, decreased function adds complexity to the signaling cascade typically seen with the constitutive EGFR activation triggered by an *EGFR* mutation.[3,37] As a result, patients with *TP53* co-mutations have significantly worse PFS compared with patients with wild-type *TP53* when treated with EGFR TKIs.[3] Amivantamab-lazertinib significantly improved PFS versus osimertinib in patients with *TP53* co-mutations (HR 0.65; $P = 0.003$).

Detectable ctDNA at baseline and during treatment are poor prognostic factors for patients with *EGFR*-mutant advanced NSCLC.[1,2] However, amivantamab-lazertinib

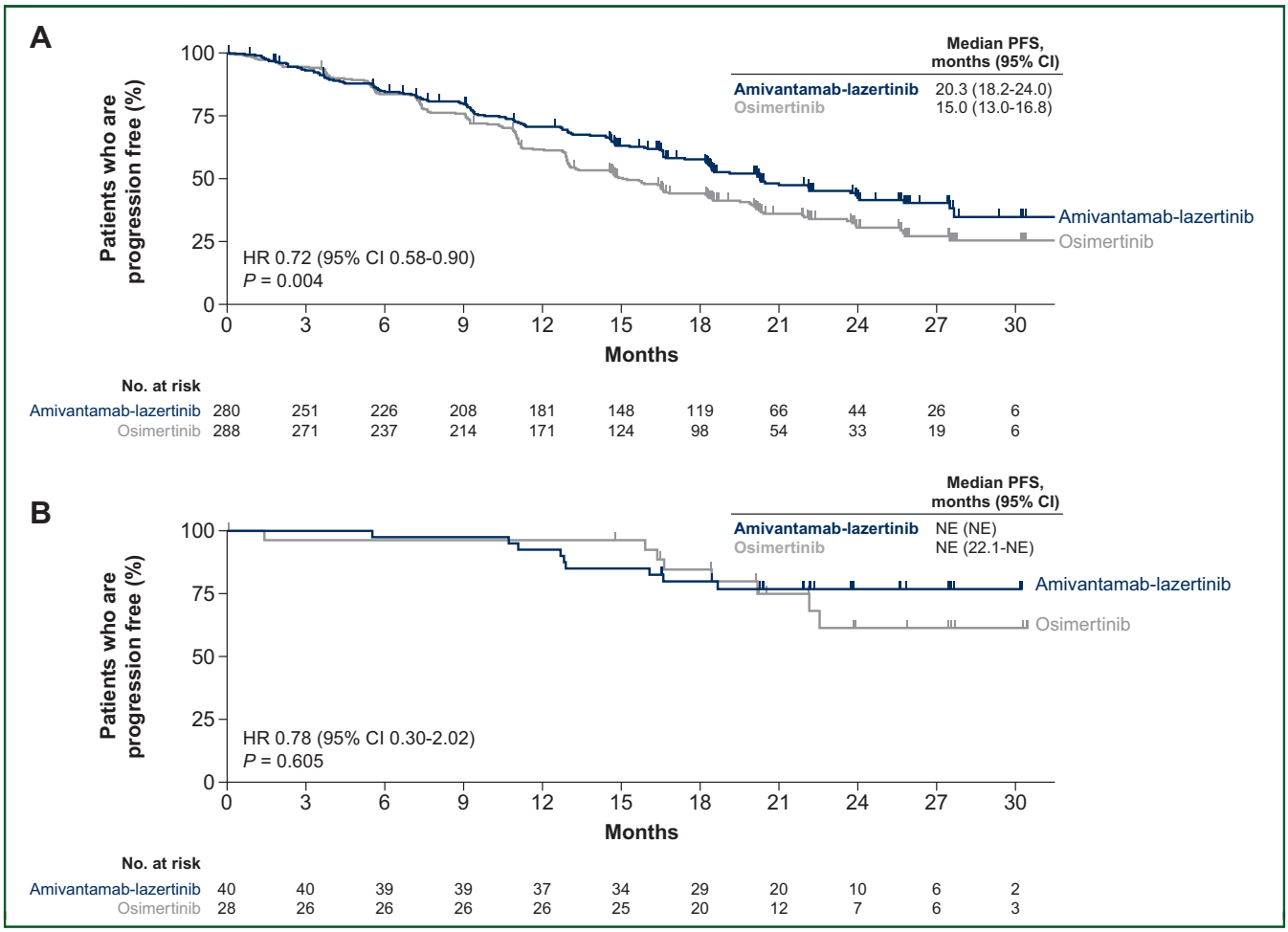

Figure 5. Progression-free survival for patients with and without high-risk features. Shown are Kaplan-Meier estimates of progression-free survival for subgroups of patients with high-risk features (A) and those without high-risk features (B). Patients with analyzable ctDNA at baseline were included in this pooled analysis. High-risk features include baseline detectable ctDNA or baseline metastases of the liver or brain. For patients with detectable ctDNA, it was assumed *TP53* co-mutations would be identified if present. Tick marks indicate censoring of data.
CI, confidence interval; ctDNA, circulating tumor DNA; HR, hazard ratio; PFS, progression-free survival.

significantly prolonged PFS for patients with *EGFR*-mutant ddPCR-detectable baseline ctDNA compared with osimertinib (HR 0.68; $P = 0.002$). Clearance of ctDNA with TKI monotherapy has been associated with improved therapeutic responses, while detectable ctDNA levels on treatment indicates incomplete disease eradication and possible treatment resistance.[38-40] Among patients who did not clear ctDNA from the FLAURA study, TKI monotherapy led to unfavorable outcomes.[40] Data from MARIPOSA showed equal rates of clearance with amivantamab-lazertinib and osimertinib (~85% at C3D1), which was comparable to the clearance rates reported for osimertinib in FLAURA (~82% at week 6).