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Research Paper

Real-world efficacy and safety of amivantamab in *EGFR exon-20*-mutant non-small cell lung cancer in a French early-access program: Amexon 20 GFPC study

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ABSTRACT

Background: Amivantamab is a bispecific anti-EGFR–MET antibody approved to treat non-small cell lung cancers (NSCLCs) harbouring *EGFR* exon 20 insertions (*EGFR-exon20ins*).
Methods: We conducted a retrospective, multicentre analysis of consecutive patients with *EGFR-exon20ins* NSCLC treated with ≥ 1 dose of amivantamab in a French early-access programme (09/03/2021–04/30/2022). The primary endpoint was real-world progression-free survival (rwPFS). Secondary endpoints included treatment duration, overall survival (OS), outcomes in patients with brain metastases (BMs), and safety.
Results: Thirty-nine patients were included (median age: 60 years; 64.1 % female, 54 % never-smokers, 33.3 % with ECOG performance status (ECOG-PS) ≥ 2; 66.7 % with BMs at baseline). Amivantamab was administered as second-line therapy in 30 % and third-line or later in 70 %. Patients received a median of 10 doses (range: 1–47)

Abbreviations: AEs, adverse events; BMs, brain metastases; CI, confidence interval; CNS, central nervous system; DCR, disease-control rate; DOR, duration of response; ECOG-PS, Eastern Cooperative Oncology Group performance status; *EGFR*, epidermal growth factor receptor gene; *EGFR-exon20ins*, *EGFR*-exon-20 insertion; HR, hazard ratio; *MET*, mesenchymal-to-epithelial transition gene; NGS, next-generation sequencing; NR, not reached; NSCLC, non-small cell lung cancer; OS, overall survival; PCR, polymerase chain reaction; PD-L1, programmed cell-death protein-1 ligand; PFS, progression-free survival; rw, real-world; TKIs, tyrosine-kinase inhibitors; TP53, tumour protein 53.

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over a median [95 % CI] of 3.4 [1.8–6.3] months. Among 37 evaluable patients, partial responses and disease control were achieved in 35 % [17 %–49 %] and 62 % [44 %–76 %], respectively; median response duration was 5.8 [2.3–11.9] months. In patients with BM, partial response occurred in 23 % and disease control in 69 %. After a median follow-up of 11.3 [8–16.7] months, median rwPFS and OS were 3.5 [2.6–5.8] and 11.3 [8–17.8] months, respectively. Outcomes were 2.8 [3.5–17.8] and 8.7 [3.5–17.8] months in patients with BMs, and 7.6 [1.6–13.5] and 16.2 [8.3–NR] months in those without BMs, respectively. Grade $\geq$ 3 adverse events occurred in 11 patients (28.2 %), mainly skin toxicity (12.8 %) and infusion reactions (5.1 %), leading to dose reductions in 17.9 % and permanent discontinuation in 10.3 %. On multivariate analysis, ECOG-PS $\geq$ 2 was the only negative prognostic factor for both rwPFS and OS.
Conclusion: Amivantamab demonstrated clinical activity in *EGFR-exon20ins*-NSCLC, including in patients with BM, with a manageable safety profile.

1. Introduction

Epidermal growth factor receptor (*EGFR*)–activating mutations play a major oncogenic role in metastatic non-small cell lung cancer (NSCLC); approximately 85 % are *exon 19* deletions or the *exon 21 L858R* substitution [1–3]. In contrast, *EGFR exon 20* insertion (*EGFR-exon20ins*) mutations, which include in-frame insertions and duplications near the C-helix of the *EGFR* kinase domain [4–8], account for only 4–12 % of *EGFR* alterations. More than 90 % these NSCLCs harbouring *EGFR-exon20ins* also confer resistance standard EGFR tyrosine kinase inhibitors (TKIs). Until recently, the standard first-line therapy for these patients was platinum-based chemotherapy [9–14], while no standard second-line treatment had been established. However, real-world (rw) second-line therapies achieved an objective response rate (ORR) of approximately 13 %, with median rw progression-free survival (rwPFS) and overall survival (OS) of, 3.5 and 12.5 months, respectively [9,15].

