Granulomatous amoebic meningoencephalitis

Sir,

An 18-year-old male presented with progressive left parietal headache evolving into severe holocranial headache associated with vomiting of 27 days duration and gradually progressive right hemiparesis since 5 days. There was no history of fever at the onset of symptoms and he had no immunodeficiency state or on immunosuppression treatment. Neurologic examination revealed altered sensorium, mild right hemiparesis with neck rigidity. Cerebrospinal fluid (CSF) examination revealed glucose of 71 mg/dL, protein of 132 mg/dL, and 20 nucleated cells/dL (all lymphocytes). Results of gram and acid-fast staining were negative. Brain computed tomography (CT) scan revealed an ill-defined hypoattenuating lesion in the left frontoparietal lobe and right superior frontal gyrus with linear overlying gyriform enhancement and moderate perilesional edema [Figure 1]. Brain magnetic resonance imaging (MRI) revealed multifocal involvement of bilateral cerebral hemisphere and left cerebellar hemisphere [Figure 2]. The largest lesion in the left frontoparietal lobe resembled a mass lesion with interspersed hemorrhages and showed linear gyriform pattern of enhancement [Figure 1d]. A diagnosis of infective process of the brain and meninges was considered and he was stated on parenteral vancomycin and ceftrixone along with parenteral steroids. Subsequently, the child developed signs of increased intracranial tension and lapsed into unresponsive state and died on day-10 of admission. A partial autopsy and brain examination revealed granulomatous amoebic meningoencephalitis [Figure 3].

Central nervous system amebiasis is a rare condition that is caused by several free-living amebic organisms and is broadly divided into granulomatous amebic encephalitis (GAE) and primary amebic meningoencephalitis (PAM).[1] GAE is a subacute to chronic infection caused by Acanthamoeba and also *Balamuthia mandrillaris* organisms, unlike primary amoebic encephalitis caused by *Naegleria fowleri* which is acute in onset, rapidly progressive, resulting in fatality within 48–72 h. The incubation period of this disease is not known and is postulated to be more than 10 days.[2] The disease occurs in the 2nd and 4th decades and 10 times more in men. Pathologically, the lesions represent focal areas of cerebritis or microabscesses and leptomeningitis of the overlying cortex. The clinical course for GAE is characterized by a long duration of focal neurologic symptoms. A spectrum of radiological findings in the form of multifocal parenchymal lesions, necrosis,

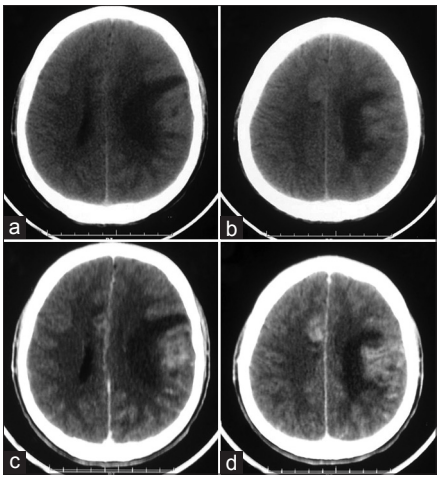

Figure 1: Noncontrast (a and b) and contrast-enhanced (c and d) computed tomography axial images shows an ill-defined hypoattenuating lesion in the left frontoparietal lobe and right superior frontal gyrus with linear overlying gyriform enhancement

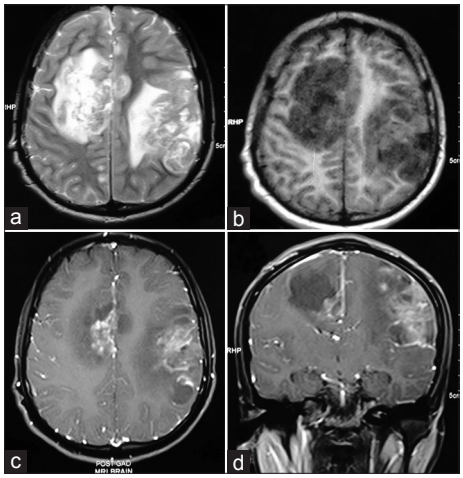

Figure 2: T2 axial (a), T1 axial (b), post contrast T1 axial and coronal (C and D) magnetic resonance images reveal multiple lesions, the largest lesion in the left frontoparietal lobe resembling a mass lesion with interspersed hemorrhages and linear gyriform pattern of enhancement

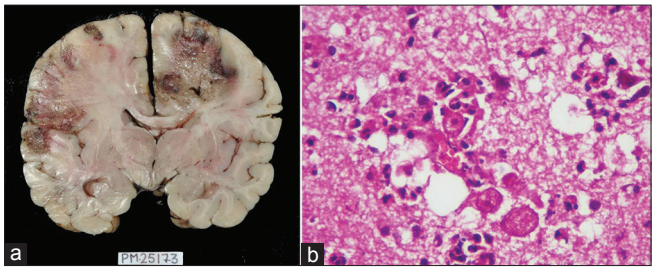

Figure 3: Gross specimen (a) showing bilateral hemorrhagic and necrotic lesions in the frontal lobes. The photomicrograph (b) demonstrating a few trophozoites among neutrophilic infiltrate in the brain parenchyma (hematoxylin and eosin (H and E), original magnification ×40)

intralesional hemorrhage, pseudotumoral lesions, and leptomeningitis has been described. The mass-like lesion (pseudotumoral pattern) often demonstrates a linear and superficial gyriform pattern of enhancement, which is a useful indicator of the diagnosis. It possibly represents a combination of enhancement in the inflamed meninges and the underlying cortex. Associated microabscesses are caused by infiltration of brain substance, by amoebae along the pial vessels. The multifocal pattern shows T2 hyperintensities and ring enhancement, with a predilection for the diencephalon, brainstem, and posterior fossa structures. Early diagnosis and surgical removal of granulomatous mass lesions, combined with chemotherapy, such as amphotericin B or clotrimazole, provides the best chance of survival. However, the prognosis remains dismal.[1-3]

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