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Ivosidenib treatment in *IDH*-mutant WHO grade 4 astrocytomas: illustrative case

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BACKGROUND Isocitrate dehydrogenase (*IDH*) is a commonly mutated gene in gliomas. Although *IDH*-mutant WHO grade 4 astrocytomas tend to occur in younger patients and show an improved survival compared to *IDH*-wildtype, prognosis remains poor and treatment options are limited. Ivosidenib is a small-molecule inhibitor of IDH-1 that has shown promise for treating low-grade *IDH*-mutant glioma. However, little is known about its efficacy in *IDH*-mutant WHO grade 4 astrocytoma.

OBSERVATIONS The authors present the case of a 36-year-old male with a *IDH*-mutant WHO grade 4 astrocytoma. He was originally treated with resection, followed by radiation therapy and temozolomide. At the second recurrence, ivosidenib was started, after lomustine therapy had failed to control the disease. Radiological surveillance showed interval decrease in tumor size starting 2 months after initiation of ivosidenib. The patient remained neurologically intact, and no adverse effects were observed.

LESSONS Here the authors report the effects of the off-label use of ivosidenib. The INDIGO trial published evidence of efficacy in using an *IDH* inhibitor for low-grade gliomas. The role of these drugs in high-grade *IDH*-mutant gliomas is currently unknown. Further studies are needed to assess their impact on overall and progression-free survival.

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KEYWORDS ivosidenib; isocitrate dehydrogenase; *IDH* mutation; high-grade glioma

Gliomas are the most common primary malignant CNS neoplasms, with an incidence of 6 cases per 100,000 persons in the United States annually.[1] Gliomas are classified by the WHO classification system into four grades, with WHO grade 4 being the most aggressive.[2] Gliomas harboring different molecular mutations may carry a more favorable prognosis than their wildtype counterparts. Isocitrate dehydrogenase (*IDH*) mutation, MGMT methylation status, and translocation of 1p19q can greatly affect prognostic outcomes and therapeutic regimens. With the recent update to the WHO glioma classification system, WHO grade 4 astrocytomas are now separated into two separate entities of *IDH*-mutant astrocytomas and *IDH*-wildtype glioblastomas depending on *IDH* status, with the latter having a worse prognosis.[3] *IDH*-mutant WHO grade 4 astrocytomas have a median progression-free survival (PFS) of approximately 2.7 years, compared to *IDH*-wildtype glioblastomas with a median PFS of 6.2–7.5 months.[4,5]

IDH is a cell metabolism enzyme that plays a crucial role in the citric acid cycle by catalyzing the oxidative decarboxylation of isocitrate to 2-oxoglutarate.[6] *IDH* gene mutations are present in approximately 10% of high-grade gliomas and more than 70% of grade 2 or 3 gliomas.[7] Their presence significantly influences tumor biology and prognosis.[8] Mutant *IDH* (mIDH) produces the oncometabolite D-2-hydroxyglutarate (2-HG), which has been implicated in promoting gliomagenesis and epileptogenesis.[9] It has been hypothesized that 2-HG disrupts the regulation of DNA methylation, thus inducing oncogene expression.[10] The current standard of care treatment for *IDH*-mutant WHO grade 4 astrocytomas is radiation therapy followed by temozolomide.[11] *IDH*-mutant WHO grade 4 astrocytomas tend to occur in younger patients and show an improved survival; however, prognosis remains poor and treatment options are limited.[12] Recent research has shown promising results for treating low-grade *IDH*-mutant glioma by inhibiting mIDH. Vorasidenib and ivosidenib are small-molecule inhibitors of IDH-1, with vorasidenib recently gaining Food and Drug Administration (FDA) approval for the treatment of low-grade *IDH*-mutant glioma in 2024 after the results of the INDIGO (Study of Vorasidenib [AG-881] in Participants With Residual or Recurrent Grade 2 Glioma With an IDH1 or IDH2 Mutation) trial.[13,14] However, little is known about the efficacy

ABBREVIATIONS α-KG = α-ketoglutarate; 2-HG = D-2-hydroxyglutarate; CK = creatine kinase; FDA = Food and Drug Administration; FLAIR = fluid-attenuated inversion recovery; IDH = isocitrate dehydrogenase; mIDH = mutant IDH; PFS = progression-free survival.

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of these inhibitors in *IDH*-mutant WHO grade 4 astrocytoma. We present a retrospective case of a WHO grade 4 astrocytoma treated off-label with ivosidenib, offering insight into its therapeutic application in high-grade gliomas.

