Discover Oncology

CASE REPORT Open Access

Treatment of a newly diagnosed glioblastoma harboring *MET* amplification with vebreltinib in combination with radiotherapy: a case report

Xinliang Liu[1,2†], Xin Wang[2†], Pengwei Yan[2], Dan Zong[2], Wenjie Guo[2] and Xia He[2,3*]

†Xinliang Liu and Xin Wang contributed equally as co-authors of this paper.

*Correspondence:
Xia He
hexiabm@163.com

Full list of author information is available at the end of the article

Abstract

MET-amplified glioblastoma (GBM) is rare, accounting for approximately 2–5% of cases. However, the treatment regimens for patients with *MET*-amplified GBM still have not reached a unanimous consensus. Our paper describes the use of vebreltinib in a newly diagnosed glioblastoma patient harboring *MET* amplification. This successful case study suggests that novel targeted MET inhibitors may provide potential clinical benefits in select cases on the basis of the MET signaling pathway status.

1 Introduction

The oncogene *MET* dysregulation is caused by *MET* amplification, mutation and fusion [1]. *MET*-amplified (7q31) glioblastoma multiforme (GBM) was first reported in 1993 and is rare, accounting for approximately 2–5% of cases [2, 3]. Indeed, *MET* amplification is almost exclusively found in grade 4 gliomas [4]. The incidence of *MET* gain is similar in recurrent GBM (rGBM) and secondary GBM (sGBM, which means *IDH*-mutant GBM progressing from lower-grade) [5]. Almost all cases of *MET* amplification show *MET* overexpression [4, 6]. *MET* overexpression can promote glioma growth and angiogenesis and sustain the glioma stem cell phenotype [7, 8].

Except for prolonged progression-free, but not overall survival afforded by the vascular endothelial growth factor antibody bevacizumab, no pharmacological intervention has been demonstrated to alter the course of disease. Specifically, targeting cellular pathways frequently altered in GBM, such as the PI3K, AKT, mTOR, p53 and RB pathways, or EGFR amplification/mutation, have failed to improve outcomes, likely due to redundant compensatory mechanisms, insufficient target coverage related in part to the blood brain barrier, or poor tolerability and safety [9].

MET receptor tyrosine kinase (RTK) is considered one of the most promising new drug targets for the treatment of GBM. Several MET inhibitors (such as crizotinib, onartuzumab and vebreltinib) have successfully entered clinical trials [10–12]. Vebreltinib is a novel oral MET inhibitor with proven intracranial efficacy. Initial results of the

multicenter, randomized phase II/III FUGEN trial have shown that vebreltinib confers significant survival benefits and lower toxicity for patients with sGBM/isocitrate dehydrogenase (IDH)-mutant GBM with the *PTPRZ1-MET* (*ZM*) fusion gene relative to chemotherapy [12]. Based on this research, vebreltinib was approved by the China National Medical Products Administration for IDH mutant GBM or sGBM patients whose previous treatment failed, and the *ZM* fusion gene on April 23, 2024 (available from https://www.nmpa.gov.cn). The drug market for vebreltinib is mainly in Eastern countries to date. In addition, studies have reported the encouraging efficacy of crizotinib for the treatment of newly diagnosed GBM (nGBM) or *MET*-amplified rGBM [10, 13].

Here, we report a case in which IDH-wild-type nGBM harboring *MET* amplification was successfully treated with vebreltinib.

2 Case presentation

A 51-year-old Chinese male presented with complaints of paroxysmal dizziness and headaches 2 months before hospital admission. This was followed by blurred vision, photophobia, drowsiness and decreased memory. There was no convulsions or projectile vomiting. The patient denied personal and family histories of tumors and worked in a furniture factory. He did not consume alcohol but was an active smoker who smoked 10 cigarettes per day for 30 years. Since the onset of the disease, he was unable to work normally and his Karnofsky performance status (KPS) score was 70.

