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Case Report

Glioblastoma with markedly reduced contrast enhancement after corticosteroid administration: Increased density and reduced diffusion capability are noteworthy ☆,☆☆

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ABSTRACT

Corticosteroids are widely used to manage peritumoral edema and associated neurological deficits in patients with brain tumors. We describe the case of a 71-year-old male patient with glioblastoma in which contrast enhancement decreased on radiographic imaging following corticosteroid administration, which has been reported previously in only 9 cases. This report aims to discuss radiographic changes in glioblastoma (density on computed tomography and diffusion capability on diffusion-weighted magnetic resonance imaging, in addition to enhancement on contrast-enhanced T1-weighted magnetic resonance imaging) following steroid administration and also following steroid discontinuation.

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Introduction

Glioblastoma is the most common primary brain and other central nervous system (CNS) malignancy, accounting for approximately 14% of all neoplasms and 50% of all malignant tumors [1]. Lymphomas of the CNS are the second most frequent, accounting for approximately 7% of all malignant neoplasms [2]. Primary diffuse large B-cell lymphoma (DLBCL) of the CNS corresponds to 80%-85% of all CNS lymphomas [3].

Corticosteroids are widely used to manage peritumoral edema and associated neurological deficits in patients with brain tumors [4]. Steroid-induced reduction in contrast enhancement on radiographic imaging is a well-known phenomenon in primary DLBCL of the CNS, in which preoperative steroid administration alters the histological features of the lesion and confounds the diagnosis after biopsy. In contrast, there are reports of only 9 cases of glioblastoma that demonstrated reduced contrast enhancement under steroid therapy [5–12].

We present a case of glioblastoma in which corticosteroid administration resulted in radiographic changes related to enhancement on contrast-enhanced T1-weighted magnetic resonance imaging (MRI), diffusion capability on diffusion-weighted MRI (DWI), and density on computed tomography (CT) after steroid administration and also after steroid discontinuation.

Case report

A 71-year-old male presented with literacy difficulties, memory impairment, and right homonymous hemianopsia. Non–contrast-enhanced CT showed a heterogeneous lesion in the left temporal, occipital, and parietal lobes that was seen on MRI as a mass lesion with irregular contrast enhancement accompanied by vascular development and perifocal edema (Fig. 1). Glioblastoma was suspected, but early surgery was difficult due to the COVID-19 pandemic. To manage the perifocal edema while awaiting surgery, the patient was commenced on betamethasone 8 mg/day.

Non–contrast-enhanced CT at 15 days after the first day of steroid treatment showed increased lesion density as well as reduced perifocal edema (Fig. 2A). Contrast-enhanced T1-weighted MRI at 18 days after the first day of steroid treatment showed markedly reduced contrast enhancement, and DWI showed reduced diffusion capability (Fig. 2B-D). Steroid treatment was discontinued after 21 days and a biopsy was performed the next day, but no definitive diagnosis was obtained.

Corticosteroids were not resumed after the biopsy. At 9 days after the first day of steroid discontinuation, non–contrast-enhanced CT showed decreased lesion density (Fig. 3A). Enhancement returned on contrast-enhanced MRI, and diffusion restriction was obscure on DWI at 12 days af-