Illustrative Case

A 36-year-old male with no significant past medical history presented to the emergency department with intermittent headaches and photophobia. On physical examination, the patient was noted to be neurologically intact. MRI of the brain showed a left frontal heterogeneously enhancing cystic lesion with notable vasogenic edema and significant mass effect (Fig. 1A). He subsequently underwent a left frontal craniotomy with gross-total resection of the lesion (Fig. 1B). Final pathology was consistent with a WHO grade 4 astrocytoma. Molecular analysis revealed the presence of an IDH-1 mutation (R132H), *MGMT* promoter methylation, and intact 1p/19q. *CDKN2A/B* homozygous deletion was not detected. The patient subsequently underwent chemoradiation treatment followed by 6 cycles of adjuvant temozolomide. Follow-up MRI 10 months after completion of chemoradiation therapy revealed subependymal enhancement along the medial margins of the lateral ventricles, progression of T2 fluid-attenuated inversion recovery (FLAIR) signal in the left frontal lobe, and new focal enhancement in the right frontal lobe, concerning for progression of disease (Fig. 1C). The patient was started on lomustine 90 mg/m^2 to treat the disease progression. Throughout the 3 months of lomustine treatment, the patient experienced nausea, vomiting, and thrombocytopenia, and follow-up MRI displayed further progression of the disease after two cycles of lomustine therapy (Fig. 1D). Lomustine treatment was discontinued, and the patient was subsequently started on ivosidenib 500 mg daily 1 month later. Interval decrease in tumor size and stabilization of disease were noted as early as 2 months after initiation of ivosidenib therapy (Fig. 1E). The patient has remained neurologically and radiologically stable on ivosidenib therapy for 9 months (Fig. 1F), and no adverse treatment effects have been observed.

Informed Consent

The necessary informed consent was obtained in this study.

Discussion

Observations

IDH is an enzyme critical to cell metabolism. *IDH* functions through the tricarboxylic acid cycle and fatty acid synthesis by catalyzing the conversion of isocitrate to α-ketoglutarate (α-KG), reducing NADP to NADPH in the process.[6] *IDH* mutations are characterized by a missense mutation that causes an arginine residue substitution in the enzyme's active site, leading to altered active site catalytic function and DNA hypermethylation. IDH-1 mutations are commonly associated with an R132H substitution, while *IDH*-2 is associated with R140Q and R172K mutations. *IDH* mutations tend to occur early in tumor development and persist throughout the disease.[6]

Mutated *IDH* causes a gain-of-function mutation by producing the oncometabolite 2-HG from α-KG and oxidizing NADPH to NADP. Accumulated 2-HG combined with depleted α-KG ultimately inhibits α-KG–dependent enzymes, disrupting cell metabolism, cell signaling, and epigenetic regulation. 2-HG affects epigenetics by interrupting normal DNA demethylation driven by ten-eleven translocation dioxygenase proteins.[6] Ultimately, the oncometabolite 2-HG acts as a driver of gliomagenesis by altering tumor metabolism and epigenetics. 2-HG is important in glioma studies, as 2-HG tumor concentration can be measured to evaluate treatment efficacy. 2-HG levels in intracranial CSF have been explored as a potential biomarker for *IDH*-mutant gliomas. Decreasing 2-HG concentration in tumors has been associated with proneural gene expression reversal and downregulation of stem cell genes.[6,15]

Glioma prognosis can be aided by imaging, histopathology, and molecular profiling. Tumor genetics can reveal the status of *IDH* mutations, MGMT promoter methylation, chromosome 1p/19q co-deletion, or TERT mutations. *IDH* mutations are implicated in gliomagenesis and tumor biology. *IDH* mutations are present in up to 90% of grade 2, 3, and 4 astrocytomas and oligodendrogliomas.[6] They are associated with a better prognosis and less aggressive tumor biology than *IDH*-wildtype.[12] The median overall survival in patients with *IDH*-mutant WHO grade 4 astrocytoma is approximately 5.9 years, whereas the median overall survival in patients with *IDH*-wildtype glioblastoma is approximately 15 months.[4,16] This is due in part to *IDH*-mutant gliomas harboring a better response to traditional therapy, possibly due to lower NADPH levels than *IDH*-wildtype gliomas.[17] *IDH*-wildtype gliomas often possess additional, aggressive genetic alterations such as epidermal growth factor receptor amplification, *CDKN2A/B* homozygous deletion, or partial chromosome 7 gain and chromosome 10 loss.[18] *CDKN2A/B* is a tumor suppressor gene alteration found in 16%–34% of WHO grade 4 astrocytomas and is one of the strongest predictors of poor prognosis when homozygous deletion is present. Deletion of *CDKN2A/B* has been shown to cause increased proliferation of glioma cells and is associated with promotion of tumor angiogenesis. Overall survival in the presence of *CDKN2A/B* homozygous deletion in WHO grade 4 *IDH*-mutant gliomas is reported to be 38 months versus 86 months in the absence of its deletion. [19]

