A 68-year-old male presented to the emergency department with a headache. He had no known comorbidities. His general physical and neurological examination was normal. He underwent an emergency magnetic resonance (MR) screening which showed a T1 heterogeneously hypointense, T2 heterogeneously hyperintense [Figure 1] lesion involving the subcortical and deep white matter of the right parietooccipital region. The lesion was noted to infiltrate across the midline through the splenium of corpus callosum. Perilesional edema was noted causing regional sulcal effacement. A focal area of diffusion restriction was noted in the inferomedial aspect of the lesion suggestive of high cellularity zone [Figure 2]. Tiny intrallesional foci of blooming were noted suggestive of microbleeds [Figure 3]. Postcontrast T1-weighted fat saturated images showed evidence of peripheral heterogeneous enhancement [Figure 4]. Single-voxel MR spectroscopy (MRS) showed elevated choline, lactate, lipid peaks, and decreased N-acetylaspartate (NAA) peak in the intrallesional [Figure 5] and perilesional [Figure 6] regions of the tumor. In addition, choline: NAA ratio was 1.7, choline: Creatinine ratio was 2.4, and NAA: Creatinine ratio was 1.41 in the intrallesional voxel and choline:NAA ratio was 2.29, choline:Creatinine ratio was 1.36, and NAA:Creatinine ratio was 1.68 in the perilesional voxel. In assumption of a malignant tumor, we performed a stereotactic-guided right parietooccipital craniotomy with radical subtotal resection of the lesion, and the histopathological examination confirmed the presence of glioblastoma multiforme (GBM). The patient underwent additional radiotherapy and chemotherapy with concomitant temozolomide. Temozolomide dosed at 75 mg/m²/day, started the night before the first radiation treatment and given for 42 consecutive days. Dexamethasone with a dosage of 16 mg daily, administered in four equal doses was given to the patient postsurgery followed by a maintenance dose of 0.5–1 mg dose of dexamethasone daily. Postoperative MR imaging (MRI) revealed postoperative changes with no evidence of abnormal enhancement/residual tumor.

**DISCUSSION**

GBM (WHO grade IV astrocytoma) is the most common primary brain malignancy, accounting for up to 15% of...
all intracranial neoplasms. It occurs most commonly in the middle age population (mean age of 55 years) and is frequently supratentorial in location[1]. An existing astrocytoma may dedifferentiate into a high-grade glioma, in which case they are secondary glioblastomas. GBMs usually form in the cerebral white matter, grow quickly, and can become very large before producing symptoms. Less than 10% form more slowly following degeneration of low-grade astrocytoma or anaplastic astrocytoma. These are called secondary GBMs and are more common in younger patients (mean age 45 vs. 62 years).[2] They are high-grade tumors that have classical MRI features as described in our patient. In MRS, elevated choline and reduced NAA suggest tumor turnover and neuronal loss; metabolite ratios (choline-creatine, choline-NAA, NAA-creatine, myoinositol-creatine) correlate with tumor grade.[3]

**CONCLUSION**

GBM is the most common primary brain tumor, accounting for up to 15% of all intracranial neoplasms. MRI is the imaging modality of choice in the evaluation of gliomas, and can accurately detect high-grade tumors. MRS is a useful problem-solving tool to grade gliomas; to differentiate them from other intra-axial lesions such as metastasis, anaplastic astrocytoma, oligodendroglioma, cerebral abscess, central nervous system lymphoma, encephalitis, radiation necrosis, tumefactive demyelination, and toxoplasmosis to delineate peritumoral microinfiltration, and to aid in preoperative mapping.

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**Conflicts of interest**

There are no conflicts of interest.

**REFERENCES**