Cysticercal Brain Abscess: A Distinct Entity Mimicking Pyogenic Brain Abscess.

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Abstract

Cysticercal brain abscess is a rare entity with the clinical presentation and radiological findings closely mimicking a pyogenic abscess. We report three cases of cysticercal abscess presenting as solitary lesion in the brain with radiological appearance like an abscess. All the patients underwent excision of the lesion with histopathological diagnosis of cysticercal brain abscess. The clinical outcome was satisfactory after surgical excision. Cysticercal abscess, though rare, should be considered as a differential diagnosis of a solitary rim-enhancing lesion, mainly if the patients belong to the endemic zone of neurocysticercosis. Surgical excision with or without a short course of the anti-parasitic agent is the treatment of choice.

Highlights:

1: Cysticercal brain abscess is a rare entity.

2: We reported three cases of the solitary lesion in the brain and the radiological appearance like the abscess. Histopathology revealed cysticercal brain abscess in all three cases. The clinical outcome was satisfactory after surgical excision.

3: Surgical excision with or without a short course of the anti-parasitic agent is the treatment of choice.

Introduction:

Neurocysticercosis (NCC) is a common parasitic infection of the central nervous system. The incidence of NCC is high in endemic countries like India and is increasing in developed countries due to the high immigration rate (Sinha and Sharma 2009; Rajshekhar 2010; Gripper and Welburn 2017). It has a varied clinical presentation based on its location in the brain and spine and the host response. It is the most common cause of acquired epilepsy in developing countries. Brain “abscess” caused by NCC is an exceedingly rare presentation and not included in the pathological stages of NCC described by Escobar et al. that consists of the colloidal, colloidal-vesicular, granular-nodular, and nodular-calcified stages (Escobar 1983). It closely mimics its more common counterpart, pyogenic abscess, both radiologically and histologically, posing a diagnostic dilemma. Distinction is crucial on account of treatment implications. We report three cases of neurocysticercal brain abscess with clinico-radiologic features, its histopathology and discuss the possible pathogenesis of this distinct entity.

Case 1:

A 51-year-old lady presented with a history of complex partial seizures for four months. There was no headache, vomiting, fever, or limb weakness. She had diabetes, hypertension as well as hypothyroid, for which she was on regular treatment. MRI brain (plain and contrast) revealed a solitary lesion in the left temporal lobe that was hypointense on T1WI, hyperintense on T2WI with peripheral ring enhancement (Fig. 2). A radiological diagnosis of high-grade glioma in the left side temporal lobe was considered, and

she underwent left-sided temporal craniotomy and excision of the lesion. The lesion was well defined, cystic, minimally vascular, and on the opening of the cyst wall, yellowish pus-like material was evacuated.The lesion was completely excised. At discharge, she was well with no motor or sensory deficits.

Histopathology revealed an inflammatory lesion composed of multiple abscess cavities with distinct serpiginous areas of necrosis lined by a palisade of epithelioid histiocytes. The surrounding inflammatory granulation tissue was composed of neutrophils, lymphocytes, plasma cells, macrophages, and eosinophils. This was in turn surrounded by dense fibrous bands encapsulating the lesion, as the layers of a typical pyogenic abscess. Within one of the abscess cavities, a remnant of degenerating cysticercal cyst was found, with its outer cuticular layer, resting on a middle cellular layer and innermost reticular layer replete with excretory ducts (Fig. 1). The cysticercal cyst was in the early degenerating colloidal stage, and its detection aided accurate diagnosis. No calcification was evident in the cyst or adjacent cyst wall.

The “pus-like” material aspirated from the cavity during surgery was submitted for aerobic and anaerobic bacterial cultures, both of which were negative. Following the histopathology report, the patient was evaluated with CT chest & abdomen, and stool exam) to look for other cysticercal cysts, but all were negative. Ophthalmological examination was also negative.The patient was managed with antiepileptic medication. Anti-parasitic medication was not given as the cyst was completely excised, and routine perioperative doses of antibiotics were given. Steroids were not prescribed as the patient had uncontrolled diabetes.

At three months follow up she was stable with no new focal neurological deficits. She was ultimately seizure-free, and the anti-epileptics were slowly tapered and stopped. Repeat MRI (plain and contrast) revealed no recurrence or appearance of new lesions. At six months follow up, she remains well and has resumed all activities of daily living.

