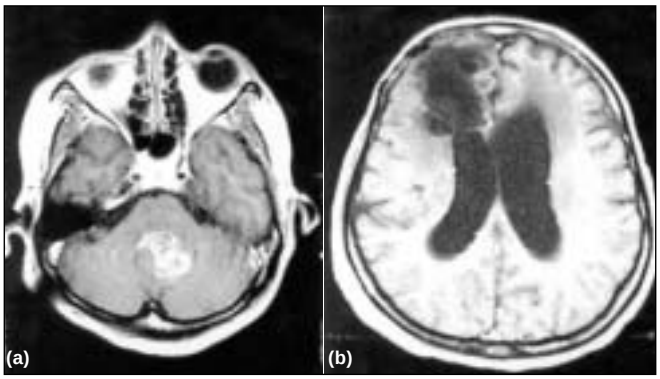

Figure 1: (a) T1W axial post gadolinium MR image showing a 2 cm enhancing mass in the vermis with mass effect on the fourth ventricle. (b) T1W axial post gadolinium MR image showing postoperative changes in the right frontal region. There is no evidence of recurrence. There is hydrocephalus

craniectomy and radical excision of the mass. The biopsy was reported as oligoastrocytoma (WHO Grade 2). She was given 56 Gy of external radiotherapy.

Choucair et al, in a large study, have reported the frequency of CSF metastases at 5-7%.[1] Compared to malignant primary tumors of the central nervous system low-grade gliomas are rarely known to metastasize through the CSF. In 1929, Cairns[2] reported the first case of CSF spread of an oligodendroglioma through the ventricles. In our case, similar histology of the frontal and vermian masses, rarity of primary vermian mixed oligoastrocytoma and the fact that the vermis was not included in the radiation field after the first surgery suggest that the vermian mass is likely to be due to CSF spread. The possible mechanisms of tumor dissemination include exfoliation of cells into the CSF of the ventricles or the subarachnoid spaces. Previous craniotomy procedures, shunt procedures, site of tumor, glioma type and tumor cell differentiation are known to affect CSF spread of gliomas.[3-5] Oligodendrogliomas are more commonly associated with CSF dissemination. Friedberg et al[6] have proposed CSF analysis of specific matrix metalloproteinases profile as a means to detect CSF spread of tumors.

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Fulminant subdural empyema- an unusual complication of pyogenic meningitis

Sir,

An otherwise normal 56-year-old diabetic patient presented with a one-day history of multiple generalized tonic-clonic seizures followed by altered sensorium. There was no history of fever. There was no history of trauma or any focus of infection. There was no focal neurological deficit or signs of meningitis.

Hematological investigation revealed leucocytosis with a total white blood cell count of 16,600/cu.mm and an ESR of 45 mm. Blood sugar was 351 mg%. Computed tomography (CT) scan of the brain revealed no abnormality (Figure 1). The lumbar CSF analysis revealed 1350 cells/mm with 96% neutrophils and 04% lymphocytes. Blood and CSF cultures did not reveal any growth. A diagnosis of pyogenic meningitis with diabetes was considered and the patient was placed on broad-spectrum antibiotics. Two days after admission to the hospital she developed recurrent attacks of seizures, lapsed into altered sensorium and developed a left pupillary dilatation. Repeat CT scan revealed a left fronto-parietal hypodense, extracerebral fluid collection with severe brain edema causing midline shift and obliteration of the basal cistern (Figure 2). An emergency left frontal burr hole was done and thick pus was evacuated. Gram's stain revealed pus cells and gram-nega-

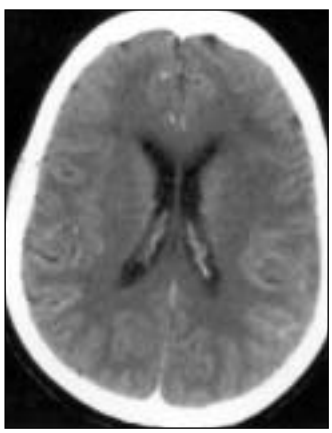

Figure 1: Plain and contrast CT scan of the brain done on 07/11/2003 shows no significant abnormality