Lessons

Ivosidenib, a small-molecule inhibitor of IDH-1, is a potential therapy for *IDH*-mutant glioma. Ivosidenib has been approved by the FDA for the treatment of acute myeloid leukemia and cholangiocarcinoma harboring *IDH* mutations. The INDIGO trial published in 2023 showed evidence of efficacy in using an IDH-1 and -2 inhibitor, vorasidenib, for low-grade gliomas.[13] Vorasidenib was found to be efficacious in treating *IDH*-mutant low-grade glioma with longer radiologically based PFS compared to placebo (27.7 months vs 11.1 months), and has an adverse event rate of at least 10% among any grade glioma.[13] Ivosidenib is currently undergoing clinical trials for its treatment of low-grade *IDH*-mutant glioma with promising results. A randomized controlled trial by Mellinghoff et al. showed significant reduction (91.1%) of 2-HG levels with decreased tumor cell proliferation in patients with low-grade gliomas treated with ivosidenib compared with controls.[20] Ivosidenib has also shown significant reduction in tumor growth rates and radiological volume reductions up to 33 months on treatment in chemoradiation-naive low-grade gliomas, with minimal adverse reactions.[20,21]

While ivosidenib has primarily been studied in low-grade gliomas, emerging literature suggests a potential role in the treatment of high-grade *IDH*-mutant gliomas. We performed a literature review utilizing the search terms "glioma and ivosidenib" in the PubMed database in March 2025, which resulted in 33 articles. Eight articles were excluded after initial title and abstract screening. Review articles (n = 14), articles using other IDH inhibitors (n = 1), and articles in which only lower-grade glioma (WHO grades 1–3) (n = 5) or no WHO grade (n = 1) was treated were excluded. A total of 16 cases of *IDH*-mutant grade 4 astrocytomas treated with ivosidenib were extracted from 4 articles (Table 1). Of these cases, 3 were retrospective case studies[22–24] and

