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## Case Report

# Newly emerged T2 high-signal intensity area mimicking oligodendroglioma expansion on intraoperative magnetic resonance imaging: A case report ☆☆☆

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## ABSTRACT

Magnetic resonance imaging (MRI) is an indispensable tool in neurosurgery, though it sometimes faces challenges such as “tumor mimicry.” While intraoperative MRI (iMRI) is widely recognized for its usefulness in achieving maximal safe resection during glioma surgery, instances of tumor mimicry still occur on iMRI. Moreover, reports on tumor mimics observed through iMRI, particularly in low-grade gliomas, remain scarce. In this article, we present a case of oligodendroglioma, where a newly emerged T2 high-signal intensity region on iMRI necessitated differentiation from tumor expansion.

A 23-year-old man presented with a newly diagnosed brain tumor and underwent surgical removal. An iMRI taken after tumor removal revealed a newly emerged T2 hyperintense area without diffusion restriction around the resection cavity, which was not observed in the preoperative MRI. Suspecting residual tumor, we performed additional resection. An MRI on the following day confirmed that the T2 hyperintense area identified on the iMRI had been completely resected but also revealed an enlarged T2 high-signal area over a wider region. Histopathology found no tumor cells in the additionally resected area, indicating that the iMRI finding was a tumor mimic. Six months later, the T2 high-signal area around the resection cavity had disappeared on MRI without any additional treatment.

**Abbreviations:** ADC, apparent diffusion coefficient; BBB, blood-brain barrier; CC, corpus callosum; CT, computed tomography; CBV, cerebral blood volume; CNS, central nervous system; DWI, diffusion weighted image; EOR, extent of resection; FLAIR, fluid attenuated inversion recovery; IDH, isocitrate dehydrogenase; iMRI, intraoperative magnetic resonance imaging; LGG, low-grade glioma; MPR, multiplanar reconstruction; MRI, magnetic resonance imaging; OD, oligodendroglioma; WHO, World Health Organization; WI, weighted image.

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This case highlights the challenge of distinguishing between T2 hyperintense mimicry and tumor enlargement during glioma surgery seen on iMRI. Despite the significant value of iMRI, our report underscores the need for careful interpretation in neurosurgical practice, particularly with non-contrast-enhancing tumors.

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## Introduction

Magnetic resonance imaging (MRI) has become essential in neurosurgical practice, especially for evaluating and differentiate various brain lesions [1]. However, on MRI, lesions that are not brain tumors may appear similar brain tumors, a phenomenon called as “tumor mimics.” Tumor mimics are by no means rare, occurring in 3.4%-4.3% of central nervous system (CNS) lesions [2,3].

Maximal safe resection is essential for treating gliomas, including oligodendroglioma (OD), as it significantly extends overall survival [4,5]. Although intraoperative magnetic resonance imaging (iMRI) plays an indispensable role in glioma surgeries by improving the extent of resection (EOR) [6,7], tumor mimics can theoretically be misidentified as actual tumors, and this may lead to an overestimation of tumor extension. Reports on tumor mimics observed on iMRI during glioma surgeries are scarce [8]. There is a need for more research on tumor mimics also in the context of low-grade glioma (LGG) surgeries.

Here, we report a rare case of OD featuring a newly emerged T2-hyperintense area on iMRI and postoperative MRI, which mimicked tumor expansion.

## Case presentation

A 23-year-old man visited our hospital after experiencing his first epileptic seizure. He showed no permanent neurological deficits but experienced transient motor aphasia. An MRI revealed a hyperintense lesion on T2 weighted images (T2WIs) localized to the superior frontal gyrus and paracingulate gyrus in the right hemisphere (Fig. 1). The lesion appeared as low intensity on T1 weighted images (T1WIs) and high intensity on fluid-attenuated inversion recovery (FLAIR) images, with no enhancement by Gd-based contrast medium. There were no abnormal vessels or increased cerebral blood volume (CBV) around the lesion. Although no calcification was observed on computed tomography (CT), the lesion extended to the brain cortex. Based on these findings, we suspected an oligodendroglioma and decided to proceed with tumor removal.

