Cerebral Amebiasis: An Uncommon Cerebral Abscess

Cerebral amebiasis is a rare form of invasive amebiasis with fatal outcome. Amebiasis is generally a disease of gastrointestinal tract which rarely spreads to other organs, especially brain. Protozoan brain infections are rare and mortality rate is quite high. Most of cerebral protozoan infections are caused by *Entamoeba histolytica* (0.6% to 8.1%).[1] This disease begins with the infection of the colon by *Entamoeba histolytica* trophozoites, which then travel to other tissues and organs through the blood.

Cerebral amebiasis is produced when trophozoites invade the central nervous system. It is rare to see cerebral involvement without any lung or liver infestation.[2] The diagnostic radiology is not specific and diagnosis relies on identification of the amoeba on immunohistopathology. Author reported two cases of cerebral amebiasis without any involvement of lung or liver. Both patients were managed by surgical decompression with amebicidal drugs.

A 47-year-old hypertensive, diabetic male presented with complaints of headache and seizures for last 2 months. The headache was mild to moderate in nature interfering with daily activities. The patient had an episode of seizure which was generalized tonic-clonic in nature and associated with loss of consciousness, followed by a similar episode 5 days later. Patient then started with progressive loss of power in left upper and lower limbs. He was a known case of AIDS stage 1 with CD4 counts of 500/mm^3 of blood. He was on retroviral therapy.

On examination, patient was conscious, cooperative, oriented with Glasgow coma scale (GCS) 15/15. On neurological examination, bilateral cranial nerves were intact and left hemiparesis with power (2/5). On evaluation with magnetic resonance imaging of brain [Figure 1a], there were multiple ring enhancing lesions with largest one in right posterior frontal region with heterogeneous enhancement and perilesional edema, most likely a mass lesion.

Patient underwent right frontoparietal craniotomy for larger lesion with microscopic excision of mass. Intraoperatively, mass was yellowish, avascular with well-defined cleavage plane. Postoperative scan was suggestive of excision of mass [Figure 1a]. Histopathological examination revealed amebiasis, i.e., mass lesion with periodic acid-Schiff (PAS) positive *Entamoeba histolytica* [Figure 1b]. Patient was then further evaluated with ultrasound abdomen, chest X-ray, stool examination, serology and culture for amebiasis that came out to be negative. Patient was started on amebicidal drugs along with antibiotics. Patient was given metranidazole 750 mg thrice daily for 10 days followed by a luminal agent paromomycin 500 mg thrice daily for 10 days to eliminate any risk of any intestinal colonization. Patient improved gradually and was discharged with power (4/5) left upper and lower limbs. On follow-up at 3 months, other cerebral lesions had regressed and were inactive and patient had regained power (5/5) in both upper and lower limbs with no difficulty in walking.

Another 84-year-old male with known case of hypertension, presented with sudden onset weakness of right handgrip followed by weakness of right upper limb, slurring of speech, deviation of mouth to left side. On neurological examination, patient had GCS-E3V3M5 and right hemiparesis (Power: right upper limb 0/5 and right lower limb 3/5). Computerized tomography findings reveal large area of ill-defined hypodensity with interspersed hyperdensity, involving the left frontoparietal lobe extending into the left temporal lobe with surrounding edema causing effacement of the adjacent cortical sulci and midline shift to the right [Figure 1d].

Patient underwent left decompressive craniectomy with tumor decompression [Figure 1c]. Intraoperatively lesion was partially adherent to dura, moderately vascular, partially suckable, firm, mucoid, with ill-defined plane of cleavage with intralesional hematoma. On histocytopathology, there were areas of necrosis with fibrinous material, neutrophilic infiltrate, areas of hemorrhage, and amoeba trophozoites in focal areas [Figure 1e]. Postoperatively, patient was started on metronidazole 750 mg thrice daily for 10 days followed by paromomycin 500 mg thrice daily for 10 days to eliminate any risk of intestinal colonization. Patient improved neurologically and was discharged in stable condition with GCS 15/15 with gradually improving right hemiparesis with power right upper limb 3/5 and right lower limb 4/5. On follow-up at 3 months, patient had regained full power and was able to walk independently.