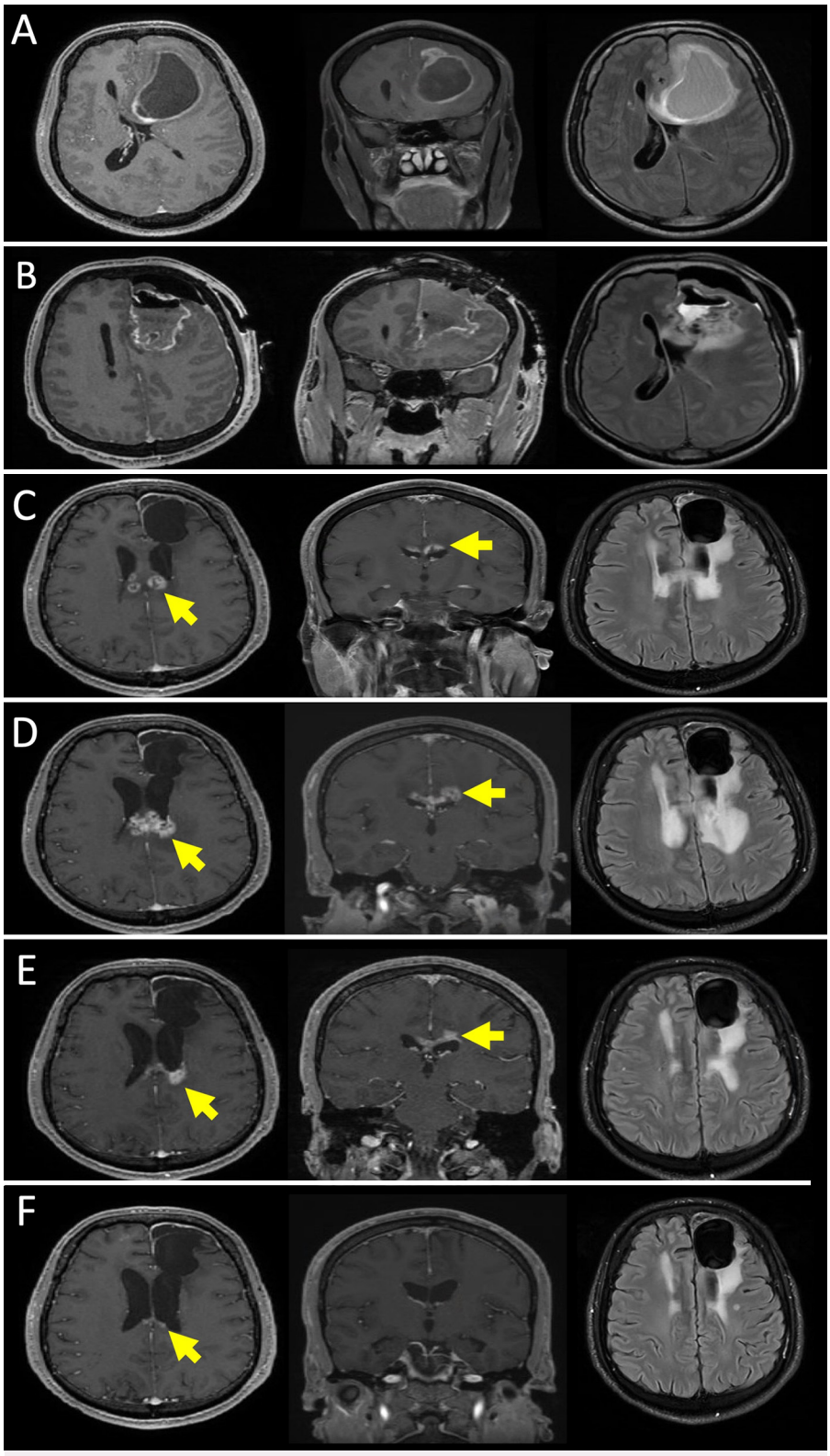

FIG 1. Axial (*left column*) and coronal (*center column*) T1 and T2 FLAIR (*right column*) MR images with gadolinium contrast. **A:** Preoperative MR images of the brain showing a cystic contrast-enhancing lesion in the left frontal lobe with associated mass effect and surrounding edema on T2 FLAIR image. **B:** Postoperative MR images demonstrating gross-total resection of the tumor. **C:** Ten months after completion of chemoradiation therapy, follow-up MR images revealed progression of disease with new contrast-enhancing subependymal lesions (*arrows*). **D:** Three months after starting lomustine therapy to treat the disease progression, follow-up MR images showed increased contrast enhancement and progression of T2 FLAIR signal intensity, concerning for further progression of disease. **E:** After starting ivosidenib therapy, 2-month follow-up MR images demonstrated decreased tumor size, improvement of contrast enhancement, and T2 FLAIR intensity. **F:** MR images of the brain at 9 months showing resolution of contrast enhancement and stability of disease.

TABLE 1. Mutant *IDH* grade 4 astrocytoma cases treated with ivosidenib reported in the literature

Authors & Year	Article Type	No. of Patients w/ Grade 4 mIDH Astrocytoma	Total No. of Patients w/ Glioma	Previous Treatments	Length of Treatment	Outcomes	Reported AEs of Ivosidenib
Mellinghoff et al., 2020[14]*	RCT	12	66	Surgery, RT, &/or chemotherapy	Median 1.9 mos	45.2% stable disease in enhancing gliomas; 33.3% decrease in tumor size from baseline; median PFS 1.4 mos for enhancing tumors	AEs among patients w/ grade ≥3: headache (2%), seizure (4%), hyperglycemia (2%), hypophosphatemia (4%)
Tejera et al., 2020[23]	Retrospective case study	1	1	RT & TMZ	4 yrs	Stable disease for 4 yrs, disease progressed at 4 yrs 1 mo, requiring additional RT & bevacizumab	No AEs
Peters et al., 2024[22]*	Retrospective case study	1	14	Some received RT & TMZ	Median 43.7 mos	62.5% stable disease in all enhancing gliomas; 50% stable disease in patients who received prior RT & TMZ, 12.5% had minor response	AEs among patients of all grades: diarrhea (26.7%), elevated CK (33.3%), QTc prolongation (16.7%)
Lanman et al., 2025[24]	Retrospective case study	2	74	RT &/or chemotherapy	Median 8 mos	1 patient had stable disease, 1 patient had disease progression; median PFS 5.6 mos	AEs among patients w/ grade 4 astrocytoma: elevated CK in 2 patients (100%)
Present study	Retrospective case study	1	1	RT, TMZ, & lomustine	13 mos	Stable disease for 13 mos after initiation of ivosidenib	No AEs

AE = adverse effect; RCT = randomized controlled trial; RT = radiation therapy; TMZ = temozolomide.
* Outcomes were not distinguished by tumor grade.