Case 2:

A 40-year-old lady presented with generalized tonic-clonic seizures for ten years which was partially controlled with anti-seizure medications. Motor and sensory examination did not reveal any deficits. CT scan (plain and contrast) revealed a right frontal hypodense lesion involving the middle frontal gyrus with ring enhancement on contrast. No calcification was noted (Fig. 3). She underwent right frontal craniotomy and excision of the abscess cavity. The content of the cavity was xanthochromic with a thin, flimsy membrane.

Histology revealed a chronic abscess cavity rimmed by several multinucleated foreign body giant cells with the cavity containing a degenerating cysticercal cyst (Fig. 1). The surrounding florid inflammatory tissue was rich in eosinophils. The content of the abscess cavity was sterile on bacterial culture. She recovered well from surgery with no motor or sensory deficits and was discharged on day five. A postoperative CT scan revealed complete excision of the lesion. Anti-parasitic medications were advised

for two weeks. Anti-seizure medications were given for two years and slowly tapered off. At four years follow up, she remains seizure-free with no recurrence.

Case 3:

A 33-year-old lady presented with focal motor seizure involving the right upper limb with secondary generalization for one month. She was started on anti-seizure medication, but for the last seven days, she developed repeated episodes of right focal motor seizures with postictal right upper limb weakness and deviation of the angle of mouth. There was no history of fever, headache, or vomiting. CT scan (plain + contrast) revealed a well-defined hypodense lesion, measuring 2x2x2.5cm, involving the left posterior frontal region with peripheral rim enhancement associated with perilesional edema and mass effect (Fig. 4). She underwent left frontal craniotomy and complete excision of the lesion. The lesion was well defined and cystic with the content being yellowish pus-like material.

Histology revealed an abscess wall with a stellate outlines containing necrotic debris along with remnants of a degenerating cysticercal cyst. The abscess wall showed mixed inflammatory granulation tissue with few foreign body giant cells and eosinophils. The outermost layer showed dense gliosis (Fig. 1). The bacterial cultures were sterile. Postoperative CT scan showed complete excision of the lesion. She recovered well from surgery and was discharged on day four with residual weakness of the right upper limb. At three months follow up, her weakness resolved completely, and she was seizure-free with no recurrence of symptoms. Albendazole was given for two weeks, and anti-seizure medications were continued for two years and then tapered off.

Discussion:

Neurocysticercosis is a common parasitic infection of the brain. It can occur intra-parenchymal, intraventricular, within the subarachnoid cistern or spine. CT/MRI is the standard modality to detect NCC. On imaging, five stages are recognized: non-cystic, vesicular, colloidal-vesicular, granular-nodular, and calcified-nodular stage (Lucato et al. 2007; Zhao et al. 2015). The colloidal vesicular stage mimics pyogenic brain abscess. On MRI, the signal in the cystic lesion is hyperintense on T2 in comparison to CSF and with peripheral enhancement suggestive of fibrosis. The colloidal vesicular stage signifies loss of immune tolerance to the larva (Kimura-Hayama et al. 2010).

Reports of cysticercal abscess are rare in literature. Kapu et al reported a single case of spinal cysticercal abscess. MRI showed T1W isointense lesion with central hypointensity and enhancement at the periphery. The abscess was excised, and the yellowish fluid was sent for culture. Histopathology of the wall showed fibrocollagenous tissue with foamy histiocytes mixed with lymphocytes. The characteristics of the parasitic wall with tegumentary layer, subtegumentary cytons, and inner loose reticular layer were suggestive of cysticercal cyst, although no scolex was identified. The yellowish fluid was sterile on culture (Kapu et al. 2012). Abraham et al. recorded bacterial superinfection of solitary cysticercal granuloma confirmed by culture. The source of the infection was presumed to be hematogenous,

although the blood culture was sterile. The biopsy revealed a cysticercal granuloma within the luminal aspect and an adjoining palisade of histiocytes, infiltrated by lymphocytes, plasma cells, and neutrophils like our cases. Hence, abscess formation can be either primary or secondary from other sources like blood (Abraham et al. 2020). The chances of superinfection are greater in the presence of associated immunocompromised conditions like diabetes, malignancy, or retro positive patients. In our series, the first patient (case 1) had uncontrolled diabetes. The previously reported case of spinal cysticercal abscess was without a preexisting immunocompromised condition (Kapu et al. 2012).

