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Case Report

Surgical management of an abscess of the insula

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Received : 20 September 2022
Accepted : 07 December 2022
Published : 23 December 2022

DOI
10.25259/SNI_871_2022

Quick Response Code:

ABSTRACT

Background: Mass lesions within the insular are diagnostically and surgically challenging due to the numerous critical cortical, subcortical, and vascular structures surrounding the region. Two main surgical techniques – the transsylvian approach and the transcortical approach – provide access to the insular cortex. Of the range of pathologies encountered, abscesses in the insula are surprisingly rare.

Case Description: A 34-year-old patient was admitted for surgical resection of a suspected high-grade glioma in the insula of the dominant hemisphere. A rapid clinical decline prompted emergent neurosurgical intervention using a transsylvian approach. Surprisingly, abundant purulent material was encountered on entering the insular fossa. Pathological analysis confirmed an insular abscess, although a source of infection could not be identified. The patient required a second evacuation for reaccumulation of the abscess and adjuvant corticosteroids for extensive cerebral edema.

Conclusion: An abscess located in the insular cortex is an incredibly rare occurrence. Surgical management using the transsylvian approach is one option to approach this region. Familiarity with this approach is thus extremely beneficial in situations requiring emergent access to the dominant insula when awake mapping is not feasible. In addition, treatment of abscesses with adjuvant corticosteroids is indicated when extensive, life-threatening cerebral edema is present.

Keywords: Brain abscess, Infection, Insula, Transsylvian approach

INTRODUCTION

Brain abscesses are life-threatening infections within the brain parenchyma. The infection begins as a localized area of cerebritis before rapidly developing into a mass lesion composed of an encapsulated collection of pus with surrounding edema. Early diagnosis and treatment are imperative to reduce the risk of mortality, which reaches a rate of approximately 20% at 1 year.[1] Initial diagnosis may be difficult, however, as few patients present with the classic symptom triad of headache, fever, and focal neurological deficits.[2,3] Instead, patients can present with a range of symptoms including signs and symptoms of increased intracranial pressure such as altered level of consciousness, confusion, nausea, and vomiting – symptoms common to numerous intracranial pathologies.[2] While radiographic imaging helps localize space-occupying lesions, it may not always prove definitive. For example, the differential of a mass lesion visualized on computed tomography (CT) or magnetic resonance image (MRI) is relatively lengthy and includes abscess, hematoma, metastasis, glioma, granuloma, and radionecrosis. The diagnostic

ability of radiographic imaging is particularly poor for lesions in the insula.[4,5] This anatomically complex cortical structure forms the floor of the lateral sulcus and is hidden from view by the frontal, parietal, and temporal opercula. Here, we present a case of an insular mass lesion that initially masqueraded radiographically as a high-grade glioma. We review the management steps taken in a patient *in extremis* and discuss more broadly the approaches for abscess in this location.

CASE REPORT

A 34-year-old right-handed woman with diabetes mellitus type two, obesity class two, and a history of migraines presented to the emergency department after a self-resolving 5 min episode of paresthesias in her right hand, arm, and face. She also noticed a foul taste in her mouth reminiscent of French onion soup. These symptoms occurred in the setting of a 7-day unremitting headache worsened by bright lights. While in the emergency department, she experienced a second episode of paresthesias and was given lorazepam and levetiracetam for suspected focal sensory seizures. She had stable vital signs and her physical examination was unremarkable aside from trace right facial asymmetry.

A head CT demonstrated a hyperattenuating focus in the left insular region surrounded by an area of low attenuation, suggestive of vasogenic edema [Figure 1]. A subsequent brain MRI showed prominent edema surrounding a 1.9 cm mass with central T1 hypointensity, heterogeneous T2 hyperintensity, and irregular peripheral enhancement [Figure 2a]. The treatment team felt that the radiographic images were most consistent with a malignant etiology. An abscess was considered less likely given a relative lack of bright signal on diffusion-weighted imaging and the absence of low apparent diffusion coefficient values on MRI. The neuroradiologist concurred with the treatment team's suspicion, noting primary or metastatic brain neoplasm as leading considerations in the radiology report while indicating an abscess as less likely. This was further supported by the absence of infectious symptoms in the patient's clinical history and a white blood cell count only mildly elevated to 11.8, a normal erythrocyte sedimentation rate of 18 mm/h, and a normal C-reactive protein of 6.2 mg/L. There was no evidence of a primary extracranial tumor on an abdomen, pelvis, or chest CT to suggest metastasis, thus a primary brain neoplasm was suspected.