Contrast-enhanced cranial MR image revealed one space-occupying lesion in the right temporal lobe ($5.7 \times 4.4 \times 4.8$ cm in size) with mixed signals, restricted diffusion–weighted imaging (DWI), extensive surrounding edema, and uneven enhancement, causing a shift of the midline to the left and another in the middle cranial base, which is satellite tumor lesion. The possibility of high-grade gliomas was considered according to the imaging diagnosis. The patient subsequently underwent subtotal supratentorial craniotomy. The patient developed some new surgery-related complications after the operation, such as difficulty in closing the right eyelid, air leakage in the cheek, mild restrictions in tongue movement, and a decreased sense of smell.

Postoperative pathology combined with immunohistochemistry (Fig. 1) revealed that the patient met the diagnostic criteria for glioblastoma (WHO grade 4), with wild-type IDH1 and an unmethylated MGMT promoter. Moreover, heteromorphic cells were observed in the middle cranial base lesion. Genetic testing revealed *MET* amplification (details in Table 1).

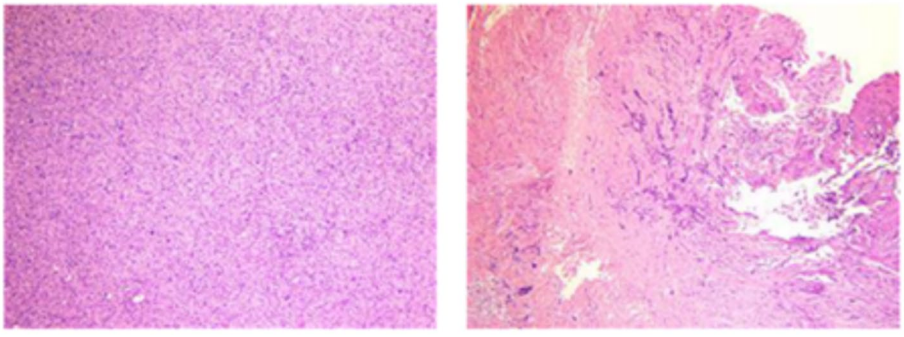

Fig. 1 Results of immunohistochemistry

Table 1 Results of the molecular genetics screening

Item	Result
MGMT promoter methylation	Negative (Methylation ratio: 0.05)
IDH1 R132/R172 mutation	Negative
TP53	Positive (p.E285K)
MET amplification	Positive
CDKN2A/2B homozygous deletion	Positive
CDK4 amplifications	Positive
1p/19q co-deletion	Negative
TERT C228T/C250T mutation	Negative
EGFR amplification/EGFR vIII	Negative
gain of chromosome 7	Negative
loss of chromosome 10	Negative
ATRX inactivation	Negative
Tumor Mutational Burden	3.77Muts/Mb
MSI Status	MSS
Copy Number Variant burden	42.0%

Note: TERT, telomerase reverse transcriptase; ATRX, α-thalassemia mental retardation X-linked

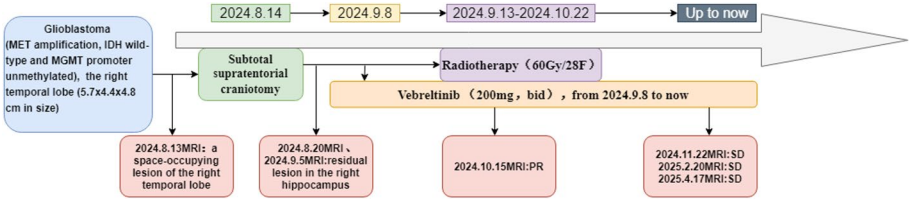

Fig. 2 Timeline of the clinical course

The patient visited the radiotherapy department of our hospital in the first part of September 2024 and received image-guided radiotherapy with PTV1 at 60 Gy/28 fractions and CTV2 at 50.4 Gy/28 fractions from mid-September 2024 to late October 2024, with concomitant and adjuvant oral vebreltinib (200 mg, bid) starting in early September 2024 (Fig. 2). The patient refused TMZ treatment. The patient underwent regular follow-up/monitoring every three months afterwards, primarily consisting of clinical evaluations and cranial MRI at the outpatient department.