The cysticercal brain abscess closely mimics a chronic abscess. Clinically both present with signs of inflammation and neurological deficits due to mass effect. Radiologically, both are identical with peripheral ring enhancement and non-enhancing center. If the cysticercal cyst wall is not found on histology, the differential diagnosis includes pyogenic abscess, tubercular abscess, and fungal abscess (Table 1). The histological clues to the diagnosis include a serpiginous or stellate outline of the cavity walled by several multinucleated giant cells, and an infiltrate rich in eosinophils. Calcospherules are rare. These features should prompt the pathologist to assiduously search for remnants of the parasite. Viable cysts do not produce an inflammatory response. A degenerating cyst elicits an inflammatory response with surrounding fibrosis. As the cysticercus dies, its structure becomes less distinct and usually starts to calcify, producing small calcospherules that aid in diagnosis even in the absence of a parasite wall. The eosinophil rich infiltrate and granulomatous response with foreign body giant cells mimics fungal granulomas or tuberculoma. Stains for fungal hyphae such as Periodic acid Schiff (PAS) and Gomori Methenamine Silver (GMS) help in ruling out fungal etiology. Culture remains the gold standard to prove the sterile nature of cysticercal abscess, and to exclude pyogenic and tubercular etiologies.

Table 1
Histological differential diagnosis of cysticercal abscess.

Nature of lesion	Layers
Pyogenic abscess	1. Central necrosis 2. Inflammatory granulation tissue composed of neutrophils and lymphomononuclear cells 3. Fibrous capsule 4. Gliosis
Tuberculoma	1. Caseous necrosis with acid-fast bacilli on Ziehl Neelsen stain 2. Epithelioid granulomas with Langhan's giant cells 3. Fibrous capsule 4. Gliosis
Fungal granuloma	1. Central necrosis with fungal hyphae highlighted on PAS and GMS stains 2. Granulomas with foreign body type of giant cells engulfing the fungal hyphae. Eosinophils present 3. Fibrous capsule 4. Gliosis
Cysticercal abscess	1. Central necrosis with serpiginous borders lined by palisade of epithelioid cells. Degenerating cysticercal cyst 2. Inflammatory granulation tissue rich in eosinophils along with foreign body type of giant cells 3. Fibrous capsule 4. Gliosis

In the lesions described here, histologically, the lesion revealed layers of an abscess with central necrosis, the wall of granulation tissue, and varying degrees of fibrosis. However, in distinction from a pyogenic abscess, these lesions displayed the presence of four-layered organization with central necrosis having characteristic serpiginous borders, surrounded by mixed inflammatory granulation tissue with palisading epithelioid cells along with multinucleated giant cells and several eosinophils, which was in turn surrounded by fibrous granulation tissue and dense gliosis. These provided a clue that the lesion was not a pyogenic abscess. Detection of parasitic remnants aided the diagnosis. The detection of cysticercal antibodies in serum can provide supportive evidence. Pathogenetic events that lead to the formation of abscess are unclear and maybe governed by factors such as the host immunity and the number of antigens exposed following degeneration of the cysticercal cyst. The possibility of bacterial superinfection has been proposed based on the culture of the abscess contents (Abraham et al. 2020).

There is no standard protocol for management as the reported cases are few (Kapu et al. 2012; Britton and Chaseling 2013; Abraham et al. 2020). The reported cases in the literature were treated with surgical excision followed by anti-helminthic drugs albendazole with or without a short course of steroid, similar to our series (Kapu et al. 2012). The patient with a spinal cysticercal cyst was treated with eight weeks of albendazole and steroid. As the culture was sterile, the patient was not treated with antibiotics. Secondary bacterial infection of solitary cysticercal granuloma can be managed with appropriate antibiotics. Abraham et al used linezolid as the microorganism was vancomycin-resistant Enterococcus spp (Abraham et al. 2020). In our series, cases 2 and 3 were treated with two weeks of albendazole with a short course of oral steroids. As bacterial cultures were sterile, no antibiotics were added in the postoperative period.