The patient was transferred to Massachusetts General Hospital (MGH) for the management of a presumed left insular tumor. Several days following her admission to MGH and before a planned awake left craniotomy, she developed expressive aphasia and intermittent disorientation to time and place. An urgent CT demonstrated enlargement of the mass to nearly 5.0 cm. Given the rapid rate of change in the size of the mass, we suspected that the insular lesion was an abscess and thus obtained a brain MRI which showed the mass to be peripherally enhancing and now obviously restricting diffusion. Her cerebral edema had also significantly worsened, causing a 2 mm midline shift to the right and mass effect on the left lateral ventricle and basal ganglia [Figure 2b]. Soon thereafter, the patient became acutely obtunded and bradycardia. Despite receiving mannitol and hypertonic saline, her examination continued to decline, and she was emergently taken to the operating room (OR) for neurosurgical intervention.

Because the patient was herniating, a decompressive left frontotemporal craniectomy was planned followed by a transsylvian approach to access and drain the presumptive insular abscess.

After exposure of the Sylvian fissure, we found that inflammatory phlegmon had layered over the entire fissure and surrounded the Sylvian veins. After much exploration, a small arachnoid window was identified and opened in the distal aspect of the fissure as we followed an M4 branch into the fissure. This opening was carried anteriorly and deepened, and as we dissected between the M2 branches, a rush of purulent fluid was encountered. A sample of this material was sent for aerobic and anaerobic bacterial, fungal,

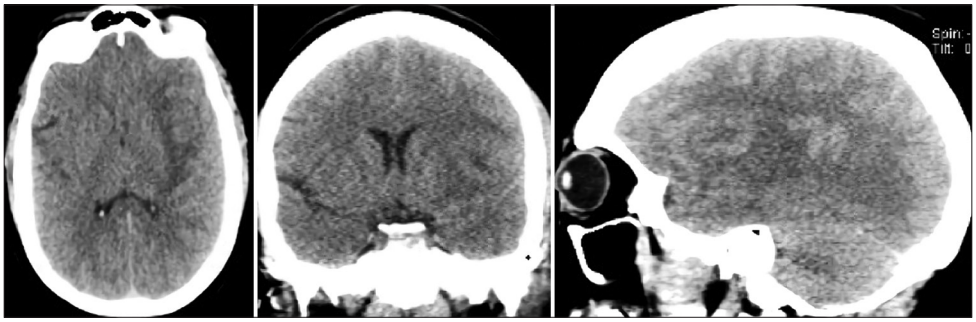

Figure 1: Axial (left), coronal (middle), and sagittal (right) CT scans on admission showing an area of low attenuation with a central hyperattenuating focus in the left frontal lobe causing mass effect on the frontal horn of the left lateral ventricle and rightward 2 mm midline shift.