1 was a randomized controlled trial.[14] All the patients with grade 4 astrocytomas in these studies had received prior radiation therapy and/or chemotherapy and were subsequently treated with 500 mg of oral ivosidenib daily. Across the studies, the most notable adverse effects of ivosidenib therapy included headaches (2%),[14] seizure (4%–13.3%),[14,22] hyperglycemia (2%),[14] hypophosphatemia (4%),[14] diarrhea (26.7%),[22] elevated creatine kinase (CK) (3%–33.3%),[22,24] and QTc prolongation (16.7%).[22] In some cases, increased seizures were associated with progression of disease.[22]

Treatment outcomes varied among the studies. Treatment response was measured throughout the studies based on MRI analysis to determine stability or progression of disease. Two of the studies divided the treatment analysis by patients with enhancing disease (all grade 4 tumors in addition to some grade 3 tumors) versus nonenhancing disease.[14,22] In 1 study, the median PFS for patients with enhancing gliomas was 1.4 months, with 45.2% experiencing stable disease and 33.3% experiencing a reduction in tumor size from baseline.[14] Peters et al. reported that 62.5% of patients with enhancing gliomas experienced stable disease and 50% of these patients who had received prior radiation therapy and/or temozolomide experienced stable disease.[22] Tejera et al. reported the case of a single patient with mIDH grade 4 astrocytoma who experienced stable disease for 4 years.[23] Lanman et al. included 2 grade 4 patients, of whom 1 experienced stable disease, with a median PFS of 5.6 months among patients with enhancing gliomas.[24]

Across all these studies, ivosidenib appears to offer disease stabilization in glioma, especially low-grade nonenhancing tumors. The literature suggests that ivosidenib is primarily associated with radiological stability rather than tumor shrinkage. However, there are still limited data on the role of ivosidenib in higher-grade enhancing gliomas, with most studies including a small sample size of grade 4 gliomas.

Prior to initiation with ivosidenib, our patient was treated with lomustine, an alkylating chemotherapy. Although lomustine may provide a modest survival benefit, it can be limited by delayed adverse effects of thrombocytopenia and hepatotoxicity. Recurrent glioblastoma treated with lomustine demonstrates a median PFS of less than 2 months and an overall survival of 6–9 months.[25] Ivosidenib was started in our patient after failure of radiation, temozolomide, and lomustine therapy to control disease progression. After initiating treatment in our patient, radiological surveillance showed a reduction in tumor size beginning 2 months after initiation of ivosidenib. Throughout the treatment period, the patient remained neurologically stable, with no adverse effects observed.

This case provides critical insights into the management of *IDH*-mutant WHO grade 4 astrocytomas using off-label ivosidenib. Our patient's positive outcome with ivosidenib therapy adds to the limited but growing literature suggesting that *IDH* inhibitors may provide a therapeutic benefit even in high-grade gliomas. In addition to tumor size reduction, our patient remained neurologically intact, without experiencing any adverse effects of ivosidenib therapy, with an overall survival of 31 months thus far. The observed radiological tumor reduction and the absence of adverse effects underscore the importance of exploring *IDH* inhibitors as a targeted therapy, even in aggressive tumor settings. Repeat resection for tumor recurrence is also important to consider as a potential treatment option. Repeat surgery may be needed if recurrent disease develops in noneloquent, surgically accessible regions or in cases of symptomatic or life-threatening recurrent disease, causing mass effect or edema. Although generalized

conclusions cannot be drawn, this case suggests that *IDH* inhibitors may be well tolerated, potentially offering a better quality of life compared to conventional chemotherapies like lomustine, which often carry significant toxicities. Further randomized trials are needed to assess the efficacy of *IDH* inhibitors in the treatment of WHO grade 4 *IDH*-mutant astrocytomas.

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Disclosures

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Author Contributions

Conception and design: Cachia, Daci, Amenta. Acquisition of data: Cachia, Owusu-Adjei, Renterghem, Hayes Meizoso, Amenta. Analysis and interpretation of data: Cachia, Owusu-Adjei, Daci, Amenta. Drafting the article: Cachia, Owusu-Adjei, Renterghem, Hayes Meizoso, Daci, Amenta. Critically revising the article: Cachia, Owusu-Adjei, Daci, Mietus, Amenta. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Cachia. Administrative/technical/material support: Daci.

Supplemental Information

Previous Presentations

Portions of this work were presented as a poster at the 29th Annual Meeting and Education Day of the Society for Neuro-Oncology, Houston, TX, November 21–24, 2024; the UMass 4th Annual Department of Surgery Research Symposium, Worcester, MA, May 1, 2025; and the AMA Research Poster Showcase at the Annual Meeting of the AMA House of Delegates, Chicago, IL, June 6–11, 2025.

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