Conclusion:

Cysticercal abscess, though rare, should be considered as a differential diagnosis of a solitary rim enhancing lesion, especially if the patients belong to endemic zone. Clinically and radiologically, it mimics a pyogenic abscess, and histopathology is critical for diagnosis. The unwary pathologist may miss the diagnosis unless aware of this entity. Surgical excision with or without a short course of antiparasitic agent is the treatment of choice.

Declarations:

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Ethics approval: Retrospective study, not required.

Consent to participate: Received from participate.

Consent for publication: Taken.

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Figures

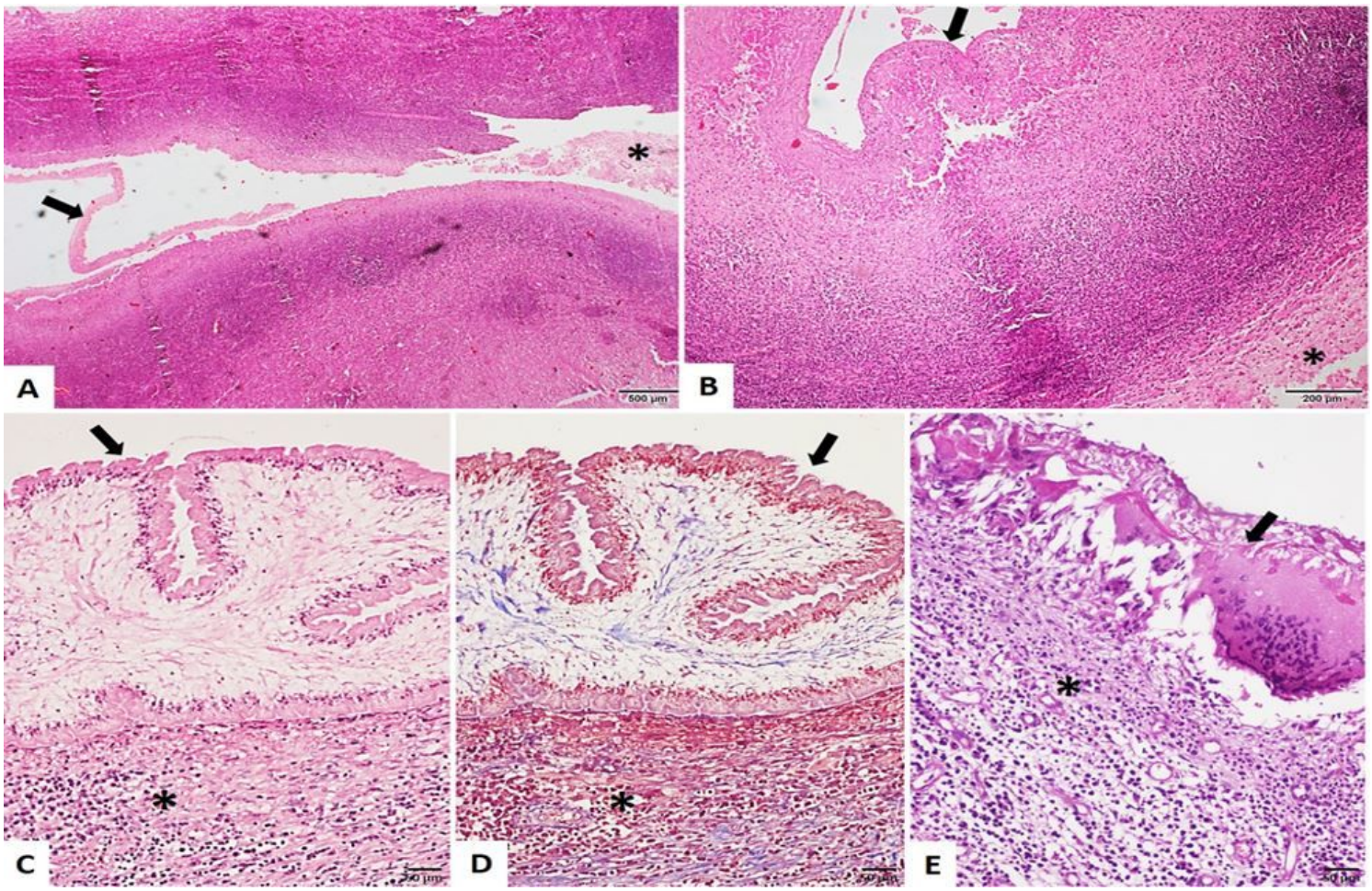

Figure 1

Case 3: Microphotograph of cysticercal abscess showing a serpiginous central cavity with necrosis (asterix, A) and remnants of degenerating cysticercus (arrow). Abscess has a layered organization with the cavity showing serpiginous outlines lined by palisade of histiocytes (arrow, B). This is surrounded by mixed inflammatory and fibrous granulation tissue which is inturn surrounded by dense gliosis (asterix). Case 1: Cysticercal cyst comprising of cuticular, cellular, and reticular layers (arrow) on H & E stain (C) and Masson's trichrome stain (D). Surrounding the cyst is abscess wall (asterix). Case 2: Abscess cavity lined by palisade of histocytes with several multinucleated foreign body giant cells (arrow, E) resting on mixed inflammatory granulation tissue. [A,B,C,E: H&E; D: Masson trichrome. Magnification = scale bar]

