A Neurocysticercosis Infestation Masquerading as Malignant Brain Tumour

Sir,

We report an interesting case of neurocysticercosis causing midline shift and mimicking a neoplastic lesion. This atypical imaging findings of such a common disease is extremely rare, and only few case reports are available in the literature.\[1-3\]

A 50-year-old lady presented to the emergency with one day history of multiple simple partial seizures involving right arm and face without any motor deficit or loss of consciousness. General and neurological examinations were otherwise unremarkable. She had past history of seizures of multiple types for 4 years. An urgent non-contrast CT scan of brain was done which showed a relatively well-defined hyperdense lesion (3.0 × 2.6 × 2.5 cm in size) located in the grey-white matter junction of left posterior frontal region. The lesion had a central focus of dense calcification and extensive disproportionate perilesional edema causing compression of ipsilateral lateral ventricle and midline shift [Figure 1]. Based on these findings, neoplastic etiology was suspected and anti-edema medicines including steroids, anti-epileptic drugs and other supportive measures were started.

After 3 months, contrast-enhanced MRI was done which revealed a well-defined multi-lobulated lesion in subcortical white matter of left posterior frontal region [Figure 2]. The peripheral rim was hyperintense on T1-WI, hypointense
on T2-WI and showed smooth enhancement following gadolinium administration. On the contrary, the core of the lesion was hypointense on T1-WI, hyperintense on T2-weighted and FLAIR images and had no enhancement. Diffusion was restricted only within the core of the lesion. Large single focus of calcifications was present on susceptibility-weighted images. Compared to the previous non-contrast CT scan, the size of the lesion had reduced to 2.4 × 2.2 × 2.1 cm with significant reduction in perilesional edema. The imaging appearance was suggestive of conglomerate peripherally enhancing ring lesions and the possibility of tubercular etiology or neoplastic etiology was debated. However, regression in size of the lesion without any definitive treatment for tuberculosis was against the possibility of tuberculosis. Patient had undergone a CECT 4 years back for her seizure which was reviewed. The previous CT scan showed a single discrete ring enhancing lesion (SDREL) with eccentric hyperdensity s/o solitary neurocysticercosis in the same location [Figure 3a]. In view of previous CT findings of NCC and resolution of edema without antitubercular therapy, the diagnosis of NCC was thought of and it was decided to follow-up this patient clinically and on imaging. The steroids were tapered over next one month. The patient was continued on anti-epileptic therapy and her seizures were controlled. Serial follow-up MRI done after 1 and 3 years [Figure 3b and c] showed progressive decrease in the size of the lesion without any surgery or antitubercular drugs supporting the final diagnosis of NCC, which had in fact, masqueraded as a brain tumour.

The enlarging ‘NCC’ due to deposition of second NCC cyst adjacent to previous cyst has been rarely reported,[1,2] however this is the first reported case in which SDREL of NCC has evolved into multiple conglomerate ring-enhancing lesions of NCC during follow up. This could be explained by deposition of multiple cysticercal larvae in one location and metachronous development of these cysts at different time points. Interestingly, the disproportionately large perilesional edema was causing midline shift, which unusual in NCC.[4] Conglomerate NCC are common presentation of single NCC lesion[5] and these may become more than 2.5 cm in size, mimicking giant NCC lesions, which is a rare form of NCC.[6]

Figure 1: Non-contrast CT scan of brain shows a relatively well-defined hyperdense lesion in left posterior frontal region predominantly at grey white matter junction. The lesion had central focus of dense calcification (b) and had extensive disproportionate perilesional edema causing mass effect (a)

Figure 2: MRI Brain after 3-months shows a well-defined conglomerate ring lesion in left posterior frontal region. The peripheral rim is hyperintense on T1-WI (a), hypointense on T2-WI (b). The core of the lesion is hypointense on T1-WI (a) and hyperintense on T2-WI (b and e). The core of the lesions is bright in diffusion trace image (c) and dark in ADC image (d) suggesting diffusion restriction. Large single focus of calcifications was present on susceptibility weighted image (f). On post contrast images (g and h), peripheral multi-lobulated smooth rim enhancement was noted without any enhancement in core of the lesion
The most common symptoms are dry cough, dyspnea, fever, anosmia, dysgeusia, acute/subacute consciousness impairment, meningoencephalitis, acute hemorrhagic necrotizing encephalopathy, Guillain-Barré and Miller Fisher syndromes, cranial nerve palsy, and ischemic/hemorrhagic neurological complications of COVID-19, which include nonspecific symptoms (headache, dizziness), weakness, and myalgia.

COVID-19 is primarily a disease with infectious and acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Coronavirus disease 19 (COVID-19) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

Most giant NCC lesions are cysts located in cisterns and may be associated with perilesional edema in adjacent brain parenchyma. These cisternal NCC may show peripheral enhancement. No report of giant parenchymal NCC lesion with multiple separte appearance has been described in NCC.

Sometimes it is challenging to differentiate NCC from tubercular or neoplastic lesions on imaging. Parenchymal and subarachnoid neurocysticercosis may be confused with a malignant brain tumour. The cyst may continue to grow and also may be difficult to differentiate from neoplastic lesion. The colloid vesicular and, to a lesser degree, granular nodular stages can show pronounced enhancement around the cyst and significant perilesional edema, making it difficult to diagnose. Lesions in different stages can demonstrate internal restricted diffusion, more often seen with the colloidal and granular intraparenchymal forms. An early and accurate diagnosis may potentially avoid an unnecessary surgical intervention for this otherwise treatable infective lesion with good outcome.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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References
1. Rajeshkhar V, Chandy MJ. Enlarging solitary cysticercus granulomas. J Neurosurg 1994;80:840-3.
2. Singh G, Sobti P, Bhatia RS, Singh D. Enlarging single CT lesions can also spontaneously resolve. Neurol India 2000;48:85-7.
3. Kumar R, Singh DK, Singh N. Racemose neurocysticercosis masquerading high-grade glial neoplasm: A rarest presentation of common disease. Indian J Neurosurg 2016;5:117-9.
4. Rajeshkhar V, Haran RP, Prakash GS, Chandy MJ. Differentiating solitary small cysticercus granulomas and tuberculomas in patients with epilepsy. Clinical and computerized tomographic criteria. J Neurosurg 1993;78:402-7.
5. Garg A, Kaur KP, Devaranjan Sebastian LJ, Gaikwad SB, Bhatia R, Singh MB, et al. Conglomerate ring-enhancing lesions are common in solitary neurocysticercosis and do not always suggest neurotuberculosis. Ann Indian Acad Neurol 2019;22:67-72.
6. Mittal A, Sehgal R, Sureka B, Kumar A, Aggarwal KC. Giant cerebral cysticercosis in an infant confused with a thalamic glioma. J Child Neurol 2014;29:NP154-6.
7. Kumar S, Thakur S, Jhobta A, Sood RG. Giant racemose neurocysticercosis with mass effect: Unusual presentation. Ann Indian Acad Neurol 2013;16:398-9.
8. Sabel M, Neuen-Jacob E, Vogt C, Weber F. Intracerebral neurocysticercosis mimicking glioblastoma multiforme: A rare differential diagnosis in Central Europe. Neuroradiology 2001;43:227-30.

Submitted: 07-Jul-2020 Revised: 16-Jul-2020 Accepted: 23-Jul-2020
Published: 07-Nov-2020

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DOI: 10.4103/aiian.AIAN_725_20