PROC (BAYL UNIV MED CENT)
2022;35(2):248–249

https://doi.org/10.1080/08998280.2021.1990703

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Phantosmia and dysgeusia as the first presentation of glioblastoma

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ABSTRACT

Glioblastoma is the most common and aggressive primary brain tumor in adults and has an extremely poor prognosis. It is rare for a high-grade glioma, or any brain tumor, to have an initial presentation of gastrointestinal symptoms. We present a rare case of a healthy 70-year-old woman who presented with symptoms of phantosmia and dysgeusia with magnetic resonance imaging findings of a brain mass confirmed via brain biopsy to be glioblastoma. Through a rare presentation, we aim to elucidate the importance of recognizing the association of phantosmia and dysgeusia to seizure auras and the critical need for diagnostic imaging to rule out organic causes such as infection and neoplasms.

KEYWORDS Dysgeusia; glioblastoma; glioma; grade IV glioma; phantosmia; seizure

Glioblastoma is a stage IV rapidly progressive glioma. Patients typically present with subacute, progressive focal neurological symptoms relative to the tumor location. Focal neurological deficits are more likely than seizures to be the presenting symptom of glioblastoma. Any neurological deficit necessitates diagnostic imaging to rule out organic causes such as infection, neoplasm, or vascular abnormalities that can mimic symptoms seen with glioblastoma. Herein we report a case of glioblastoma presenting with new onset symptoms of seizure auras without a prior history or other focal neurological symptoms.

CASE DESCRIPTION

A 70-year-old woman presented with the main complaint of nausea over the past 6 months that had increased in frequency from two to three times per week to four to five times per week without known triggers. Upon further inquiry, she described these episodes as starting with a sudden sensation of foul smell and taste that caused her to feel nauseous and dizzy, requiring her to sit down. These episodes would last seconds without loss of consciousness. She denied any prior history of these episodes. She also denied headaches, numbness, tingling, loss of balance, focal weakness, fainting, incontinence, tongue biting, convulsions, loss of tone, or prior history of seizures. She did not have memory, hearing, vision, or balance problems. Except for a past medical history of hypertrophic cardiomyopathy as a complication of hypertension, hyperlipidemia, gastroesophageal reflux disease, and osteopenia, she was previously healthy. We did not identify any significant family history or personal history related to her presentation. A neurological exam showed no focal abnormalities. Given her symptoms of olfactory and gustatory auras, we suspected a diagnosis of a brain lesion, infection, or temporal seizures.

In magnetic resonance imaging (MRI) of her brain, an expansile T2 prolongation slice hyperintense FLAIR signal was seen in the anteromedial right temporal lobe, also involving the right frontal operculum and gangliocapsular structures *(Figure 1)*. This area of signal abnormality measured at least $5.6 \times 4.9 \times 5.7$ cm with no associated enhancement or hemorrhage. There was mild effacement of the right lateral ventricle and bowing of the septum pellucidum without overt midline shift. Scattered foci of T2 prolongation were seen in the periventricular and subcortical white matter, nonspecific but likely microangiopathic. No parenchymal enhancement, restricted diffusion, parenchymal susceptibility, or extra-axial fluid collection was seen. The imaging findings were representative of the features of a primary intracranial malignancy. Computed tomography of the chest,

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The authors report no conflicts of interest. The patient's spouse gave permission for publication of this case report.
Received July 30, 2021; Revised October 4, 2021; Accepted October 4, 2021.

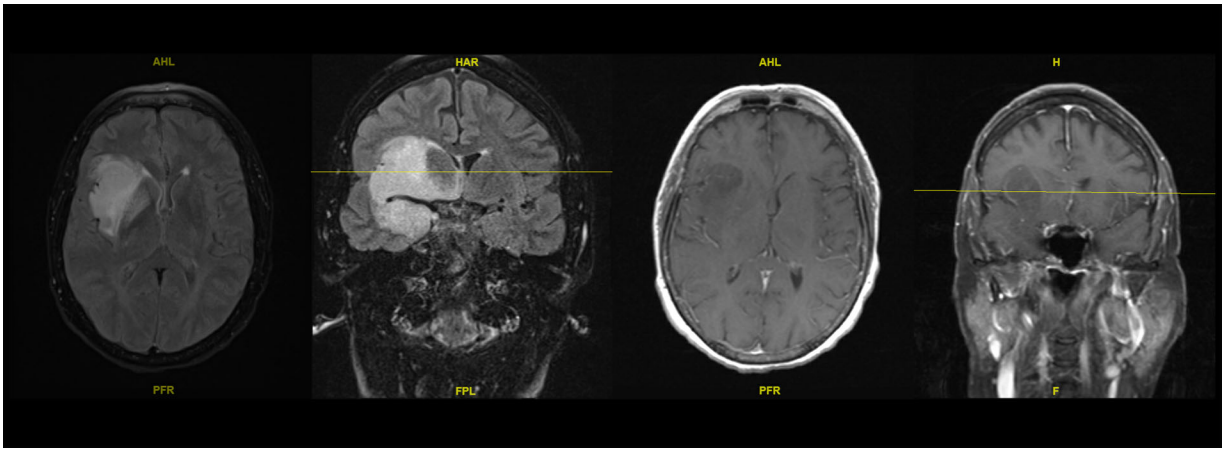

Figure 1. Brain MRI with T1 postcontrast (axial/coronal) and FLAIR (axial/coronal) showing expansile signal abnormality in the anteromedial right temporal lobe with associated involvement of the frontal operculum and gangliocapsular structures.

abdomen, and pelvis did not reveal evidence of metastatic disease.

The patient was started on Keppra for suspected seizure activity secondary to the organic finding on imaging. She was referred to neurosurgery and underwent right pterional craniotomy with subtotal resection given invasion of basal ganglia. Postoperative MRI showed partial resection with soft tissue changes with pneumocephalus and small bifrontal extra-axial fluid collections.

Pathology demonstrated a hypercellular glial neoplasm with mild to moderate nuclear pleomorphism, rare mitosis, and prominent vascular proliferation but no necrosis. Those changes are diagnostic for World Health Organization grade IV glioblastoma. IDH1 and IDH2 mutation detection was negative (wild-type). The tumor cells were positive for *P53* and *GFAP*, and the proliferation index of Ki67 was about 30%, further supporting the diagnosis. Cytogenetics revealed positive co-polysomy of 1q25 and 19p13 without deletion of 1p36 and 19q13. This finding correlates with a shorter survival and shorter progression-free survival.[1]

The patient had an uneventful recovery; phantosmia and dysgeusia resolved on Keppra. She was referred to neuro-oncology and it was agreed that she would undergo a full course of chemotherapy given her overall good health and excellent performance status despite her age.

DISCUSSION

High-grade gliomas commonly present with headaches, seizures, and focal neurological symptoms.[2] The presenting clinical symptoms are dependent on the tumor size and location. Patients typically experience progressive neurological symptoms that lead them to seek medical attention. It is rare for glioblastoma to present solely as abnormalities in olfaction and taste without any other neurological findings. While there does not appear to be any reported cases of glioblastoma presenting solely with these dual symptoms, there has been one report of dysphagia and phantosmia as a first presentation of anaplastic astrocytoma.[3] It is important to recognize that episodic changes in smell and taste can represent seizure auras, and these symptoms should prompt investigation for organic causes such as infection, neoplasm, or vascular events.

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