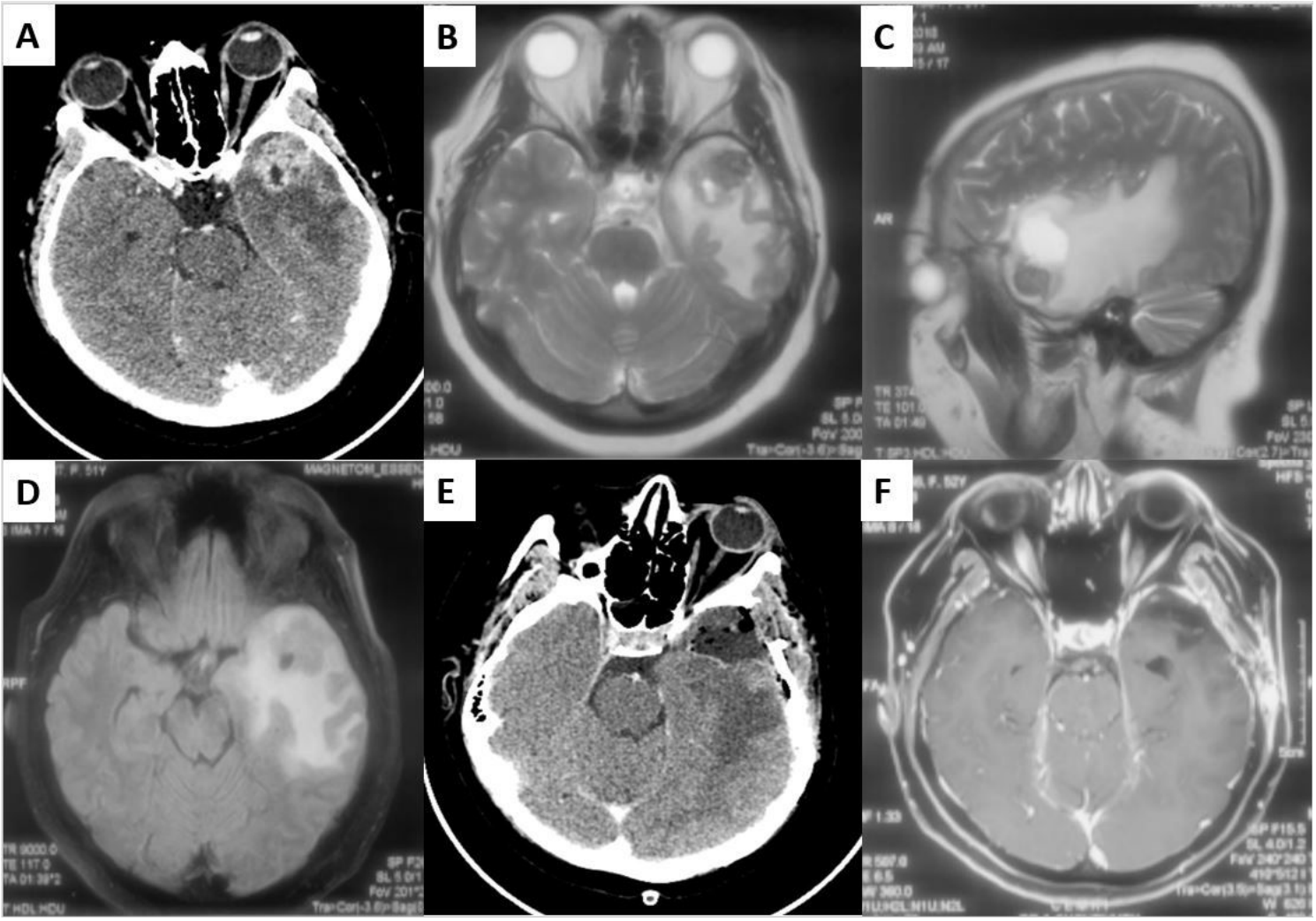

Figure 2

(Case 1): Left temporal cysticercal abscess. A: CT (axial, contrast) shows left temporal solitary lesion with enhancement, B: MRI (T2W, axial) shows left temporal solitary lesion with lateral part hypointense and medial part hyperintense, C: MRI (T2W, Sagittal) shows superior part was hyperintense and inferior part was hypointense, C: MRI (FLAIR, axial) shows