We performed tumor resection, sacrificing small vessels exposed in the operative field. The tumor was negative for 5-aminolevulinic acid-induced protoporphyrin IX fluorescence. On iMRI, after resecting the tumor, a new hyperintense area on T2WIs appeared just beneath the surgical cavity, which was absent in the MRI taken 2 weeks prior to the surgery (Figs. 2A-E). This new lesion, confined to the white matter just lateral to the corpus callosum (CC), showed hyperintensity on

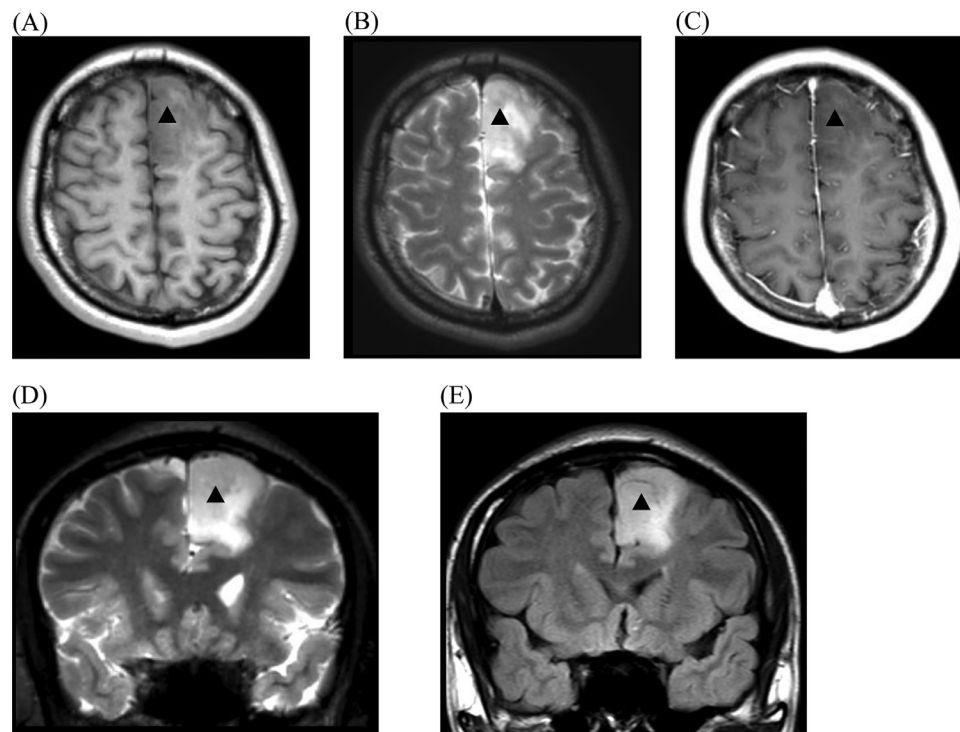
T2WIs without diffusion restriction. Although we were skeptical about the possibility of tumor growth in such a short period, we directly inspected the newly emerged T2-high area under the microscopy and found it to be a soft and whitish brain area, hardly distinguishable from the surrounding normal brain tissue (Fig. 2F). Given the inability to rule out tumor growth, we excised the area and submitted it as a separate specimen for pathological diagnosis.

On a postoperative MRI taken the next day, the T2 hyperintense area that appeared intraoperatively had been excised, but the new T2-high signal area without diffusion restriction had further enlarged outside of the surgical cavity (Fig. 3). The pathological diagnosis of the tumor was oligodendroglioma, IDH-mutant and 1p/19q-codeleted, classified as CNS WHO grade 2; however, no tumor cells were found in the additionally resected specimens. Therefore, we concluded that the newly emerged lesion was not an expansion of the tumor and that gross total resection was achieved. The patient was discharged home on the seventh postoperative day without any neurological deficits. The T2 high-signal area seen on the MRI the day after surgery disappeared 6 months later without atrophy and was judged to be a transient imaging change caused by the surgical operation (Fig. 4).

## Discussion

We encountered a rare case of OD where an expansion of the T2-hyperintense area on iMRI mimicked a newly emerged tumor lesion, which was eventually identified as a nontumoral lesion through pathological diagnosis and follow-up MRI (Fig. 4). The value of iMRI is now undeniable, as it facilitates the evaluation of residual tumors and complications during glioma surgeries without increasing the risk of postoperative infection or bleeding [6,7,9,10]. On the other hand, despite the lack of reports on tumor mimics on iMRI during OD surgery, tumor mimics can indeed occur on iMRI [8], making iMRI a double-edged sword. For instance, if tumor mimics are mistaken for residual tumors during surgery, the iMRI findings could lead to undue pressure to perform unnecessary extractions. In our case, the new lesion identified on iMRI was located in a noneloquent area, and the disadvantages of additional removal were considered minimal. Nevertheless, since the lesion was devoid of residual tumors, careful interpretation of iMRI findings is essential.