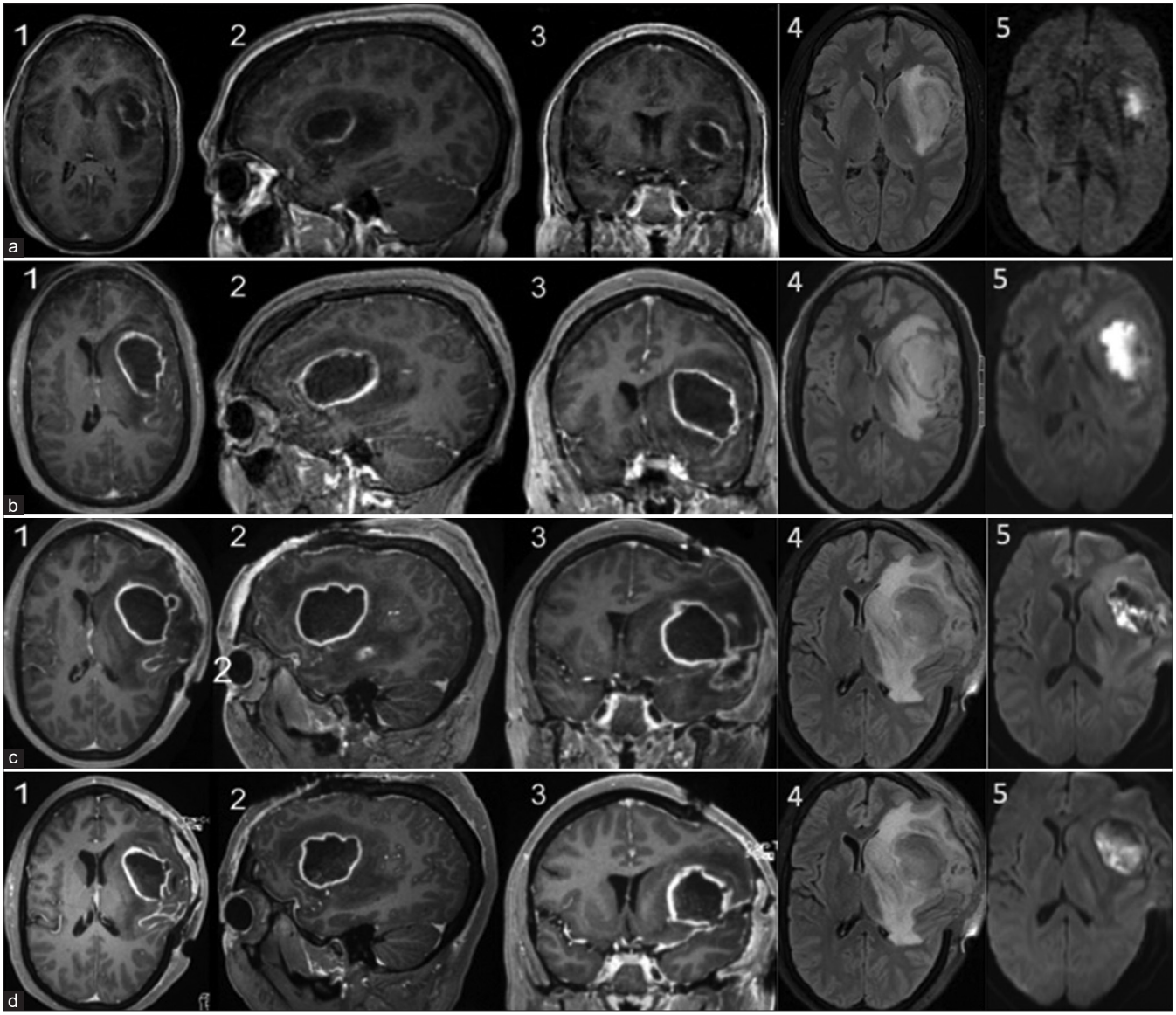

Figure 2: Serial brain magnetic resonance imaging (MRI) scans performed throughout the patient's hospital course: (a) on admission to MGH showing left insular mass with irregularly marginated peripheral enhancement with central necrotic and hemorrhagic foci causing mass effect and a 2 mm midline shift to the right. Fluid-attenuated inversion recovery (FLAIR) imaging demonstrates surrounding edema and diffusion-weighted imaging (DWI) demonstrates restricted diffusion in the center of the lesion. (b) Following acute clinical decline before first surgery revealed an interval increase in the left insular lesion with more well-defined peripheral enhancement. Mass effect has progressed causing increased rightward midline shift. FLAIR imaging shows increased peripheral edema. (c) Postoperative demonstrating a mildly decreased size in the lesion. There is a loculated rim-enhancing component along the superolateral margin of the resection cavity and a focus of apparent discontinuous rim enhancement along the inferolateral posterior aspect of this loculation. (d) Surveillance MRI on post operative day 7 shows an expansion of the previously partially collapsed appearance of the abscess cavity walls. Extensive FLAIR hyperintensity with mass effect surrounding the abscess cavity has increased causing increased extracranial herniation and left to right midline shift. The images show T1 postcontrast axial (labeled as 1), sagittal (labeled as 2), and coronal (labeled as 3); axial FLAIR (labeled as 4); and axial DWI (labeled as 5).

and mycobacterial testing. The cavity in the insula was then entered, and the remaining purulence was evacuated. After irrigating the region, friable inflammatory-appearing tissue was noted surrounding the cavity walls. The patient's bone flap was left off to allow for maximal brain decompression. She was started on vancomycin, cefepime, and metronidazole for the treatment of an intracerebral abscess. A postoperative head CT showed excellent brain decompression with a reduction of the midline shift and resolution of herniation. A loculated rim-enhancing component along the superolateral margin of the dominant abscess cavity seen on postoperative MRI was suspicious for residual abscess [Figure 2c].