perilesional edema, E: CT (axial, contrast) shows complete excision of the lesion. F: Follow up (6-months) MRI (contrast, axial) shows complete excision of the lesion.

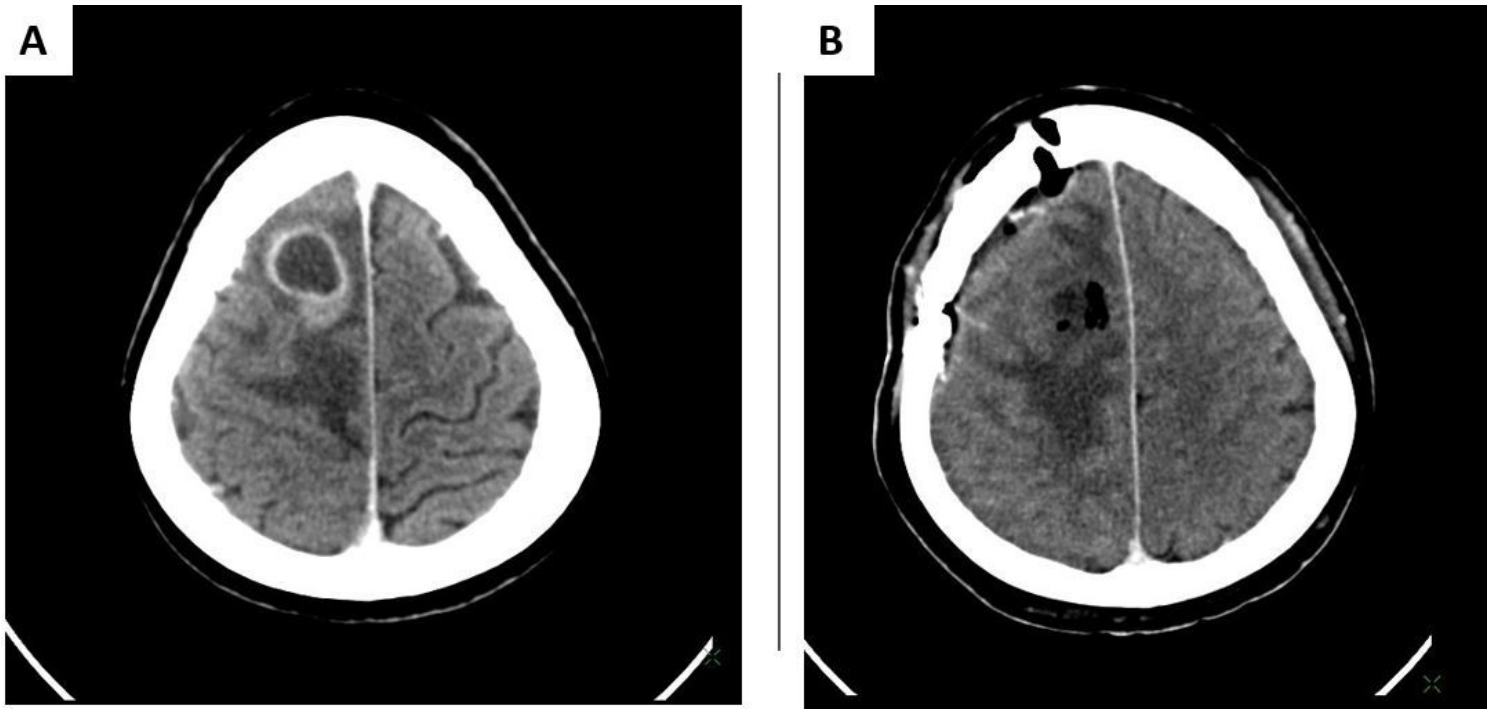

Figure 3

(Case 2): A: CT (axial, contrast) shows right frontal rim enhancing solitary lesion with perilesional edema, B: CT (axial, contrast) shows complete excision of the lesion.