Amivantamab (JNJ-61186372) is a bispecific anti-EGFR–mesenchymal–epithelial transition (MET) humanised antibody. By binding to the extracellular domain of both EGFR and MET receptors, amivantamab inhibits ligand binding and downstream signalling pathways. It also induces endocytosis and degradation of the antibody–receptor complex, and triggers Fc-dependent trogocytosis by macrophages and cytotoxicity by antibody-dependent natural killer cells [16–18].

In a phase I study, amivantamab demonstrated promising efficacy against *EGFR-exon20ins* NSCLCs [19], with an ORR of 40 % and a median (95 % confidence interval [CI]) duration of response (DOR) of 11.1 months (6.9–not reached [NR]). Median PFS and OS were 8.3 (6.5–10.9) months and 22.8 (14.6–NR) months, respectively. More recently, amivantamab also showed efficacy as first-line therapy in combination with platinum-based chemotherapy [20]. These findings led to its authorisation for this indication in 2021 in the US and, subsequently, its availability in France through an early-access programme between June 2021 and April 2022.

The objective of this study was to evaluate the efficacy and safety of amivantamab in patients with NSCLC harbouring *EGFR-exon20ins*.

2. Methods

2.1. Study design and patients

This retrospective, national, non-interventional study included patients with metastatic *EGFR-exon20ins* NSCLC who were enrolled in an early-access programme for amivantamab. Patients received standard-dose amivantamab infusions (1050 mg, or 1400 mg for those weighing > 80 kg) until disease progression or unacceptable toxicity, unless specified otherwise.

Patient information was collected retrospectively from medical records and included demographics, Eastern Cooperative Oncology Group performance status (ECOG-PS), smoking status, occupational exposure, personal and family medical history, NSCLC characteristics (histology, TNM stage [16], number and location of metastatic sites at diagnosis, as well as details of treatments, their efficacies, and duration. Patients were consecutively enrolled without selection at each centre according to inclusion criteria. Molecular genetic analysis reports from each centre were also collected. Safety was evaluated according to Common Terminology Criteria for Adverse Events; dose reductions and treatment discontinuations due to adverse events (AEs) were recorded.

2.2. Statistical analysis

The primary endpoints were median rwPFS and 1-year OS. Secondary endpoints included time on treatment, treatment beyond progression, response to amivantamab (complete response, partial response, stable disease or progressive disease), rwORR, and rw disease control rate (rwDCR), all assessed according to RECIST 1.1.

Clinicopathological characteristics are presented as numbers (percentages) for qualitative variables and as medians [95 % CI] for quantitative variables; groups were compared using χ^2 tests. The Kaplan–Meier method was used to estimate OS and PFS in the overall population and in predefined subgroups. OS was measured from the start of amivantamab administration to death from any cause, irrespective of subsequent therapy. The log-rank test was applied to compare survival across treatment categories. Treatment response was assessed by local investigators according to RECIST 1.1 in patients who had received at least 15 days of therapy.

Univariate Cox models were used to identify potential prognostic variables for OS and PFS (threshold $p = 0.20$). Multivariable analysis was performed using a backward stepwise Cox regression model, with OS or PFS as the dependent variable and prognostic factors as explanatory variables. Hazard ratios (HRs) with 95 % CIs and p-values were calculated; $p < 0.05$ was considered statistically significant. Statistical analyses were performed using SAS version 9.4 (SAS Institute Inc., Cary, NC, USA).

This non-interventional study was conducted in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines. It was approved by a national ethics committee, the French Advisory Committee on Information Processing in Medical Research in the Field of Health, and the French National Data Protection Authority (CNIL), in compliance with the General Data Protection Regulation. All participating departments approved the study protocol. All surviving patients were informed by their referring physicians and given the option to decline participation.

3. Results

From June 2021 to April 2022, 39 patients were enrolled across 28 participating centres. The median age was 60 years; 64.1 % were female, and 53.8 % were never-smokers (Table 1). ECOG-PS was $\geq$ 2 in 33.3 % of patients, and 66.7 % had brain metastases (BMs) at amivantamab initiation. Most tumours (89.7 %) were lung adenocarcinomas. Molecular analyses were performed on tissue samples in all but one patient, for whom plasma liquid biopsy was used. Next-generation sequencing (NGS) was used in 32/39 (82.1 %) patients, and polymerase chain reaction (PCR) in 7/39 (17.9 %). *EGFR-exon20ins* mutations were located in the C-helix (2/39, 5.1 %), far loop (18/39, 46.2 %), or near loop (19/

Table 1
Characteristics of the 39 NSCLC patients with *EGFR-exon20* deletions or insertions enrolled in an early access program.