During the follow-up period, no disease progression was observed on surveillance MRIs according to the Response Assessment in Neuro-Oncology (RANO) Criteria 2.0 (Fig. 3). Moreover, the patient's symptoms did not worsen. Additionally, no serious vebreltinib-related adverse events occurred in this patient(Table 2).

3 Discussion

With a deeper understanding of the molecular pathophysiology of glioblastoma (GBM), more selective treatment options have been developed and applied in clinical practice [14]. To the best of our knowledge, this patient is the first reported case of a *MET*-amplified nGBM patient treated with vebreltinib.

Based on the early symptoms, the differential diagnosis was cerebral ischemia and migraine, and then GBM was diagnosed by MRI and postoperative pathology. As for the postoperative complications, such as difficulty in closing the right eyelid, air leakage in the cheek, mild restrictions in tongue movement and a decreased sense of smell, we

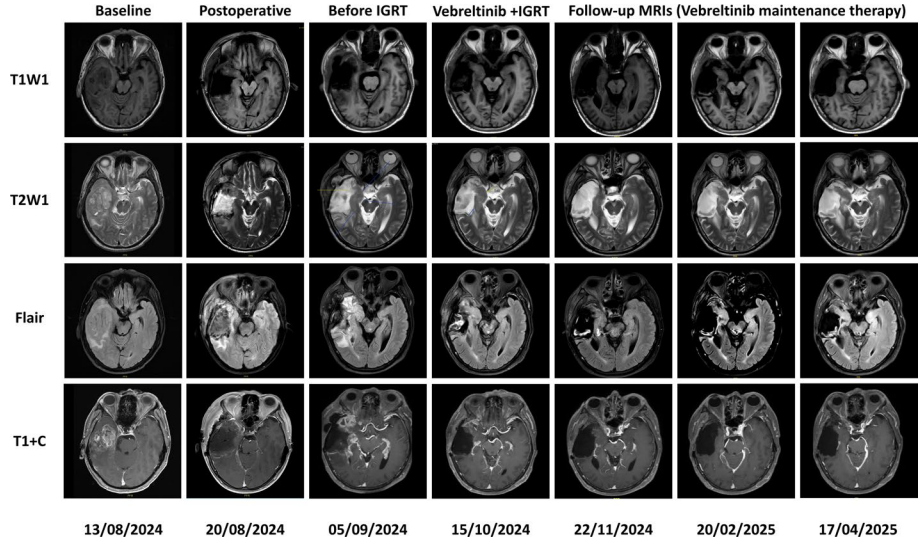

Fig. 3 Dynamic MR image during the clinical course. T1 + C, contrast-enhanced T1-weighted image; T1WI, T1-weighted image; T2WI, T2-weighted image; IGRT, image-guided radiotherapy

Table 2 AEs during vebreltinib treatment

Adverse events	Grading
Hypertriglyceridemia	Grade 1
Hypercholesterolemia	–
Aspartate aminotransferase increased	–
Alanine aminotransferase increased	–
Hypoalbuminemia	–
White blood cell count decreased	–
Platelet count decreased	–
Hypothyroidism	–
Decreased blood sodium	–
Rash	Grade 1
Diarrhoea	–
Decreased appetite	Grade 1
Fatigue	–
Nausea	–
Vomiting	–
Musculoskeletal pain	–
Oral mucosal reactions	–

Note: AEs during treatment evaluated by the Common Terminology Criteria for Adverse Events (CTCAE 5.0)

believe that surgical resection affected the Facial Nerve, Hypoglossal Nerve and Olfactory Nerve.

During the period of approximately 8.5 months from the time of diagnosis to the present, the patient in this case did not experience disease progression. The progression-free survival reported to date is similar to that reported in previous studies [15]. Notably, in addition to *MET* amplification, this patient had other mutations that were negatively associated with efficacy, such as *CDKN2A/2B* homozygous deletion and *CDK4* amplification [16, 17]. Synergistic effects arising from interactions between these mutations have been observed [18], though comprehensive exploration of these mechanisms falls outside the scope of this investigation.