Culture of the purulent material grew *Streptococcus intermedius* – a bacterium normal to the oral cavity and upper respiratory tract flora. An odontogenic infection was investigated, but a dental examination showed no clinical or radiographic evidence of acute or chronic intraoral infections. There was no history of dental infections or dental procedures other than routine cleaning and no oral piercings. Investigation into other possible sources also failed to find evidence of infection: (1) no valvular vegetations on transthoracic echocardiogram, (2) no pulmonary arteriovenous malformations seen on chest CT, (3) negative blood cultures, (4) negative human immunodeficiency virus antigen/antibody tests, and (5) no past medical history, family history, or examination findings suggesting hereditary hemorrhagic telangiectasia.

After 1 week of antibiotics, a surveillance brain MRI on POD 7 showed a likely recurrence of the abscess with rightward midline shift progression, worsening parenchymal herniation through the craniectomy defect, and subfalcine and uncal herniation [Figure 2d]. The patient was brought back to the OR for repeat transsylvian aspiration and extension of the craniectomy to allow for increased brain decompression. Intraoperatively, the brain was noted to be swollen with extensive inflammatory debris in the fissure. A rush of purulent material was again encountered on accessing the cavity. After evacuation of the reaccumulated abscess material, several locations of the abscess wall were sampled due to concerns about a potential underlying malignancy. Before closing, the craniotomy was extended posteriorly to create additional space for brain herniation. Notably, the sampled insular abscess cavity fluid was sterile and did not yield growth in cultures, reflecting the early effectiveness of the antibiotic regimen.

A postoperative head CT showed a collapsed abscess cavity and decreased parenchymal herniation through the craniectomy site. Histologic examination showed portions of brain with abundant necrosis and a dense neutrophilic infiltrate, consistent with abscess and excluding a superimposed underlying malignancy [Figures 3a and b]. Surrounding the necrotic regions were areas of granulation tissue and granulation-type tissue indicative of early formation of a capsule [Figures 3c and d]. Brown-Hopps and GMS stains were negative for microorganisms.

One week after surgery, head CT revealed a re-expansion of the previously collapsed abscess cavity, significant edema with mass effect, and a subcutaneous hematoma [Figure 4a]. Surgery was deemed imprudent and she was started on dexamethasone for medical management of cerebral edema and as an attempt to decrease the production of presumed sterile inflammatory fluid. Serial head CTs showed progressive reduction in the volume of the abscess cavity and slow resolution of the cerebral edema.

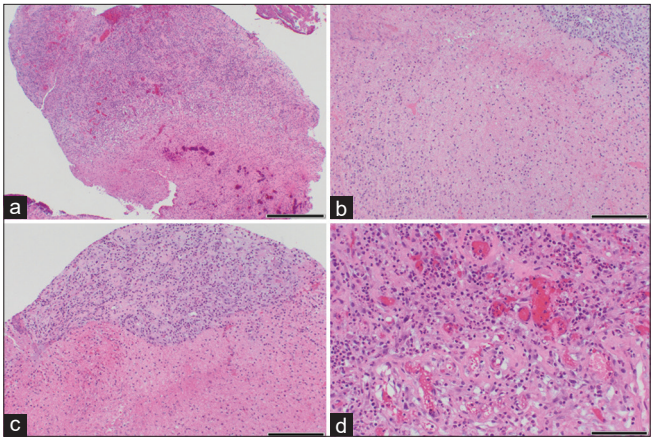

Figure 3: Hematoxylin and eosin-stained images show abscess with capsule (a), regions of necrosis (b), regions with early capsule formation (c), and granulation tissue with neutrophilic infiltrate (d). Scale bars denote 500 um (a), 200 um (b and c), and 100 um (d).

The patient continued to improve and was discharged home on steroids and antibiotics. She ultimately completed 1 month of steroid therapy and 6 weeks of oral metronidazole and penicillin G. She continues to be followed outpatient and has returned to her neurologic baseline. A 2-month follow-up head CT demonstrated improving cerebral edema and no evidence of a persistent or recurrent abscess [Figure 4b]. Her most recent follow-up MRI showed substantially decreased brain herniation and edema [Figures 4c-e].