Characteristic	Value*
Age at metastatic disease diagnosis Median (range), years	60 (36–83)
Female sex	25 (64.1 %)
Smoking status	
Smoker (current or stopped < 1 yr)	4 (10.3 %)
Former-smoker (stopped > 1 yr)	14 (35.9 %)
Non-smoker (<100 cigarettes/lifetime)	21 (53.8 %)
Stage at diagnosis, n = 36	
I/II	2 (5.6 %)
III	2 (5.6)%
IV	32 (88.8 %)
Histology	
Adenocarcinoma	35 (89.7 %)
Others	4 (10.3 %)
PD-L1 status, n = 34	
<1%	17 (43.6 %)
1–49 %	13 (33.3 %)
>50 %	4 (10.3 %)
Other concomitant anomalies†, n = 39	
Exon19	1 (2.6 %)
EGFR amplification	3 (7.7 %)
TP53	7 (18 %)
PIK3CA	1 (2.6 %)
MET amplification	2 (5.1 %)
CTNNB1	1

* Results are expressed as number (%) unless states otherwise.
† Gene names: *TP53*, tumor protein 53; *PIK3CA*, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha; *MET*, mesenchymal-to-epithelial transition; *CTNNB1*, β-catenin.

39, 48.7 %). Concomitant molecular abnormalities were identified in 15/39 (38.5 %) patients, most frequently tumour protein 53 (TP53) alterations (n = 7) or MET amplification (n = 2).

Programmed death-ligand 1 (PD-L1) status was available for 34/39 (87.2 %) patients and was negative, 1–49 %, or ≥ 50 % in 43.6 %, 33.3 % or 10.5 %, respectively (Table 1). At inclusion, 64 % of patients had ≥ 3 metastatic sites, most commonly brain (66 %), lung (54 %) and bone (64 %). Of the 26 patients with BMs at amivantamab initiation, 10 (38.5 %) had previously received cerebral radiotherapy (specific modalities not available). First-line therapy consisted of platinum-based chemotherapy in 35/39 (89.7 %) patients, while 4/39 (10.3 %) received an EGFR-TKI.

Amivantamab was prescribed as second-line treatment in 30 % of patients and as third-line or later in 70 %. The median interval between diagnosis and initiation of amivantamab was 18.3 months (range: 11.4–24.1). Patients received a median of 10 infusions (range:1–47). The median duration on amivantamab was 3.4 months [95 % CI, 1.8–6.3]. At data cut-off (12 March 2024), 34/39 (87.2 %) patients had progressive disease, 2/39 (5.1 %) remained on treatment, and 10/39 (25.6 %) were alive.

After a median follow-up of 11.3 months [95 % 8–16.7], the median rwPFS and OS were 3.5 months [2.6–5.8] and 11.3 months [8–17.8], respectively. Six-month rwPFS was 33.3 %. While the 12- and 24-month OS rates were 48.7 % and 25.2 %, respectively (Fig. 1**A and B**). Among 37/39 (94.9 %) patients evaluable for response, rwORR was 35 % [17 %–49 %], rwDCR was 62 % [44 %–76 %] and median DOR was 5.8 months [2.3–12.2].

In the subgroup of 26/39 (66.7 %) patients with BMs at amivantamab initiation, median rwPFS and OS were 2.8 months [3.5–17.8] and 8.7 months [3.5–17.8], respectively. For the 13/39 (33.3 %) without BMs, median rwPFS and OS lasted 7.6 months [1.6–13.5] and 16.2 months [8.3–NR], respectively. OS did not differ significantly between the two groups. Univariate and multivariable analyses identified ECOG-PS ≥ 2 as the only factor independently and significantly and associated with poorer PFS and OS (Table 2).