First, patients selection based on the HGF/MET signaling pathway status may provide survival benefits in GBM [19]. The phase II GO27819 study investigated the effects of onartuzumab (a monovalent MET inhibitor) plus bevacizumab (Ona + Bev) compared with placebo plus bevacizumab (Pla + Bev) in rGBM patients [11]. These results demonstrated that adding onartuzumab to bevacizumab did not yield further clinical benefits. Interestingly, subgroup analysis revealed that patients with elevated HGF could benefit from Ona + Bev [11, 20]. Recently, Pham et al.. reported a patient treated with tepotinib for *ZM* fusion and *MET* amplification GBM who achieved a surprising survival time, resulting in a complete and durable response for 35 months [21]. Another case report revealed that rGBM patients treated with crizotinib overexpressing both mesenchymal lymphoma kinase (ALK) and MET proteins had significantly longer survival times than those treated with MET alone [22]. This case report suggests that the more targets a GBM patient receives treatment, the more effective the treatment will be.

Second, when is it optimal to add MET inhibitors? First- or second-line therapy? These questions emphasize the importance of determining the optimal time point for the use of MET inhibitors in clinical practice. There are currently studies with positive results in both modalities (Table 3). Experience from the VE-BASKET study suggests that its use may be more beneficial in patients with nGBM [23].

Finally, aberrant activation of the MET signaling pathway promotes tumor cell migration, proliferation, invasion, survival, and tumor angiogenesis through various pathways, such as the START, PI3K/AKT, and Ras/MAPK pathways (Fig. 4) [25]. However, the clinical prognostic value of MET overexpression in GBM is still a topic of debate. Carvalho et al.. reported that MET overexpression was associated with poorer OS in

Table 3 Summary of published studies and case reports

Ref.	Study Design	No. of patients	Study arms	ORR (%)	mOS (months)	mPFS (months)	PFS6 (%)	grade ≥ 3 adverse events (%)
Martínez-García 2022 GEINO 1402 [10]	Ib, single-arm, multicenter nGBM 2014–2020	38	Stupp regime + crizotinib	28.6	22.6	10.7	71.5	32
Jiang 2024 FUGEN study [12]	II/III, randomized, multicenter, open-label sGBM/IDH mutant GBM patients with failed previous treatment, *ZM* fusion	vebreltinib group: 42 chemotherapy group: 39	vebreltinib TMZ or (cisplatinum with Etoposide)	9.5 2.6	6.31 3.38*	1.87 1.05*	NA	7 12.2
Cloughesy 2017 GO27819 study [11]	II, randomized, double-blind, placebo-controlled, multicenter rGBM 2012–2013	Ona + Bev: 64 Pla + Bev: 65	Onartuzumab + Bev (Ona + Bev) placebo + Bev (Pla + Bev)	22.2 23.7	8.8 12.6	3.9 2.9	33.9 29.0	38.5 35.9
van den Bent 2020 [24]	II, *MET* amplification	10	INC280	0	NA	NA	NA	NA

Note: *, $p < 0.05$; mOS, median overall survival; mPFS, median progression-free survival; ORR, objective response rate; Bev, bevacizumab; NA, not available; TMZ, temozolomide; Stupp regime, (an official) standard radiotherapy with concomitant and adjuvant TMZ; *ZM*, *PTPRZ1-MET*