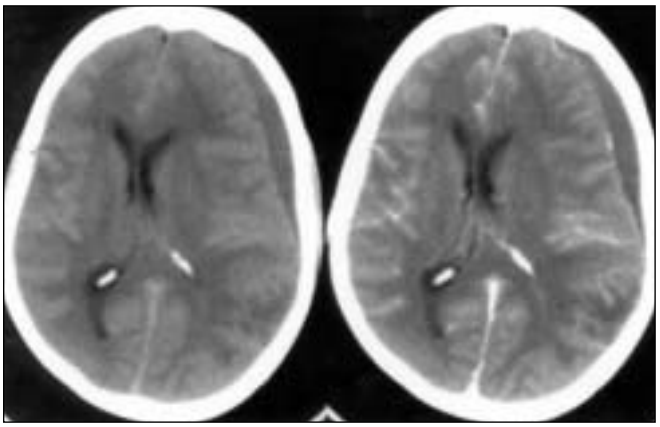

Figure 2: Plain and contrast CT scan of the brain done on 09/11/2003 shows a left fronto-parietal hypodense, extracerebral fluid collection with severe brain edema causing midline shift and obliteration of the basal cistern

tive bacteria and the culture showed growth of Klebsiella species. The patient deteriorated rapidly and died.

Subdural empyema complicating meningitis is relatively common in infants, but is rare in adults.[1,3] Our experience in the present case suggests that subdural empyema should be suspected in patients with pyogenic meningitis who develop recurrent seizures, focal neurological deficit or deteriorate neurologically.[3-5]

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Periodic lateralized epileptiform discharges in a child with solitary cysticercus granuloma

Sir,

Periodic lateralized epileptiform discharges (PLEDs) are a well-defined electroencephalographic entity known for the past four decades. PLEDs have been associated with both partial and generalized seizures, and typically with status epilepticus (SE).[1] PLEDs were found in only 0.5% of 8002 pediatric electroencephalographic (EEG) studies.[2] PLEDs have been described in association with a variety of conditions including cerebrovascular accidents, viral encephalitis, subdural hematoma, metabolic abnormalities, mitochondrial encephalomyopathy, diffuse neurocysticercosis,[3] neurosyphilis and acquired immunodeficiency syndrome. Approximately two-thirds of pediatric cases are related to central nervous system infections.[2]

A ten-year-old child, the youngest of nine siblings, was brought with a five-hour history of repeated right focal motor seizures. He had twitching of tongue and right hand associated with tonic deviation of head to the right side. Initial episodes were secondarily generalized, but after admission, he continued to have right focal motor seizures without loss of consciousness. A clinical diagnosis of status epilepticus was made. He was treated with intravenous lorazepam and phenytoin loading dose with which his seizures were controlled.

Contrast brain CT scan (Figure 1) showed features suggestive of a solitary cysticercus granuloma (SCG) in the left medial frontal lobe. Electroencephalogram (EEG) done the following day (Figure 2) showed persistent periodic bursts of biphasic sharp-wave discharges up to 500 mv in amplitude, periodic lateralized epileptiform discharges (PLEDs) over the left hemisphere with predominance to the central and parietal regions, occurring at a frequency of one per second. Serum anticysticercal antibody was positive.

EEG repeated after ten days had normalized. The child has been seizure-free during 15 months of follow-up and a repeat CT scan seven months after the initial presentation showed

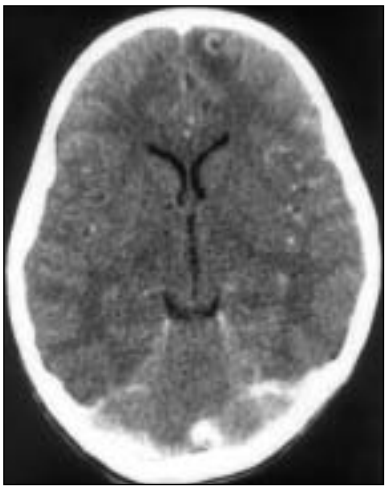

Figure 1: Post-contrast axial CT scan of the brain showing a ring-enhancing lesion in the left medial frontal lobe suggestive of solitary cysticercus granuloma