Among 23/26 (88.5 %) patients evaluable for central nervous system (CNS) response, 23.1 % achieved an objective response, and 69 % had

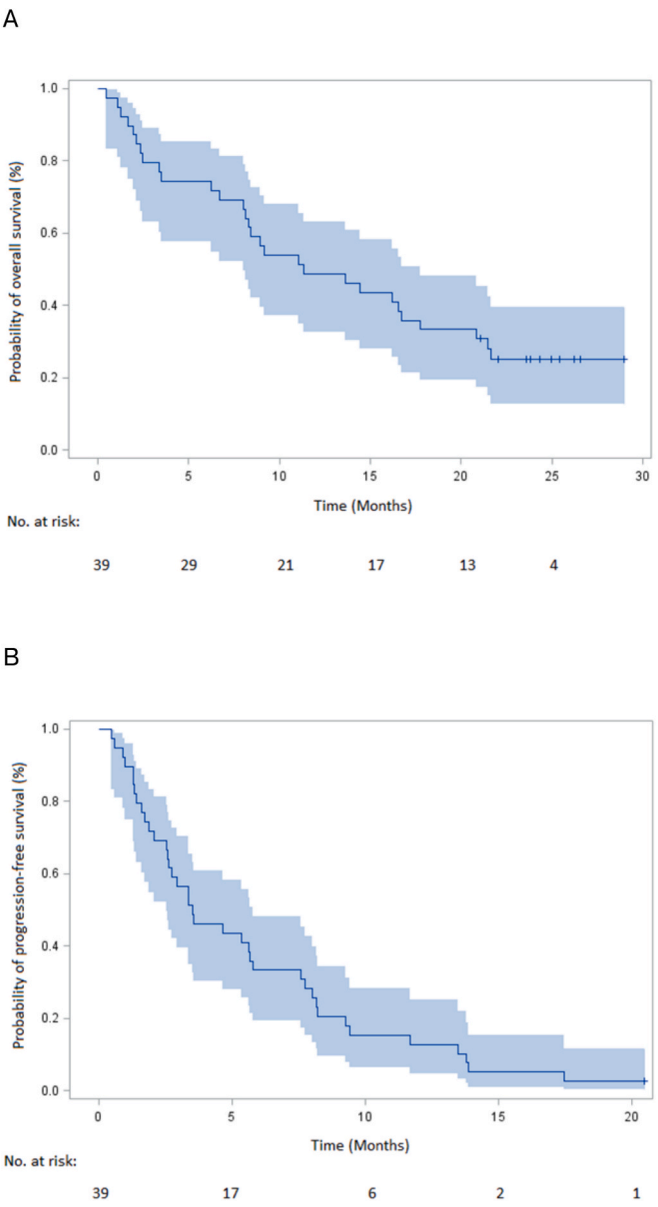

Fig. 1. (A) Overall survival (OS) and (B) real-world progression-free survival (rwPFS) curves with shaded 95% confidence intervals for 39 patients with *EGFR exon 20 insertion*–positive NSCLC. Fig. 1 (A) OS and (B) rwPFS curves with shaded 95 confidence intervals for the entire population.

BM control. The median rwDOR was 5.8 months (range: 2.3–11.9).

Following progression after amivantamab, 21/39 (53.8 %) patients received subsequent therapy, most commonly single-agent chemotherapy (13/21, 61.9 %).

Grade ≥ 3 AEs occurred in 11/39 (28.2 %) patients, including skin toxicity (5/39, 12.8 %), infusion-related reactions (2/39, 5.1 %), digestive disorders (1/39, 2.6 %), interstitial pneumonia (1/39, 2.6 %), neurological disorders (1/39; 2.6 %) and fever (1/39, 2.6 %). AEs led to dose reductions in 7/39 (17.9 %) patients and permanent discontinuation in 4/39 (10.3 %) (Table 3).

4. Discussion

For this heavily pretreated and poor-prognosis cohort (66.7 % with BMs and 33.3 % with ECOG-PS ≥ 2), patients with *EGFR-exon20ins* NSCLC treated with amivantamab achieved a median rwPFS of 3.5 months and median OS of 11.3 months, with an rwORR of 35 % and an

Table 2
Univariate and multivariate analyses of patient characteristics associated with overall survival.

Characteristic		Univariate analysis		Multivariate analysis (n = 33)	
	n	**OS (95 % CI)**	**p value**	**HR (95 % CI)**	**p value**
Sex					
F	25	11.3 (6.7–NR)	0.16	0.4 (0.15–1.06)	0.06
M	14	11.3 (3.5–16.5)			
Age, yr					
<70	28	NR (3.5–NR)	0.03	0.44 (0.14–1.37)	0.16
≥70	11	8.8 (6.3–16.2)			
Smoker					
Non/former	35	13.6 (8.3–17.8)	0.56	1.26 (0.35–4.53)	0.72
Current	4	4.9 (2.5–NR)			
PD–L1, %					
<50	32	14 (6.3–21.4)	0.423	0.51 (0.13–1.97)	0.33
≥50	4	8.6 (8.1–21.6)			
ECOG-PS					
0–1	24	15.3 (8.3–20.8)	0.05	4.79 (1.74–13.17)	0.002
2–4	12	2.2(1.1–11.3)			
Brain metastases					
No	13	16.2 (8.3–NR)	0.22	1.85 (0.72–4.76)	0.2
Yes	26	8.7 (3.5–17.8)			

NR, not reached.