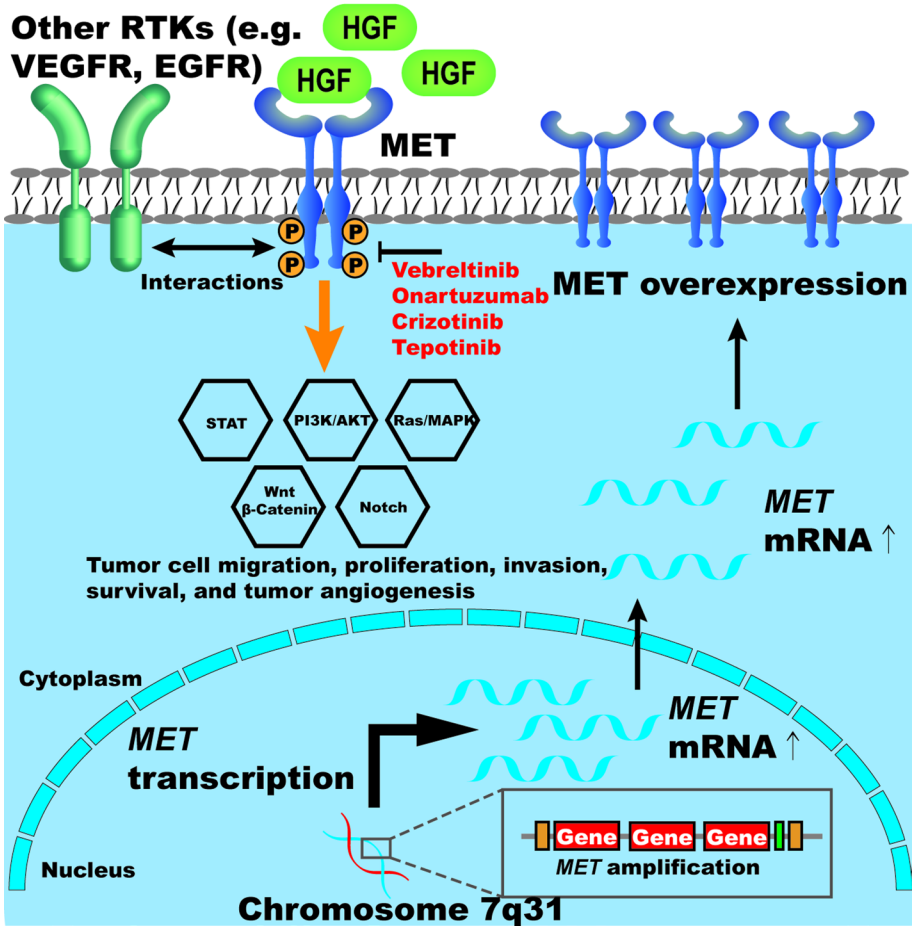

Fig. 4 Mechanism of MET signaling regulation and its downstream signaling pathways

rGBM patients receiving second-line Bev treatment [26]. A study by Kong et al.. yielded similar findings [27]. This may be associated with the activation of the MET/STAT4/PD-L1 pathway and increased number of tumor-associated macrophages [28]. In contrast, Kwak et al.. reported that the survival of GBM patients with MET overexpression was significantly better than that of GBM patients without MET overexpression [4] and that *MET* amplification was not related to the prognosis of GBM [4]. Differences in the thresholds and methods of detection may be one reason for this debate [29]. Moreover, it is still unclear whether or to what extent different *MET* gene alteration patterns play a role in this process.

MET amplification leads to increased transcription and translation of the MET protein with sustained activation of downstream effector signaling, including START, PI3K/AKT, and Ras/MAPK.

4 Conclusion

In summary, this case suggests that patients with *MET*-amplified GBM may benefit clinically from MET inhibitors. However, whether *MET* amplification can serve as a predictive factor for the response of GBM patients to vebreltinib still needs further investigation.

Author contributions
X.L. drafted the manuscript and performed the literature review. X.W. collected the patient information and prepared figures. P.Y. revised it critically for important intellectual content. P.Y., D.Z., W.G. and X.H. conceived and designed this study. All authors reviewed the manuscript.

Funding
This paper was supported by [National Natural Science Foundation of China] under Grant [number: 82172804].

Data availability
Data is provided within the manuscript or supplementary information files.

Declarations

Ethics approval
This case report was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Review Committee of The Affiliated Cancer Hospital of Nanjing Medical University with an approval number of KY-2024-115.