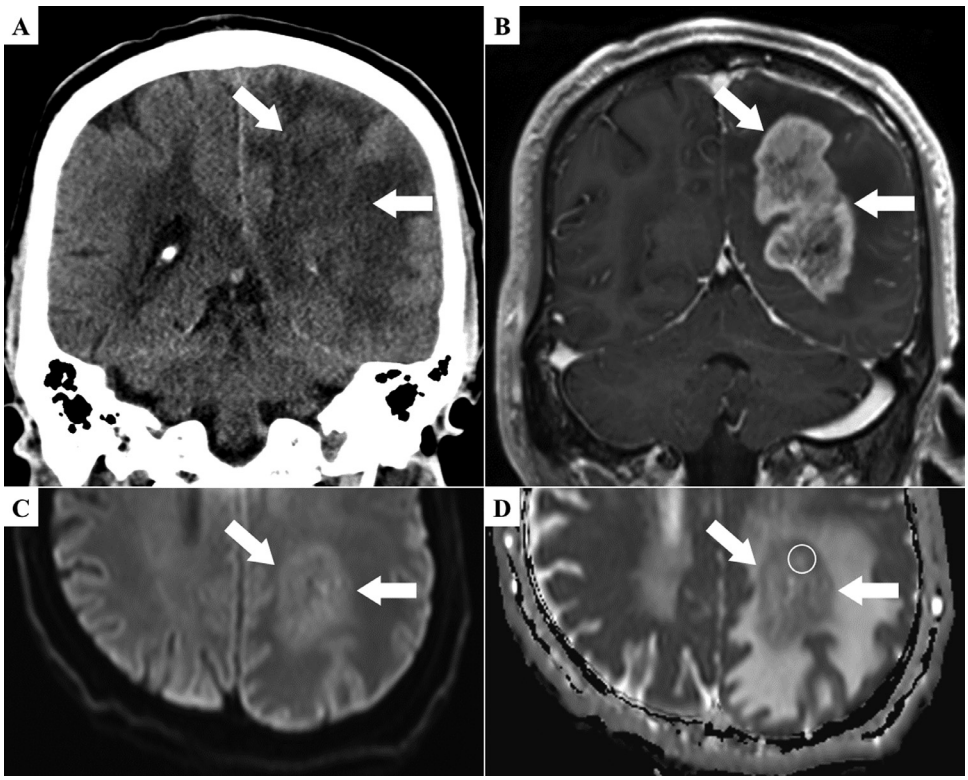

Fig. 1 – Imaging obtained before steroid administration. (A) Non–contrast-enhanced CT shows a heterogeneous lesion in the left temporal, occipital, and parietal lobes (arrows). (B) Contrast-enhanced T1-weighted MRI shows a mass lesion with irregular contrast enhancement accompanied by vascular development (arrows) and perifocal edema. (C, D) DWI shows slightly restricted diffusion (arrows), with an apparent diffusion coefficient (ADC) of 1.128 × 10^{-3} mm^2/s (circle).

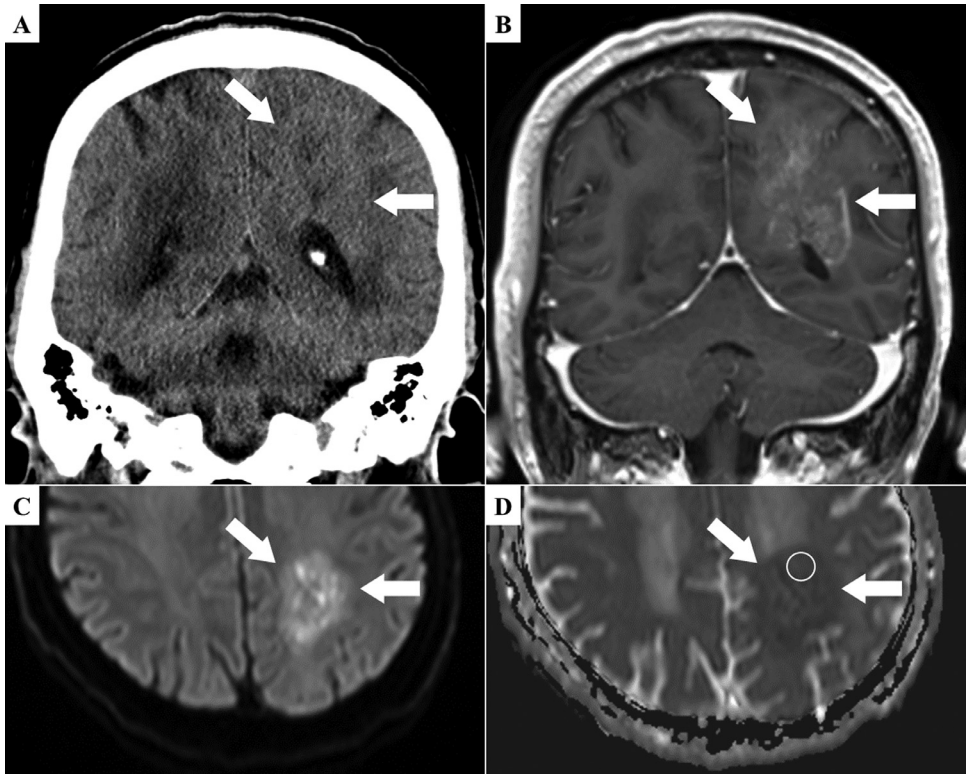

Fig. 2 – Imaging obtained after steroid administration. (A) Non–contrast-enhanced CT at 15 days after the first day of steroid treatment shows increased lesion density and reduced perifocal edema (arrows). (B) Contrast-enhanced T1-weighted MRI at 18 days after the first day of steroid treatment shows markedly reduced contrast enhancement (arrows). (C, D) DWI at 18 days after the first day of steroid treatment shows reduced diffusion capability (arrows), with an ADC of 0.716 x 10^{-3} mm^2/s (circle). The regions of markedly restricted diffusion indicate possible areas of intratumoral ischemia.