Amebiasis is caused by an obligate *Entamoeba histolytica* which replicates in intestine and transmitted by feco-oral

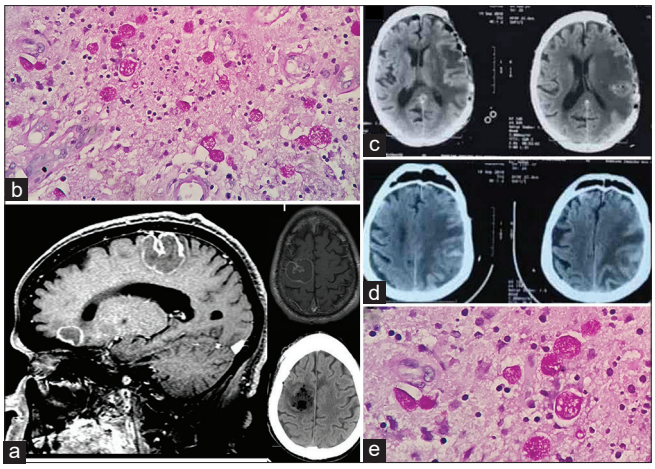

Figure 1: (a) MRI brain suggestive of showing multiple ring enhancement lesions typical for absess/mass, post operative scan suggestive of excision of mass. (b)Histopathology suggestive of trophozoites with PAS-positive, vacuolated cytoplasm. (c)Preoperative CT scan suggestive of left fronto-parietal mass with edema causing midline shift. (d) Post operative scan suggestive of excision of mass with post operative changes. (e) Histopathology suggestive of amoebic trophozoites with eccentrically placed nucleus with ingested erythrocytes within the cytoplasm

route.[3] Intestinal manifestations can lead to cerebral amebiasis in only 50% of cases.[4] In our study, both patients presented without any intestinal manifestations. Patient may present with features of raised intracranial pressure, headache, vomiting, seizure, altered sensorium, meningitis, hemiparesis, and hemiplegia with cranial nerve involvement.[5,6] In our series, one of patients presented with headache, seizure with hemiparesis and other one presented with unconsciousness and hemiparesis. With no intestinal manifestation in our cases, we did not go for gastrointestinal evaluation initially.

Extraabdominal spread to brain is rare and fatal and if not managed can lead to sudden deterioration.[7] Cerebral amebiasis may present with an abscess (single or multiple) or mass of granulation tissue (ameboma). One of our patients had multiple ring enhancing lesions, while other one presented with mass lesion with surrounding edema. Frontal lobe and basal ganglia regions are the most common sites involved with left-sided involvement more common as compared to right.[4] Radiologically, picture of cerebral amebiasis is indistinguishable from other abscesses or masses in brain.[8]

Pathologically, cerebral amebiasis is characterized initially by poorly defined lesions with irregular margins to late ones with well-defined margins and central necrosis.[4] Trophozoites with single rounded nucleus with PAS positive cytoplasm and ingested red blood cells is hallmark of *Entamoeba histolytica*.[9] Our cases were diagnosed on the basis of histopathological basis. Doubtful cases must be followed by immunohistochemistry and immunofluorescence with specific antibodies for *E. histolytica* to distinguish from free living *N. fowleri*.[10]

Both of our patients presented with mass lesions so surgical decompression was done and diagnosis made on histopathology. Patients were evaluated for possible lesions in intestine, liver, and lungs but no focus was found anywhere. Both patients were started on ambecidal drugs and responded to treatment and improved neurologically.

Cerebral amebiasis is invasive amebiasis with fatal outcome if not diagnosed in timely manner. Cerebral amebiasis should always be kept in differential diagnosis of brain abscesses and intraaxial masses. Although radiology is not much helpful in diagnosis, competent pathologist establishes the diagnosis of cerebral amebiasis. It should be suspected in acutely ill immune-compromised patients and in areas of increased prevalence of disease.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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Submitted: 12-Jun-2020 **Revised:** 29-Jun-2020
Accepted: 02-Jul-2020 **Published:** 21-May-2021

DOI: 10.4103/aian.AIAN_583_20