Consent for publication
The patient and his legal guardian has provided written informed consent authorizing the disclosure of the patient's personal information and associated imaging data. All authors approved the final version and agreed to be accountable for all aspects of the work.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Radiotherapy, Affiliated Changzhou No.2 People's Hospital of Nanjing Medical University, The Third Affiliated Hospital of Nanjing Medical University, Changzhou Medical Center, Nanjing Medical University, Changzhou 213000, Jiangsu, China
[2]Department of Radiotherapy, Jiangsu Cancer Hospital, The Affiliated Cancer Hospital of Nanjing Medical University, Jiangsu Institute of Cancer Research, Nanjing 210009, Jiangsu, China
[3]Collaborative Innovation Center for Cancer Personalized Medicine, Nanjing Medical University, Nanjing 210009, Jiangsu, China

Received: 25 February 2025 / Accepted: 12 June 2025
Published online: 17 June 2025

References

1. Jie G-L, Peng L-X, Zheng M-M, Sun H, Wang S-R, Liu S-YM, Yin K, Chen Z-H, Tian H-X, Yang J-J, et al. Longitudinal plasma Proteomics-Derived biomarkers predict response to MET inhibitors for MET-Dysregulated NSCLC. Cancers (Basel). 2023;15:302. https://doi.org/10.3390/cancers15010302.
2. Snuderl M, Fazlollahi L, Le LP, Nitta M, Zhelyazkova BH, Davidson CJ, Akhavanfard S, Cahill DP, Aldape KD, Betensky RA, et al. Mosaic amplification of multiple receptor tyrosine kinase genes in glioblastoma. Cancer Cell. 2011;20:810–7. https://doi.org/10.1016/j.ccr.2011.11.005.
3. Wullich B, Müller HW, Fischer U, Zang KD, Meese E. Amplified Met gene linked to double minutes in human glioblastoma. Eur J Cancer. 1993;29A:1991–5. https://doi.org/10.1016/0959-8049(93)90460-w.
4. Kwak Y, Kim S-I, Park C-K, Paek SH, Lee S-T, Park S-H. C-MET overexpression and amplification in gliomas. Int J Clin Exp Pathol. 2015;8:14932–8.
5. Pierscianek D, Kim Y-H, Motomura K, Mittelbronn M, Paulus W, Brokinkel B, Keyvani K, Wrede K, Nakazato Y, Tanaka Y, et al. MET gain in diffuse Astrocytomas is associated with poorer outcome. Brain Pathol. 2013;23:13–8. https://doi.org/10.1111/j.1750-3639.2012.00609.x.
6. Burel-Vandenbos F, Ngo-Mai M, Dadone B, Di Mauro I, Gimet S, Saada-Bouzid E, Bourg V, Almairac F, Fontaine D, Virolle T, et al. MET Immunolabelling is a useful predictive tool for MET gene amplification in glioblastoma. Neuropathol Appl Neurobiol. 2017;43:252–66. https://doi.org/10.1111/nan.12320.
7. Abounader R, Laterra J. Scatter factor/hepatocyte growth factor in brain tumor growth and angiogenesis. Neuro Oncol. 2005;7:436–51. https://doi.org/10.1215/S1152851705000050.
8. Boccaccio C, Comoglio PM. The MET oncogene in glioblastoma stem cells: implications as a diagnostic marker and a therapeutic target. Cancer Res. 2013;73:3193–9. https://doi.org/10.1158/0008-5472.CAN-12-4039.
9. Le Rhun E, Preusser M, Roth P, Reardon DA, van den Bent M, Wen P, Reifenberger G, Weller M. Molecular targeted therapy of glioblastoma. Cancer Treat Rev. 2019;80:101896. https://doi.org/10.1016/j.ctrv.2019.101896.
10. Martínez-García M, Velasco G, Pineda E, Gil-Gil M, Alameda F, Capellades J, Martín-Soberón MC, López-Valero I, Tovar Ambel E, Foro P, et al. Safety and efficacy of Crizotinib in combination with Temozolomide and radiotherapy in patients with newly diagnosed glioblastoma: phase Ib GEINO 1402 trial. Cancers (Basel). 2022;14:2393. https://doi.org/10.3390/cancers14102393.
11. Cloughesy T, Finocchiaro G, Belda-Iniesta C, Recht L, Brandes AA, Pineda E, Mikkelsen T, Chinot OL, Balana C, Macdonald DR, Randomized, Double-Blind, et al. Placebo-Controlled, multicenter phase II study of onartuzumab plus bevacizumab versus placebo plus bevacizumab in patients with recurrent glioblastoma: efficacy, safety, and hepatocyte growth factor and O6-Methylguanine-DNA methyltransferase biomarker analyses. J Clin Oncol. 2017;35:343–51. https://doi.org/10.1200/JCO.2015.64.7685.
12. Jiang T, Bao Z, Yang F-D, Mao Q, Li S, Qu Y, Wang L, Mou Y, Yu R-T, Wu J, et al. Efficacy and safety of the vebreltinib in patients with previously treated, secondary glioblastoma/idh mutant glioblastoma with PTPRZ1-*METFUsion gene* (FUGEN):