ter the first day of steroid discontinuation (Fig. 3B-D). The second biopsy was performed at 14 days after the initial biopsy. Pathological examination showed microvascular proliferation, anisonucleosis and necrosis, and confirmed a diagnosis of glioblastoma, IDH-wildtype (Fig. 4).

Discussion

Glioblastoma and primary DLBCL of the CNS both usually show contrast enhancement and occasionally have similar radiographic findings. However, preoperative differentiation is extremely important because the neurosurgical strategies for these brain tumors are substantially different. For glioblastoma, maximal resection contributes to better prognosis [13], whereas in primary DLBCL of the CNS, stereotactic biopsy is recommended to confirm the diagnosis [14].

Corticosteroids are widely used to reduce perifocal edema and manage associated neuropathy in patients with brain tumors. However, steroid administration also causes changes in the tumor itself. Steroid-induced regression on radiographic imaging is well known in primary DLBCL of the CNS, whereas steroid-induced reduction in contrast enhancement has been reported in limited cases of glioblastoma [5–12]. It is considered that in primary DLBCL of the CNS, the reduced contrast enhancement following steroid administration is caused by induction of tumor cell apoptosis [15], whereas in glioblastoma, the cause is probably decreased vascular permeability caused by repair of the blood-brain barrier [16–18]. The response in glioblastoma may depend on the dosage of corticosteroid [12]. As with primary DLBCL of the CNS, biopsy after steroid administration for glioblastoma may not yield a definitive diagnosis [19]. In such a case, there may be a delay in treatment due to clinical suspicion of primary DLBCL of the CNS. Therefore, it is of high importance to obtain a correct diagnosis even when radiographic imaging is obtained after steroid administration.

In the current case, in addition to reduced contrast enhancement for the glioblastoma, the steroid treatment resulted in diffusion restriction on DWI and increased density was observed on non–contrast-enhanced CT. These findings are the opposite to the radiographic changes seen in primary DLBCL of the CNS after steroid administration. To the best of our knowledge, no previous study has reported the simultaneous occurrence of reduced diffusion capability and markedly reduced contrast enhancement after steroid administration. The above findings suggest that steroid administration reduces vascular permeability and decreases the water content of the tumor tissue itself; i.e., that steroid administration may increase the relative cell density. After steroid discontinuation, the contrast enhancement returned, diffu-