DISCUSSION

The insular cortex is a rare location for cerebral abscesses with very few cases published in the literature. The most common locations for cerebral abscesses are, of decreasing frequency: the frontal and temporal lobes; frontal-parietal region; parietal lobe; cerebellum; and occipital lobe.[6] Bacteria usually enter the brain by contiguous spread of infections (e.g., chronic otitis media, mastoiditis, and sinusitis) or hematogenous seeding from infections of distant sites (e.g., cardiac valve and pulmonary infections).[1-3] However, an estimated 10–30% of patients present with cryptogenic brain abscesses where the source of the infection remains unknown.[7,8]

In this case, the initial imaging findings were more consistent with a high-grade neoplasm, and thus, we had prepared to perform an awake left craniotomy for tumor resection using neuromonitoring. Once the patient's condition declined rapidly and the imaging more clearly demonstrated an abscess etiology, our clinical decision-making changed. If the patient was clinically stable, stereotactic needle drainage would have represented a very reasonable option. However, the patient's clinical herniation compelled us to proceed with a more aggressive approach to decompress the left hemisphere and perform a microsurgical evacuation.

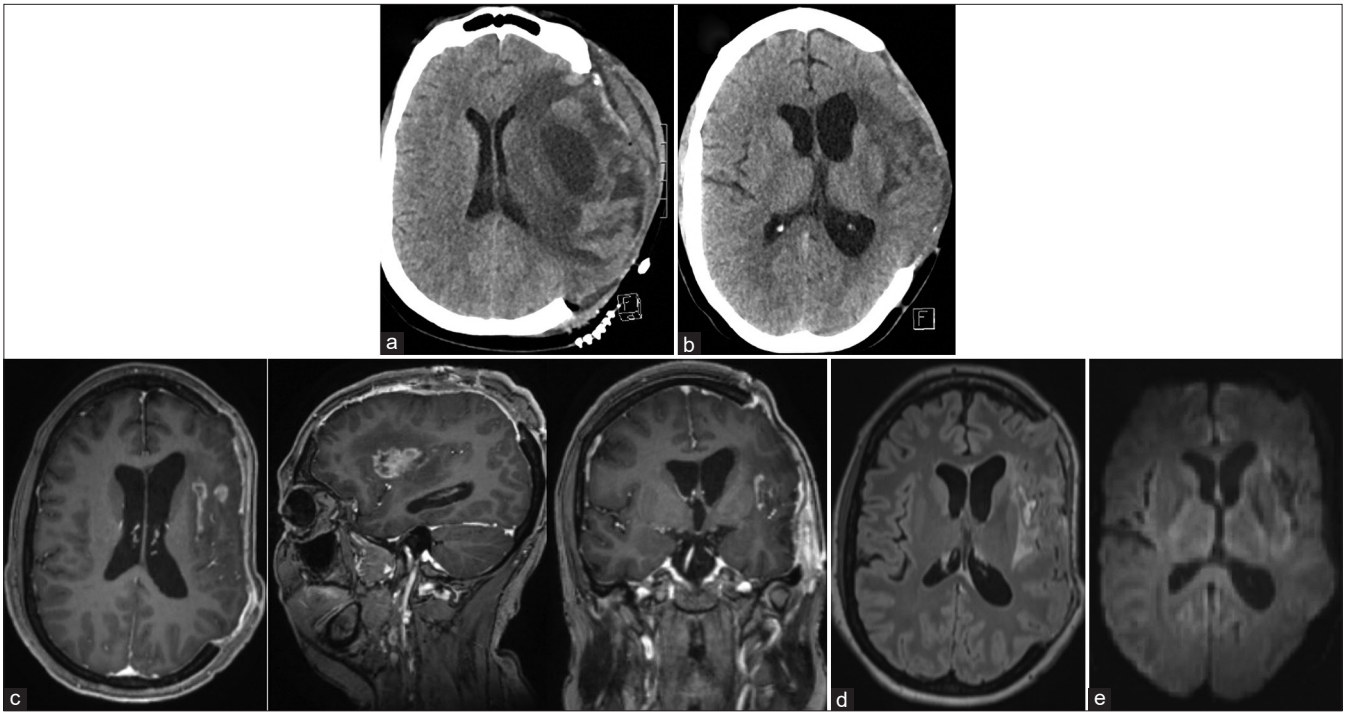

Figure 4: (a) Postsurgical changes from left-sided craniectomy with extensive edema of brain parenchyma causing herniation of the left cerebral hemisphere through the craniectomy defect, minimal rightward midline shift, and effacement of the left lateral ventricle. (b) A follow-up scan 2 months after discharge shows improving cerebral edema and no abnormal enhancement to suggest persistent or recurrent abscess. T1 postcontrast axial, sagittal, and coronal (c), axial fluid-attenuated inversion recovery (d), and axial diffusion-weighted imaging (e) sequences from her most recent follow-up MRI taken 3 months after discharge shows substantially decreased brain herniation and swelling of the left cerebral hemisphere with residual enhancement within the insula and mild residual FLAIR hyperintensity involving the left frontal lobe, insula, and left temporal lobe.

Standard treatment

Intracranial abscesses are treated with surgical drainage followed by long-term antibiotic treatment.[9,10] Surgical drainage is preferably accomplished by stereotactic needle aspiration.[9-12] If needle aspiration fails to decompress the abscess or if the abscess recurs, surgical excision of the abscess may be required.[9-12] In addition, surgical excision is indicated and is the initial treatment of choice if the patient has clinical or radiographic signs of increased intracranial pressure.[9-12] Drainage of the abscess may be delayed or not required in certain circumstances, such as if the abscess is <2.5 cm or if it occurs in the setting of bacteremia.[9-12]