Table 3
Main adverse events occurring in the 39 *EGFR-exon-20ins*–NSCLC patients given amivantamab in an early access program.

Type of event	Adverse events				
	Total (n, %)	Grade 1	Grade 2	Grade 3	Grade 4
Cutaneous	29 (74.4)	11 (28.2)	13 (33.3)	5 (12.8)	
Gastrointestinal	7 (17.9)	2 (5.1)	4 (10.3)	1 (2.6)	
Infusion reaction	11 (25.6)	4 (10.3)	5 (12.8)	1 (2.6)	1 (2.6)
Ocular	4 (10.3)	1 (2.6)	3 (7.8)		
Asthenia	9 (23.1)	6 (15.4)	3 (7.8)		
Neurological	4 (10.3)	2 (5.1)	1 (2.6)	1 (2.6)	

rwDCR of 62 %.

The characteristics of amivantamab-treated patients in this early-access programme are consistent with European epidemiological data. From January 2019 to December 2021, the European Thoracic Oncology Platform registry included 175 patients with *EGFR-exon20ins* NSCLC from 33 centres across nine countries: median age, 64.0 years; 56.3 % women; 76.0 % never or former smokers; and 95.4 % with adenocarcinomas [21]. As in our cohort, some had bone (47.4 %) or brain (32.0 %) metastases. The mean PD-L1 tumour proportion score was 15.8 %. *EGFR-exon20ins* was detected in tissue (90.7 %), plasma (8.7 %) or both (0.6 %), using mainly targeted NGS (64.0 %) or PCR (26.0 %). Mutations were most frequently insertions (59.3 %), followed by duplications (28.1 %), deletion–insertions (7.7 %), and *T790M* (4.5 %). Unlike our French study, insertions and duplications were located predominantly in the near loop (codons 767–771, 83.1 %) and the far loop (codons 771–775, 13 %), with only 3.9 % within the C-helix (codons 761–766).

The limited treatment-efficacy data available for such populations mostly concern patients managed before targeted therapies became available [5] and show very modest efficacy of conventional treatments [22]. In a large series of 11,397 Asian patients, 189 (1.7 %) had *EGFR-exon20ins* [23]. In those patients, classical EGFR-TKI treatment was associated with significantly shorter PFS than in those with *exon 19* deletions or the *L858R* substitution. After platinum-based chemotherapy, classical TKIs and immune checkpoint inhibitors were associated with shorter PFS than docetaxel, with HRs [95 % CIs] of 2.16 months [1.35–3.46] and 1.49 months [1.21–1.84], respectively.Osimertinib was associated with longer PFS in patients with *EGFR-exon20ins* in the near loop than the far loop (median, 5.6 vs. 2.0 months; HR, 0.22 [0.07–0.64]).

According to the American Flatiron Health electronic health record database [24] which included 114 patients with *EGFR-exon20ins* NSCLC (one-third with BMs) diagnosed between 1 January 2011 and 29 February 2020, median first-line rwPFS and OS were 3.7 and 13.6 months, respectively. Second-line or later treatments achieved rwORRs of 9.6 %–14.0 %. Second-line osimertinib monotherapy conferred no clinical benefit. In another US analysis [25] of 15 patients with *EGFR-exon20ins* NSCLC, 7/15 (46.7 %) had received at least one prior TKI, with a median of two prior lines. Among the 14 evaluable patients, the ORR was 36 %, with a DCR of 64.3 %. A pooled analysis of outcomes after second-line or later treatments found ORRs of 0 % for EGFR-TKIs, 3.3 % for immune checkpoint inhibitors, and 13.9 % for chemotherapy, with median PFS of 2.1, 2.3, and 4.4 months, and OS of 14.1, 8.8, and 17.1 months, respectively [26].