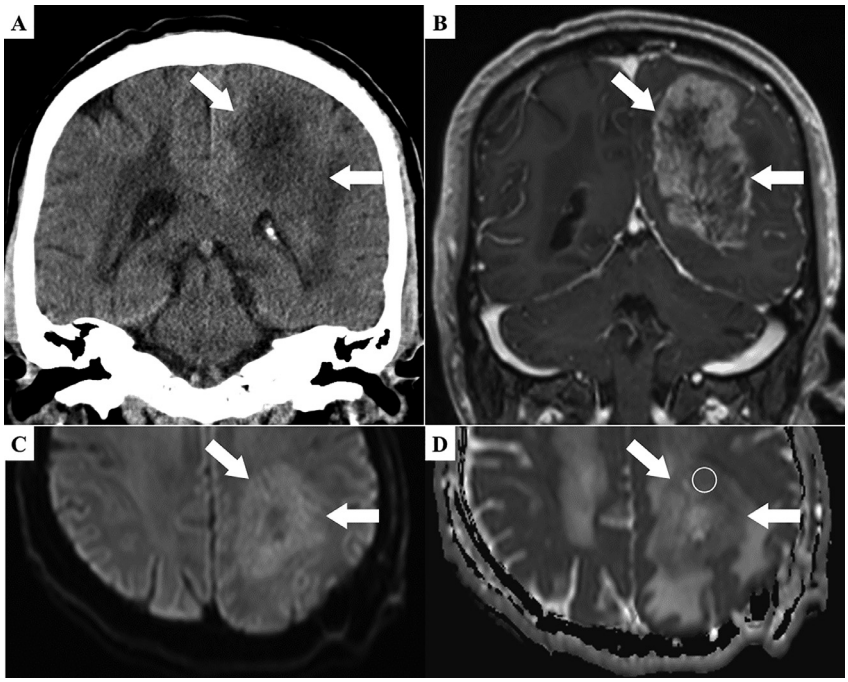

Fig. 3 – Imaging obtained after discontinuation of steroid administration. (A) Non–contrast-enhanced CT at 9 days after the first day of steroid discontinuation shows decreased lesion density (arrows). (B) Contrast-enhanced T1-weighted MRI at 12 days after the first day of steroid discontinuation shows recurrent contrast enhancement (arrows). (C, D) DWI at 12 days after the first day of steroid discontinuation shows obscure diffusion restriction (arrows), with an ADC of 0.989 × 10^{-3} mm^2/s (circle).

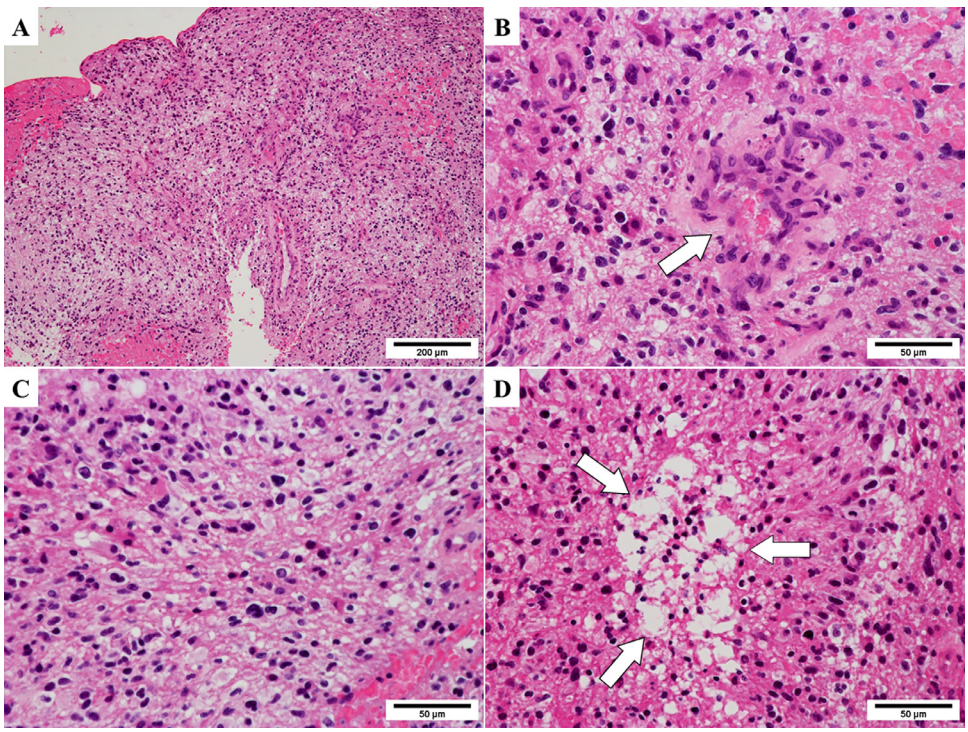

Fig. 4 – (A) Pathological examination of the hematoxylin-eosin stained section of the second biopsy at low magnification (10 ×). High-power (40 ×) photomicrographs show (B) microvascular proliferation (arrow), (C) anisonucleosis and (D) necrosis (arrows) consistent with glioblastoma, IDH-wildtype.

sion restriction became obscure, and the density of the lesion on non–contrast-enhanced CT decreased. We speculate that these changes on DWI and non–contrast-enhanced CT also indicate the water content or relative cell density of the tumor tissue in glioblastoma.

Conclusion

Steroid treatment of glioblastoma resulted in increased density on non–contrast-enhanced CT and diffusion restriction on DWI, in addition to a reduction in enhancement on contrast-enhanced T1-weighted MRI. These findings suggest that corticosteroid administration decreased vascular permeability in glioblastoma and reduced the water content of the tumor tissue itself. These findings may be useful in differentiating glioblastoma from primary DLBCL of the CNS.

Ethics approval

This study was carried out in accordance with the principles of the Declaration of Helsinki and its later amendments, and approved by the institutional review board at Kyoto University Hospital (approval number: R2088-3).

Patient consent

Informed consent was obtained from the patient for publication.

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