In non-contrast-enhancing tumors, such as low-grade gliomas (LGGs), determining whether a T2 high-signal region around the surgical cavity represents a tumor can be challenging [11]. Therefore, it is advisable to consider clinical information in such cases. In other words, it is generally unlikely for



**Fig. 1 – Preoperative MRI findings.** Axial sections of MRI reveal a hyperintense lesion on T2WIs (A) localizing to the superior frontal gyrus and paracingulate gyrus in the right hemisphere (arrowheads). T1WIs before and after the administration of Gd-based contrast medium (B and C) show a hypointense lesion without any enhancement. The tumor extends to the brain cortex on coronal sections of T2WIs and FLAIR images (D and E). Abbreviations: FLAIR, fluid-attenuated inversion recovery; MRI, magnetic resonance imaging; WI, weighted image.

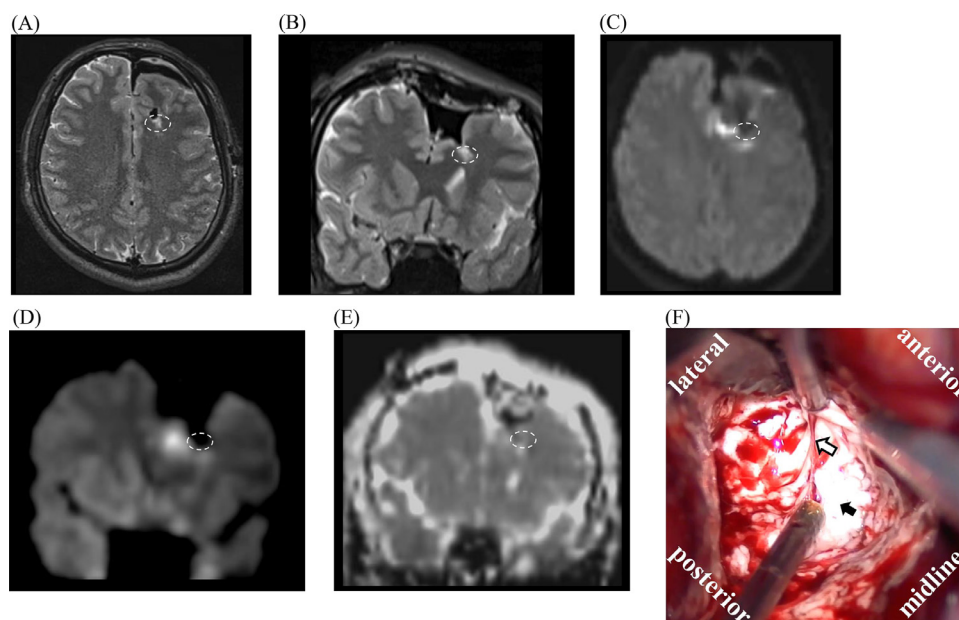
a slowly growing tumor to extend to a new site on intraoperative imaging, suggesting that newly emerged T2 hyperintense regions on iMRI are likely tumor mimics. In this context, a recent preoperative MRI is helpful for differentiating the lesion. The optimal timing for taking a preoperative MRI for LGG surgeries has yet to be established. However, given that the imaging growth rate of LGGs is approximately 2 mm/year in diameter [12], evaluating an MRI performed 2 weeks prior to surgery in this case is considered reasonable.

The present case is unique because the T2 high-signal region appeared and enlarged quickly, then spontaneously disappeared after several months. Determining the underlying mechanism of the T2 hyperintense lesion development is difficult, as there have been no reports of similar cases. According to previous reports, the etiologies of tumor mimics on preoperative MRI have been identified as vascular, inflammatory nondemyelinating, demyelinating, infectious, among others (e.g., Wallerian degeneration, edematous change)[2,3]. Given the rapid change and reversibility of this condition, demyelination, Wallerian degeneration, and infectious diseases are unlikely in our case. No pathological findings of inflammatory cell infiltration or cerebral infarction were found in the additionally resected specimens. Collectively, by the process of elimination, we believe that an edematous change caused the tumor mimic.