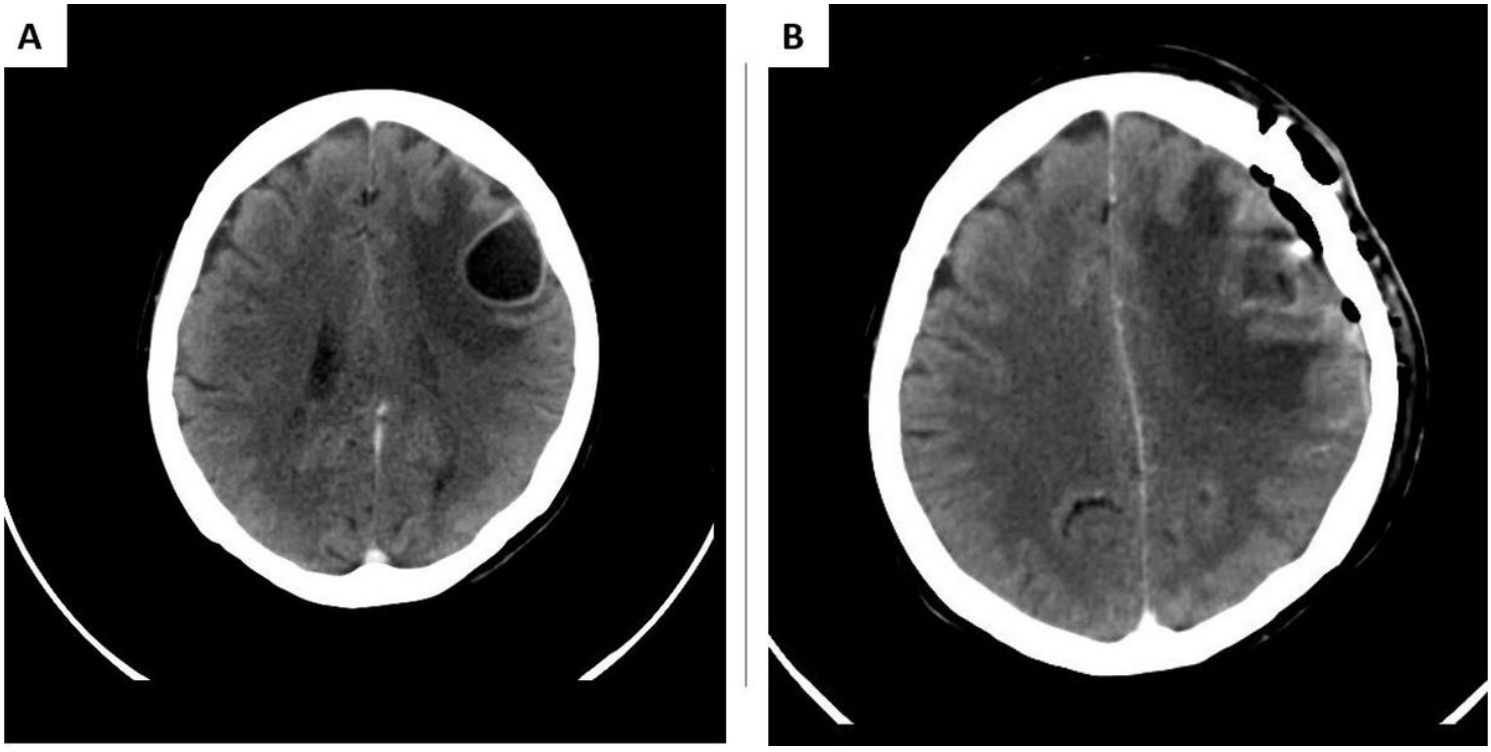

Figure 4

(Case 3): A: CT (axial, contrast) shows left frontal rim enhancing lesion, B: CT (axial, contrast) shows complete excision of the lesion.