[40] Patients in the osimertinib arm from MARIPOSA who did not clear ctDNA at C3D1 had a severely diminished median PFS of 9.1 months compared to 16.5 months for patients in the amivantamab-lazertinib arm. Patients in both arms without ctDNA clearance at C3D1 experienced the shortest PFS among all high-risk patient subgroups evaluated, indicating that the subgroup without clearance has an aggressive phenotype that remains difficult to fully suppress. However, amivantamab-lazertinib significantly improved PFS for patients without clearance compared with osimertinib (HR 0.49; $P = 0.015$), further supporting the advantage of therapeutic regimens with multiple mechanisms of action in this high-risk subgroup.

In summary, the median PFS for osimertinib in high-risk subgroups (detectable ctDNA at baseline: 14.8 months; detectable ctDNA at C3D1: 9.1 months; *TP53* co-mutation: 12.9 months; liver metastases: 11.0 months) was shorter than that for the overall osimertinib-treated population from MARIPOSA (16.6 months) and FLAURA2 (16.7 months).[41] It was also less than what was observed in the initial phase III FLAURA study, which reported an investigator-assessed median PFS for osimertinib of 18.9 months.[15] The fact that patients with high-risk features have notably shorter PFS than what was seen previously highlights the critical need for new treatment regimens and the importance of giving the most efficacious therapy first. The PFS seen with osimertinib in high-risk subgroups is comparable to what was seen with gefitinib or erlotinib (first-generation EGFR TKIs) in the FLAURA trial.[15]

In conclusion, amivantamab-lazertinib significantly improved PFS versus osimertinib in patients with previously

untreated *EGFR*-mutant NSCLC, including those in high-risk subgroups. Amivantamab-lazertinib effectively overcomes the effect of these negative prognostic features and is thus a promising new standard-of-care option for patients.

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DATA SHARING

The data sharing policy of Janssen Pharmaceutical Companies of Johnson & Johnson is available at https://www.janssen.com/clinical-trials/transparency. As noted on this site, requests for access to the study data can be submitted through the Yale Open Data Access (YODA) Project site at http://yoda.yale.edu.

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