Cerebral edema is classified into 3 categories: cytotoxic, vasogenic, and interstitial [13,14]. Cytotoxic edema, linked to cell

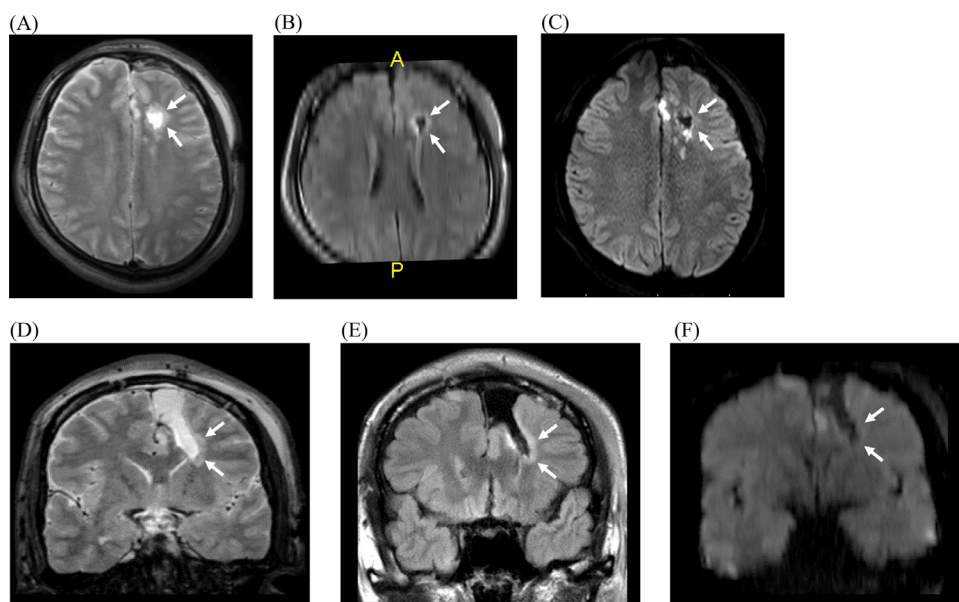
death in the brain and considered irreversible, typically leaves the blood-brain barrier (BBB) intact. It is associated with conditions such as cerebral infarction, encephalitis, and hypoxic encephalopathy; cytotoxic edema is unlikely in our case. Vasogenic edema arises from the disruption of the BBB due to various pathological conditions, including brain tumors, infections or inflammatory responses, and traumatic brain injuries, including those from surgical manipulations. In our case, it is conceivable that intraoperative manipulations might have caused BBB disruption in the brain tumor patient. Additionally, local interstitial edema could also have contributed to the observations in the present case. Interstitial edema, which includes osmotic and hydrostatic components, is characterized by water accumulation in the brain parenchyma while the BBB remains preserved. Although conditions such as hydrocephalus, intraoperative abnormally high blood pressure, or hyponatremia were not present, the resection of the tumor involved sacrificing small veins that appeared intraoperatively (Fig. 2), potentially leading to local impeded venous return and subsequent interstitial edema.

Assuming that vasogenic and interstitial edema are the causes of the T2-high signal intensity in this case, there are 2 points that need clarification: first, the very rapid appearance of the T2 high-signal region, and second, the reason for the change occurrence in this case, which does not happen every time. One of the advantages of iMRI is its ability to evaluate images before postoperative reactive changes occur,