Steroid administration is often avoided in patients with cerebral abscesses out of concern for worsened patient outcomes.[13,14] While studies have demonstrated that steroids suppress the efficacy of antibiotics and inhibit the migration of leukocytes,[15] a meta-analysis showed that adjuvant dexamethasone in the treatment of cerebral abscesses was not associated with increased mortality and withholding steroids did not provide a mortality benefit.[14] Furthermore, other studies have demonstrated that adjunctive steroids actually improve clinical outcomes in patients with severe cerebral edema.[16,17] This is likely because steroids have anti-inflammatory properties capable of ameliorating brain swelling and inflammatory secretions contributing to life-threatening intracranial pressures.[14,15] Although data from randomized studies are lacking, current recommendations advocate using adjuvant steroids in patients with abscesses and depressed mental status with significant mass effect on imaging.[17,18,20]

Surgical approaches

Neurosurgical procedures involving the insular cortex can be challenging. Situated in the depths of the Sylvian fissure, the insular cortex is covered by the frontal, temporal, and parietal lobes and hidden under a dense network of critical arterial and venous blood vessels.[19] Directly medial to the insula lie basal ganglia structures, including the extreme capsule, claustrum, external capsule, putamen, and internal capsule.[19] Surrounding the insula are several white matter tracts involved in language, speech, and motor functions.[19] The two main surgical approaches to the insular cortex are the transsylvian and transcortical (TC) routes. There are advantages and disadvantages to these techniques, and

in many cases, surgeon preference and comfort level may drive the choice of approach. Lesion location within the insula also influences surgical strategy. The Berger-Sanai classification system divides the insula into four zones based on perpendicular bisecting lines through the Sylvian fissure and the foramen of Monro [Figure 5].[20,21] Zone 1 lies anterior to the foramen of Monro and superior to the Sylvian fissure, zone 2 is posterosuperior, zone 3 is inferoposterior, and zone 4 inferoanterior.[20,22] Classification of lesions to one or more of these zones informs the surgeon of relevant anatomy, such as the peri-Sylvian language network in association with zones 1–3, the primary motor and sensory cortices with zone 2, the primary auditory cortex with zone 3, and the lenticulostriate arteries with zone 4.[21] The extent of resection and functional outcomes can also be predicted using the Berger-Sanai classification system.[20,22] Regardless, it is helpful for the cranial surgeon to be facile with at least one technique to access the insula microsurgically when needed, knowing that integrating several approaches may also be needed.

