Spontaneous Resolution of a Confounding Insular Glioma

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Introduction

Insular gliomas were previously considered inoperable lesions, typically treated via biopsy or chemotherapy and/or radiation if not observation alone. Stereotactic biopsies of low grade insular gliomas can underestimate tumor grade, or fail to establish malignancy. Moreover, the survival advantages of maximal safe resection for insular gliomas are increasingly recognized. As such, initial surgical resection is increasingly performed. As with most lesions, a definitive diagnosis exists for apparent insular gliomas, with definitive diagnosis generally obtained upon resection. We report an illuminating case that presented similarly to an insular glioma undergoing malignant transformation, yet which resolved spontaneously following a non-diagnostic biopsy.

Case Report

A 53-year-old woman with no prior neurological history presented to the emergency department after an isolated episode of word finding difficulty and dizziness, followed by syncope and a reported 30-minute loss-of-consciousness. No tongue biting, bowl/bladder incontinence, or other sign of ictal activity was observed, and she recovered to her neurologic baseline. Medical history was significant for occasional nighttime headaches—severe enough to wake her from sleep—and chronic right-sided hearing impairment associated with prior chronic otitis media status post mastoidectomy. Physical exam was unremarkable, with no other focal neurologic deficits; family history was significant for malignancy in two second- and third-degree relatives.

Head CT demonstrated low attenuation changes suggestive of vasogenic edema in the left anterior perisylvian region, with loss of insular grey-white differentiation and ambiguous mass effect. Follow-up MRI demonstrated a 3.5cm expansile, T2-hyperintense lesion involving most of the left insula and extending into the anterior left temporal lobe, within which was noted a 12mm region of contrast enhancement and central necrosis. Imaging findings were felt most consistent with high-grade glioma. Level I etanercept 750 mg BID PO and Dexamethasone 1mg q4h PO were initiated for seizure prophylaxis and perilisal edema, and a stereotactic biopsy was subsequently performed.

Intraoperatively, the focus of maximal enhancement was targeted, and the operation proceeded uneventfully. Final pathology identified reactive gliosis and diffuse, non-specific histopathologic abnormalities, without definitive neoplastic features. Systemic work-up was unremarkable, including blood counts, serum C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), echocardiogram, and CSF analysis. Microbiology testing of the biopsy specimen was also negative, including Gram, mycobacteria, Nocardia, fungi, and acid-fast bacillus stain. The case was presented at multi-disciplinary conference, at which the patient was recommended for awake craniotomy in the intra-operative MRI suite to obtain tissue diagnosis, and potentially proceed with maximal safe resection, as indicated. The patient consented to awake craniotomy in the intra-operative MRI suite. However, the preoperative MRI failed to demonstrate the substantial previously observed insular T2 hyperintensity, despite new significant FLAIR signal surrounding the needle tract. The prior area of contrast enhancement was similarly greatly reduced.

Given these imaging findings, the possibility of an insular glioma was felt now to be decided unlikely. Although the still-awake patient was prepped in pins for the procedure, a mutual decision was reached to abort the case. All further studies failed to establish a definitive diagnosis; however, in light of what was subsequently determined to be extremely poor dentition and a history of recent self-extraction of a tooth, as well as a small central focus of diffusion-restriction on the original MRI, abscess was thought to be the most likely diagnosis. The patient elected to observe the lesion, and follow-up MRI at 3 months demonstrated further resolution of the lesion with overall restoration of normal regional anatomy. As of last follow-up at 6 months, the patient has remained completely asymptomatic with no evidence of ictal activity, paroxysmal spells, or other neurologic abnormalities; continued radiographic stability, and no clinical event indicating a diagnosis.

Discussion

In terms of the generalizability of this case to the neurosurgical readership, our experience with this patient has not prompted us to change practice overall practice patterns in our institution; we continue to advocate for early surgical intervention for most patients with a significant clinical suspicion for glioma. Nevertheless, in the setting of increasingly favored surgery for insular gliomas at institutions worldwide, we felt this case to be an excellent and timely reminder of the imperative to critically scrutinize imaging findings, and thoroughly explore all aspects of the clinical history. Moreover, in all such neuro-oncologic cases, a forthright discussion with the patient regarding diagnostic uncertainty must complement the surgical informed consent.

Conclusions

Our case illustrates the complexity of managing insular lesions and highlights potential for alternate treatment options that can mimic insular glioma. Additionally, it provides a humbling reminder that, even in the presence of seemingly pathognomonic imaging findings, a differential diagnosis of insular lesions must be thoughtfully considered in patient counseling and pre-surgical planning.

References


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Figure 2: Axial magnetic resonance imaging suggestive of insular glioma, with concerning features including confluent, anatomically restricted fluid-attenuated inversion recovery hyperintensity (arrow) and nodular central enhancement with probable areas of internal necrosis (broad arrow).

Figure 3: Magnetic resonance imaging demonstrating complete resolution of previously noted contrast enhancement, with minimally reduced region of insular T2 hyperintensity (arrows). No teca, a strip of expected T2 hyperintensity was noted surrounding the needle tract, excluding a technical error underlying the signal change at the lesion (broad arrow).

Figure 5: Magnetic resonance imaging demonstrating further lesion resolution with a return to near-normal regional anatomy.