A randomised, multicentre, Open-Label, phase II/III trial. J Clin Oncol. 2024;42. https://doi.org/10.1200/jco.2024.42.16_suppl.2003.
13. Chi AS, Batchelor TT, Kwak EL, Clark JW, Wang DL, Wilner KD, Louis DN, Iafrate AJ. Rapid radiographic and clinical improvement after treatment of a MET-Amplified recurrent glioblastoma with a Mesenchymal-Epithelial transition inhibitor. J Clin Oncol. 2012;30:e30–33. https://doi.org/10.1200/JCO.2011.38.4586.
14. Solomon BJ, Drilon A, Lin JJ, Bazhenova L, Goto K, De Langen J, Kim D-W, Wolf J, Springfeld C, Popat S, et al. 1372P Repotrectinib in patients (Pts) with NTRK Fusion-Positive (NTRK+) advanced solid tumors, including NSCLC: update from the phase I/II TRIDENT-1 trial. Ann Oncol. 2023;34:S787–8. https://doi.org/10.1016/j.annonc.2023.09.2405.
15. Gilbert MR, Wang M, Aldape KD, Stupp R, Hegi ME, Jaeckle KA, Armstrong TS, Wefel JS, Won M, Blumenthal DT, et al. Dose-Dense Temozolomide for newly diagnosed glioblastoma: A randomized phase III clinical trial. J Clin Oncol. 2013;31:4085–91. https://doi.org/10.1200/JCO.2013.49.6968.
16. Appay R, Dehais C, Maurage C-A, Alentorn A, Carpentier C, Colin C, Ducray F, Escande F, Idbaih A, Kamoun A, et al. CDKN2A homozygous deletion is a strong adverse prognosis factor in diffuse malignant IDH-Mutant gliomas. Neuro Oncol. 2019;21:1519–28. https://doi.org/10.1093/neuonc/noz124.
17. Li KK-W, Shi Z-F, Malta TM, Chan AK-Y, Cheng S, Kwan JSH, Yang RR, Poon WS, Mao Y, Noushmehr H, et al. Identification of subsets of IDH-Mutant glioblastomas with distinct epigenetic and copy number alterations and stratified clinical risks. Neuro-Oncol Adv. 2019;1:vdz015. https://doi.org/10.1093/noajnl/vdz015.
18. Olmez I, Zhang Y, Manigat L, Benamar M, Brenneman B, Nakano I, Godlewski J, Bronisz A, Lee J, Abbas T, et al. Combined C-Met/Trk Inhibition overcomes resistance to CDK4/6 inhibitors in glioblastoma. Cancer Res. 2018;78:4360–9. https://doi.org/10.1158/0008-5472.CAN-17-3124.
19. Tasaki T, Fujita M, Okuda T, Yoneshige A, Nakata S, Yamashita K, Yoshioka H, Izumoto S, Kato A. MET expressed in glioma stem cells is a potent therapeutic target for glioblastoma multiforme. Anticancer Res. 2016;36:3571–7.
20. Szerlip NJ, Pedraza A, Chakravarty D, Azim M, McGuire J, Fang Y, Ozawa T, Holland EC, Huse JT, Jhanwar S, et al. Intratumoral heterogeneity of receptor tyrosine kinases EGFR and PDGFRA amplification in glioblastoma defines subpopulations with distinct growth factor response. Proc Natl Acad Sci U S A. 2012;109:3041–6. https://doi.org/10.1073/pnas.1114033109.