The TS approach, involves splitting the Sylvian fissure and meticulous dissection of the Sylvian and peri-Sylvian vessels.[23] Different approaches toward splitting the Sylvian fissure afford different final anatomical exposures. The anterior TS approach requires a standard pterional craniotomy. Dissection begins where the distal Sylvian fissure is widest, typically below the pars triangularis region of the inferior frontal gyrus.[24] The dissection proceeds from distal to proximal along the middle cerebral artery's (MCA) sphenoidal (M1) and insular (M2) segments.[24] The frontal lobe is separated from the temporal lobe by following a cortical artery to create a natural separation plane.[24] The dissection continues by following opercular arteries down to insular arteries to develop the plane further, eventually exposing the MCA bifurcation, limen insulae, and short gyri of the insula.[21-24] The posterior TS approach requires a larger pterional craniotomy that incorporates additional parietal and posterior temporal bone to allow additional exposure of the posterior angular gyrus.[21,25] The dissection typically begins below the pars triangularis, similar to the anterior approach, but proceeds in the opposite direction, proximal to distal along the opercular (M3) segment of the MCA.[24] The opercular surfaces of the frontal, parietal, and temporal lobes are separated, and the dissection continues along opercular arteries to expose the circular sulcus and long gyri of the insula.[21-25]

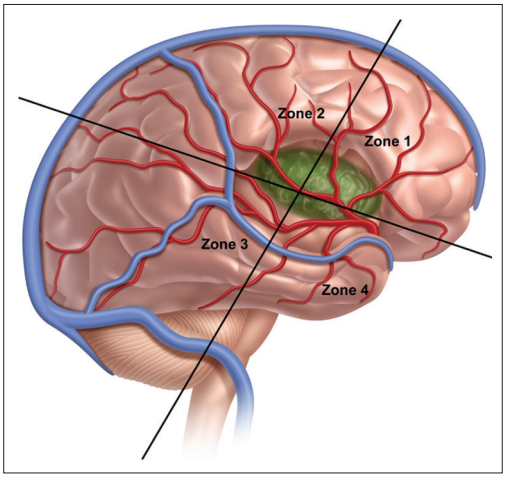

Figure 5: A representative illustration of the abscess through the transsylvian approach in relation to the four insular zones. The transsylvian approach preserves the overlying eloquent cortex in the frontal and temporal lobes but requires working between critical vascular structures including branches of the MCA and superficial Sylvian vein. The Berger-Sanai classification separates the insular cortex into four zones using perpendicular lines through the Sylvian fissure and the foramen of Monro. Zone 1 is above the Sylvian fissure and anterior to the foramen of Monro. Zone 2 is superior to the Sylvian fissure and posterior to the foramen of Monro. Zone 3 is inferior to the Sylvian fissure and posterior to the foramen of Monro. Zone 4 is inferior to the Sylvian fissure and anterior to the foramen of Monro.

Several critical vascular structures traverse and surround the Sylvian fissure and insula. Veins within and overlying the Sylvian fissure are preferably preserved as coagulation can increase venous pressure in the remaining Sylvian veins, particularly in patients with poor collateral outflow through the superior sagittal sinus or vein of Labbé.[23,26] If preservation limits surgical exposure, some sacrifice may be required to enable sufficient opening of the fissure but should only be done so judiciously.[23] Long perforating arteries arising from M2 branches of the MCA should also be preserved. These arteries supply the corona radiata and inadvertent coagulation may result in postoperative hemiparesis.[27-29] Conversely, the many small perforating vessels on the undersurface of M2 ought to be coagulated as the parent M2 vessel is at risk should these be torn.[29] The manipulation of these perforating vessels, however, puts the parent M2 vessel at risk for postoperative vasospasm.[23-29] Finally, frontal lobe ischemia may follow opercula retraction due to compression of the M3 branches coursing along the medial surface of the opercula.[21,27-31]

Eloquent cortical regions involved in speech, language, and motor functions also surround the operative corridor.[24,31] Deep to the posterior segment of the superior peri-insular sulcus, corticospinal tract motor fibers converge to form the posterior limb of the internal capsule.[26,31] Superiorly, the pars opercularis and pars triangularis within the inferior frontal gyrus of the dominant hemisphere correspond to Broca's area.[31,32] Inferiorly, the superior temporal gyrus

contains Wernicke's area.[31,32] The arcuate fasciculus, a bundle of axons connecting Wernicke's area with Broca's, lies just beneath the frontal and parietal opercula and surrounds the insular cortex.[31,32] Other spatially associated fasciculi involved in language processing are the inferior fronto-occipital fasciculus (IFOF) occupying the posterior two-thirds of the temporal lobe stem and the uncinate fasciculus running below the superior peri-insular sulcus.[24,31,32] It is recommended to perform this approach awake if on the dominant side and to also incorporate subcortical stimulation regardless of the side due to the proximity to the corticospinal tract.