Amivantamab efficacy in this early-access programme should be compared with results from clinical trials. In the phase I Chrysalis trial, which included 19 patients with *EGFR-exon20ins* NSCLC (median age, 62 years; 59 % women; 49 % Asian), all had received prior platinum-based chemotherapy and a median of two previous lines of therapy. Amivantamab achieved an ORR of 40 %, with a median DOR of 11.1 months, median PFS of 8.3 months, and a median OS of 22.8 months [14.6–NR].

Outside clinical trials, data on amivantamab efficacy are limited [27–29]. From January 2018 to June 2022, 42 patients with amivantamab-treated *EGFR-exon20ins* NSCLC (16 in a phase I study, and 26 through an early-access programme) were analysed at the Korean Samsung Medical Center. The ORR was 33 % and the DCR was 76 % [27]. Median PFS, stratified by mutation position in the near or far loop for the 31 patients with available data, did not differ significantly: 11.8 months (range, 2.3–21.3) vs. 11.3 months (range, 3.4–19.2) ($p = 0.69$). No significant PFS difference was observed according to *TP53*-mutation status. In contrast, patients with PD-L1–positive (>1%) tumours had poorer prognoses, with median [95 % CI] OS of 11.3 months [5.0–17.6] compared with 19.5 months [5.3–33.7] in PD-L1–negative tumours ($p = 0.04$). In a retrospective analysis of 44 patients who received second- or third-line amivantamab, the median time to next treatment was 9.2 months [6.5–NR] after a median follow-up of 5.5 months.

Two adjusted-treatment comparisons using individual-level data from the Chrysalis study versus rw management of *EGFR-exon20ins* NSCLC provided evidence of amivantamab efficacy in the second-line and later settings [30]. In the first [30], median OS was 22.77 months with amivantamab compared with 12.52 months in the adjusted US and European cohorts (HR: 0.47; $p < 0.0001$) and median PFS was 6.93 vs. 4.17 months (HR: 0.55; $p < 0.0001$) [30,31]. The second analysis [31,] restricted to European data, confirmed this benefit, with median OS of 23.13 months vs. 11.47 months (HR: 0.48, $p = 0.0207$) and median PFS of 6.93 vs. 4.86 months (HR: 0.42, $p < 0.0001$).

BMs remain a major challenge in *EGFR-exon20ins* NSCLC. Trials such as Chrysalis [19], which included only patients with previously treated asymptomatic BMs, reported variable rates of intracranial progression. Effective treatment requires drugs that cross the blood–brain barrier. In our analysis, patients with BMs at baseline had median rwPFS and OS of 2.8 and 8.7 months, respectively, with no significant differences compared with patients without BMs. Among patients evaluable for CNS response, 23.1 % achieved an objective BM response and 69 % achieved intracranial disease control. The median DOR was 5.8 months (range: 2.3–11.9). These findings suggest that amivantamab has activity against BMs, despite its large molecular size, unlike TKIs.

In our study, AEs led to dose reductions in 17.9 % of patients and permanent discontinuation in 10.3 %, compared with 13 % and 4 %, respectively, in the Chrysalis trial [19]. In that trial, grade 3 AEs occurred in 40 (35 %) patients, most commonly hypokalaemia (5 %), rash (4 %), pulmonary embolism (4 %), diarrhoea (4 %), and neutropenia (4 %). These results prompted evaluation of amivantamab combined with chemotherapy versus chemotherapy alone as first-line

therapy in *EGFR-exon20ins* NSCLC [20]. The ORR was significantly higher with the combination (73 % vs. 43 %), with longer responses (median DOR 9.7 vs. 4.4 months). Median PFS was also significantly longer (11.4 vs. 6.7 months). Since publication of the Papillon trial [21–27] results, this combination therapy become the first-line standard of care for metastatic *EGFR-exon20ins* NSCLC.

The main limitation of our study is its retrospective design. Other limitations include non-standardised follow-up, lack of independent review of therapeutic responses, and the relatively small sample size. Although the comparison of outcomes by *EGFR-exon20ins* location would be informative, only two of our patients harboured mutations in the C-helix, precluding meaningful analysis. Nevertheless, the multicentre setting, which included a high proportion of patients typically underrepresented in clinical trials (those with ECOG-PS $\geq$ 2 or BMs), allows a better understanding of amivantamab's contribution in this population.