21. Pham LC, Weller L, Gann CN, Schumacher KM, Vlassak S, Swanson T, Highsmith K, O'Brien BJ, Nash S, Aaroe A et al. Prolonged Complete Response to Adjuvant Tepotinib in a Patient with Newly Diagnosed Disseminated Glioblastoma Harboring Mesenchymal-Epithelial Transition Fusion. *Oncologist* 2024, oyae100. https://doi.org/10.1093/oncolo/oyae100
22. Le Rhun E, Chamberlain MC, Zairi F, Delmaire C, Idbaih A, Renaud F, Maurage CA, Grégoire V. Patterns of response to Crizotinib in recurrent glioblastoma according to ALK and MET molecular profile in two patients. CNS Oncol. 2015;4:381–6. https://doi.org/10.2217/cns.15.30.
23. Kaley T, Touat M, Subbiah V, Hollebecque A, Rodon J, Lockhart AC, Keedy V, Bielle F, Hofheinz R-D, Joly F, et al. BRAF Inhibition in BRAFV600-Mutant gliomas: results from the VE-BASKET study. J Clin Oncol. 2018;36:3477–84. https://doi.org/10.1200/JCO.2018.78.9990.
24. van den Bent M, Azaro A, De Vos F, Sepulveda J, Yung WKA, Wen PY, Lassman AB, Joerger M, Tabatabai G, Rodon J, et al. A phase ib/ii, Open-Label, multicenter study of INC280 (Capmatinib) alone and in combination with buparlisib (BKM120) in adult patients with recurrent glioblastoma. J Neurooncol. 2020;146:79–89. https://doi.org/10.1007/s11060-019-03337-2.
25. Bradley CA, Salto-Tellez M, Laurent-Puig P, Bardelli A, Rolfo C, Tabernero J, Khawaja HA, Lawler M, Johnston PG, Van Schaeybroeck S, et al. Targeting C-MET in Gastrointestinal tumours: rationale, opportunities and challenges. Nat Rev Clin Oncol. 2017;14:562–76. https://doi.org/10.1038/nrclinonc.2017.40.
26. Carvalho B, Lopes JM, Silva R, Peixoto J, Leitão D, Soares P, Fernandes AC, Linhares P, Vaz R, Lima J. The role of C-Met and VEGFR2 in glioblastoma resistance to bevacizumab. Sci Rep. 2021;11:6067. https://doi.org/10.1038/s41598-021-85385-1.
27. Kong D-S, Song S-Y, Kim D-H, Joo KM, Yoo J-S, Koh JS, Dong SM, Suh Y-L, Lee J-I, Park K, et al. Prognostic significance of C-Met expression in glioblastomas. Cancer. 2009;115:140–8. https://doi.org/10.1002/cncr.23972.
28. Wang Q-W, Sun L-H, Zhang Y, Wang Z, Zhao Z, Wang Z-L, Wang K-Y, Li G-Z, Xu J-B, Ren C-Y, et al. MET overexpression contributes to STAT4-PD-L1 signaling activation associated with Tumor-Associated, Macrophages-Mediated immunosuppression in primary glioblastomas. J Immunother Cancer. 2021;9:e002451. https://doi.org/10.1136/jitc-2021-002451.
29. Guo R, Luo J, Chang J, Rekhtman N, Arcila M, Drilon A. MET-Dependent solid Tumours - Molecular diagnosis and targeted therapy. Nat Rev Clin Oncol. 2020;17:569–87. https://doi.org/10.1038/s41571-020-0377-z.

Publisher's note