TC approach

The TC approach involves performing frontal, parietal, or temporal operculum corticectomies to create windows of access to the insular cortex.[33] Different combinations of opercular routes with the creation of multiple cortical windows, which are then connected at the level of the resection cavity, may be required for large insular lesions.[33] Using this technique, the TC is able to provide a larger exposure of the insula than the TS approach while still preserving the bridging superficial Sylvian veins.[31] For this reason, the TC approach is particularly useful for large and posterior insular lesions as well as those that have a substantial superior extent.[33]

To ensure parenchymal transgression occurs through functionally silent cortex, the TC approach requires intraoperative cortical and subcortical mapping to locate regions critical for speech, language, motor, and cognitive function.[28,31] During insular surgery of the dominant side, intraoperative mapping is particularly important for identifying eloquent cortex involved in speech and language. Subcortical mapping is also used to locate the corticospinal tract within the internal capsule and language tracts such as the arcuate fasciculus and the IFOF.[31,32]

Intraoperative cortical and subcortical stimulation is not entirely infallible and carries its own risks. Direct cortical stimulation can cause intraoperative seizures,[34,35] which may prohibit further cortical mapping and cause termination of the procedure. Postoperative seizures are another risk of direct stimulation and contribute to extended hospital stays and increased morbidity.[35] Afterdischarges (ADs), defined as stimulus-provoked repetitive epileptiform discharges, can also occur.[36] The clinical implications of ADs are poorly understood, but they may cause errors in functional mapping during surgery.[35] The accuracy of functional mapping is also influenced by patient participation. For example, mapping language and speech requires intraoperative assessments of verbal responses, which are significantly influenced by the patient's level of consciousness and cooperativeness.

The transsylvian and TC approaches may also be combined in certain instances, particularly in cases of large insular masses and those that extend into adjacent brain structures, in which aspects of the target are best accessed by a hybrid approach.

CONCLUSION

This case demonstrates the diagnostic and surgical challenges of a mass lesion within the insula. Gliomas are relatively common in the insular lobe and paralimbic region – up to 25% of low-grade and 10% of high-grade gliomas are found in this region.[36] However, it is important to maintain a broad differential when assessing these patients. As demonstrated in our case, nonneoplastic conditions in the confined space of the insula can mimic gliomas on radiographic imaging. Particularly during the late cerebritis or early capsule formation stages, an abscess may present with radiographic features characteristic of glial tumors, such as irregular enhancing margins on T1-weighted imaging surrounding a hypointense mass. In addition, patients may not present with overt infectious symptoms to raise suspicion of an infectious process.

All patients with cerebral abscess are treated with long-term antibiotics which are often initiated after stereotactic needle aspiration or, less preferably, before if drainage of the presumed abscess is delayed. Open surgical evacuation becomes necessary when there are signs of increased intracranial pressure, a progressive increase in the abscess diameter, or abscess material reaccumulates despite needle aspiration attempts. Adjuvant corticosteroids are warranted when extensive cerebral edema – or reaccumulating sterile inflammatory fluid – is present.

Surgical access to insular abscesses or other insular pathologies is challenging due to numerous critical structures within and surrounding this region: the frontal, parietal, and temporal opercula contain eloquent cortex necessary for speech and motor functions; surrounding the insula are fasciculi involved in speech and directly medial to the insula are basal ganglia structures; and overlying and coursing through the insula are critical vessels supplying the insula, cortex, and basal ganglia.

Access to the insula can be achieved through the TS or TC approach. In the TS approach, the surgeon splits the Sylvian fissure and follows branches of the MCA, creating a dissection plane along the opercula to the insular cortex. While this approach offers limited surgical exposure, intraoperative cortical mapping is not imperative, and it can, therefore, be used during emergent situations. Conversely, the TC can provide greater surgical exposure but requires intraoperative cortical and subcortical mapping to ensure that corticectomies are performed through the functionally silent cortex.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Leavitt L, Baohan A, Heller H, Kozanno L, Frosch MP, Dunn G. Surgical management of an abscess of the insula. Surg Neurol Int 2022;13:591.