In conclusion, this rw multicentre study confirms the efficacy and safety of amivantamab in patients with *EGFR-exon20ins* NSCLC, including those with BMs and poor ECOG-PS. These findings support its role as a viable treatment option in routine clinical practice.

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The funding source had no role in the analysis and interpretation of the results.

Author Contributions

Conception and design: TP, LG, FG, CC, JBA. Data collection and analysis: all authors. First draft of the manuscript: TP, JBA, CC. All authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

CRediT authorship contribution statement

Thomas Pierret: Writing – review & editing, Writing – original draft, Validation, Methodology, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Laurent Greillier:** Writing – review & editing, Validation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Florian Guisier:** Writing – review & editing, Validation, Data curation, Conceptualization. **Catherine Daniel:** Validation, Investigation. **Renaud Descourt:** Validation, Investigation. **Loick Galland:** Validation, Investigation. **Olivier Molinier:** Validation, Investigation. **Chantal Decroisette:** Validation, Project administration, Investigation, Funding acquisition, Data curation. **Alexis Cortot:** Validation, Investigation. **Diane Moreau:** Validation, Investigation. **Laurence Bigay Gamé:** Validation, Investigation. **Marie Wislez:** Validation, Investigation. **Nicolas Cloarec:** Validation, Investigation. **Hubert Curcio:** Validation, Investigation. **Nicolas Delberghe:** Validation, Investigation. **Jacques Cadranel:** Validation, Investigation. **Boris Duchemann:** Validation, Investigation. **Anne Claire Toffart:** Validation, Investigation. **Christos Chouaïd:** Writing – original draft, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Jean-Bernard Auliac:** Writing – review & editing, Writing – original draft, Validation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: *Personal fees, grants and non-financial support:* Thomas Pierret: AstraZeneca, Roche, Bristol-Myers Squibb, Merck Sharp & Dohme, and Janssen; Laurent Greillier: AstraZeneca, Boehringer Ingelheim, Roche, Sanofi Aventis, Bristol-Myers Squibb, Merck Sharp & Dohme, Lilly, Novartis, Pfizer, Takeda, Bayer, and Amgen; Florian Guisier: AstraZeneca, Roche, Sanofi Aventis, Bristol-Myers Squibb, Merck Sharp & Dohme, Pfizer, Takeda; Renaud Descourt: AstraZeneca, Roche, Bristol-Myers Squibb, Merck Sharp & Dohme, Pfizer, Takeda, and Chugai; Chantal Decroisette: Merck Sharp & Dohme, Bristol-Myers Squibb, AstraZeneca, Novocure, Pfizer, Amgen, Roche, Regeneron, Takeda, Janssen-Cilag; Alexis Cortot: Merck Sharp & Dohme, Bristol-Myers Squibb, AstraZeneca, Novocure, Pfizer, Amgen, Roche, Regeneron, Takeda, Janssen-Cilag; Laurence Bigay Gamé: Merck Sharp & Dohme, Bristol-Myers Squibb, AstraZeneca, Pfizer, Amgen, Roche, Janssen-Cilag; Marie Wislez: Merck Sharp & Dohme, Bristol-Myers Squibb, AstraZeneca, Novocure, Pfizer, Amgen, Roche, Regeneron, Takeda, Janssen-Cilag; Hubert Curcio: Merck Sharp & Dohme, Bristol-Myers Squibb, AstraZeneca, Novocure, Pfizer, Amgen, Roche, Janssen-Cilag; Jacques Cadranel: Roche, Eli Lilly, Pfizer, Bristol-Myers Squibb, Novartis, AstraZeneca, Takeda, Sanofi, Amgen; Boris Duchemann: Merck Sharp & Dohme, Bristol-Myers Squibb, AstraZeneca, Roche, Regeneron, Takeda, Janssen-Cilag; Christos Chouaïd: Chugai, Daïchi-Sankyo, Abbvie, AstraZeneca, Boehringer Ingelheim, GSK, Roche, Sanofi Aventis, Bristol-Myers Squibb, Merck Sharp & Dohme, Lilly, Novartis, Pfizer, Takeda, Bayer, Amgen; Jean-Bernard Auliac: AstraZeneca, Boehringer Ingelheim, GSK, Roche, Sanofi Aventis, Bristol-Myers Squibb, Merck Sharp & Dohme, Lilly, Novartis, Pfizer, Takeda, Bayer, and Amgen.

Data Availability

Data are available from the corresponding author upon reasonable request.

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