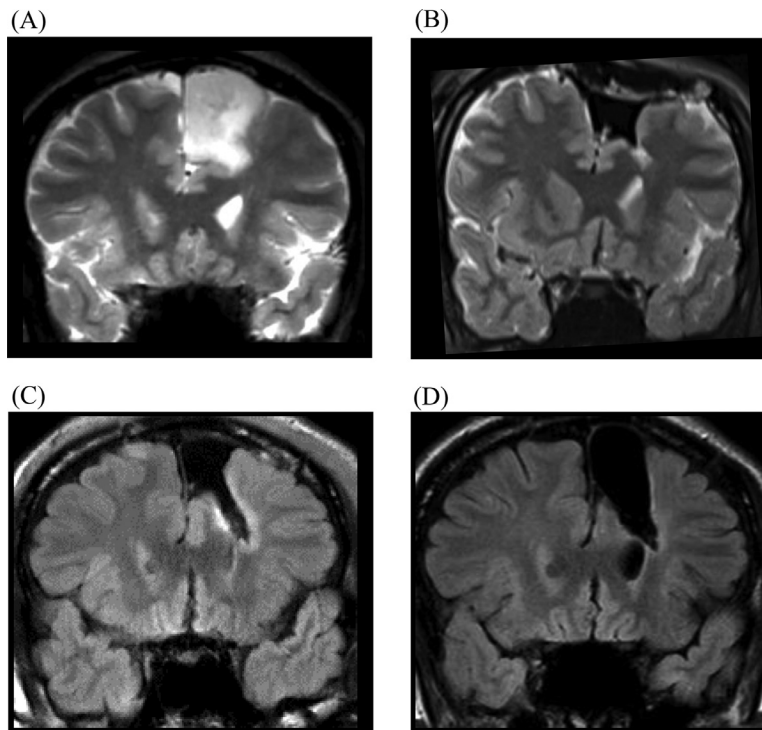
**Fig. 2 – Intraoperative MRI and microscopic findings.** Intraoperative MRI after resecting the tumor reveals an expansion of T2 hyperintense area around the resection cavity on axial (A) and coronal sections (B). The lesion shows no diffusion restriction (dotted circles) on axial DWI (C) and coronal sections of reconstructed DWI and ADC maps (D and E). MPR was performed using Centricity™ Universal Viewer (GE Healthcare, Barrington, IL, USA). An intraoperative microscopic image of the newly emerged T2-high area (F) displays whitish brain tissue, that is difficult to differentiate from the surrounding brain area (arrow). A blood vessel (vein) traverses the surgical field (blank arrow).

Abbreviation: ADC, apparent diffusion coefficient; DWI, diffusion weighted image; MPR, multi-planar reconstruction; MRI, magnetic resonance imaging.



**Fig. 3 – Postoperative MRI findings.** An MRI performed on the following day confirms that the T2 hyperintense area visible on the iMRI was resected. It also reveals an enlarged T2 high-signal area covering a wider area on T2WIs (A and D) and FLAIR images (B and E) (arrows). The T2 high-signal area exhibits no diffusion restrictions on DWIs (C and F) (arrows). The images (B and F) were reconstructed using MPR software, Centricity™ Universal Viewer (GE Healthcare, Barrington, IL, USA). Abbreviations: MRI, magnetic resonance imaging; DWI, diffusion-weighted image; FLAIR, fluid-attenuated inversion recovery; iMRI, intraoperative magnetic resonance imaging; MPR, multi-planar reconstruction; WI, weighted image.





**Fig. 4 – MRI findings over time. Coronal sections of MRI taken preoperatively (A, T2WI), intraoperatively (B, T2WI), postoperatively (C, FLAIR), and 6 months after surgery (D, FLAIR) illustrate the changes over time. T2 or FLAIR hyperintense areas enlarged intraoperatively and shortly postoperatively but disappeared without atrophy after 6 months. Abbreviations: FLAIR, fluid-attenuated inversion recovery; MRI, magnetic resonance imaging; WI, weighted image.**

especially the T2 hyperintense rim around the surgical cavity [11]. However, iMRI has demonstrated a T2-hyperintense area at the resection margin in 21.4%–50.0% of LGG cases, suggesting that very early T2-high reactive changes can be detected by iMRI [11,15]. Additionally, the location of the lesion and the direction of venous return may have been involved. The standard venous return around the deep white matter of the superior/middle frontal gyrus (equivalent to the outside of the CC) flows to the deep venous system through the network of subependymal veins (i.e., a ventriculopetal pattern) [16]. Conversely, in cases with a developmental venous anomaly, blood flow sometimes drains into the superficial venous system through cortical veins to the superior sagittal sinus (i.e., ventriculofugal pattern) [16]. In glioma, thick peritumoral veins often develop, and the tumor may have altered the venous return pattern from ventriculopetal to ventriculofugal in the present case. Thus, injuries to small veins during surgery resulted in impaired venous return and ultimately caused the edematous change just beneath the surgical cavity. Further case accumulation is warranted.

In conclusion, we report a case of OD that required differentiation from tumor enlargement due to an enlarged T2 high-signal area observed on iMRI. Although the precise mechanism behind the pathogenesis of tumor mimics remains unclear, tumor mimics can occur on iMRI, underscoring the importance of careful image interpretation.

### Patient consent

We obtained informed consent concerning publication and handled clinical information anonymously in accordance with the principles of the Declaration of Helsinki and the “Act on the Protection of Personal Information” in Japan.

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