

Ischemic Cerebral infarction in a young man by disseminated Neurocysticercosis

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ABSTRACT

Background: Disseminated variety with cerebral infarction is a rare manifestation of Neurocysticercosis.

Aims and Objectives: To study a case of disseminated Neurocysticercosis with left hemiparesis

Methods: A 26 year old apparently healthy man presented with recurrent generalized tonic clonic seizure and severe headache. His clinical examinations revealed left hemiparesis and brisk reflexes. His imaging studies revealed multiple ring enhancing lesions all over the brain parenchyma with a 'Starry Sky' appearance and right parieto-occipital lobe infarct. He was treated with anticonvulsants and steroids. In the 6th week of follow up his seizure is controlled and headache is relieved considerably but hemiparesis persists.

Results: He is diagnosed as a case of multiple Neurocysticercosis with right Temporo-parieto-occipital infarct.

Study Limitations: Very few cases of disseminated Neurocysticercosis with hemiparesis are reported in recent times. Further studies are necessary to assess the correlation between disseminated Neurocysticercosis and infarction.

Conclusion: Neurocysticercosis should be kept in mind as a differential diagnosis of stroke in young.

Keywords: seizure, headache, Neurocysticercosis, infarct

INTRODUCTION

Human cysticercosis is infection by the larval (cysticercus) stage of the pork tapeworm *Taenia solium*. The infection is one of the most important causes of seizure in the developing world¹. Disseminated cysticercosis with cerebral infarction is an uncommon manifestation Neurocysticercosis. More than 50 cases of disseminated neurocysticercosis were reported worldwide², the majority being from India. We report a case of disseminated Neurocysticercosis with ischemic cerebral infarct.

CASE REPORT

A 26 year old male presented with recurrent attacks of generalized tonic clonic seizure since the morning of the day of admission and headache for the last 6 months with intensification in last 2 days.

The seizure had no particular precipitating factor like sleep deprivation, head trauma or systemic illness. It was not preceded by any unusual sensory or motor sensations inside the body. The longest seizure episode lasted for approximately 1 minute with post-ictal confusion. He was soon rushed to the hospital, treated there with I.V. Lorazepam and loaded with i.v. Phenytoin @20mg/kg body wt. There was associated history of recurrent nausea, vomiting for the last 2 days, blurring of vision in all directions for the last 15 days.

He had also a history of chronic non specific headache for the last 6 months. The headache was initially confined to the left temporal region with no specific aggravating or relieving factors like exposure to sun, TV watching, change of posture, excitement, any particular food or beverages. Over the last 2 months the headache became generalized, persistent and mildly progressive. In last 2 days the headache has become too severe to be controlled by analgesics. He had no past history of similar episodes nor he had any major illness or hospitalization.

On examination his vitals were stable. There were few non tender pea-size subcutaneous nodules distributed over forehead, nape of the neck and the flexor aspect of the elbows. His gait was ataxic and power of the left upper limb was 3/5 and lower limb was 2/5 with exaggerated deep tendon jerks and extensor plantar response in the left side.

Routine investigations revealed normal Total count, differential count (except ESR of 50mm/hr) glucose, renal and liver function tests. His bleeding, clotting parameters including procoagulant screening revealed no abnormality. Tests for HIV 1 & 2 antibodies (ELISA) were negative. An echocardiogram failed to show cysticerci in the heart. X-rays of the skull and extremities, USG whole abdomen were normal. Fundus examination revealed mild bilateral papilledema. CSF study was not done in the face of raised intracranial tension.

Magnetic resonance imaging (MRI) scan showed multiple cysts in the various stages of developments (many showing eccentric dot like scolices) in frontal, temporal, parietal, occipital cortex, thalamic region, tongue, and soft tissues of the posterior neck displaying a so called 'Starry Sky' appearance(Figure 1, Figure 2, Figure 3). CT brain(plain) showed parieto-occipital region infarction(Figure 4a). There DW MRI showed infarction in the right parieto-occipital region(Figure 4b) . The lateral ventricles were compressed.

The patient was treated with mannitol, i.v. dexamethasone beside Phenytoin. Unfortunately his convulsion was not controlled with accepted highest dose Phenytoin alone so later oral sodium valproate and clobazam were added. He was also prescribed oral Prednisolone @1mg/kg body wt for 4 weeks in a tapering fashion.

On the 6th week of follow up his seizures were well controlled with those three anticonvulsants, headache significantly relieved but left sided hemiparesis persisted. His fundoscopy revealed no papilledema.

DISCUSSION

Neurocysticercosis is diagnosed with Del Brutto et al criteria. Either 1 absolute criterion or a combination of 2 major, 1 minor and 1 epidemiologic criterion are necessary for the diagnosis³. In our case 1 absolute criterion was fulfilled (demonstration of cystic lesion containing a scolex).

Active lesions (vesicular cyst) appear on MRI as hypodense cyst without enhancement, transitional lesions (colloidal stage) appear as a ring or nodular shadow with contrast enhancement and inactive lesions (dead cysts or granuloma) appear as calcified dots⁴. Actually vesicular cysts are minimally antigenic triggering little or no perilesional inflammation so does not enhance with contrast media. Colloidal cysts are dead or dying stages with host immune response triggering inflammation and marked perilesional edema so enhances with contrast whereas granuloma are dead cysts¹.

The main features of disseminated cysticercosis include intractable epilepsy, dementia, enlargement of muscles, subcutaneous and lingual nodules and a relative absence of focal neurological signs or obviously raised intracranial pressure, at least until late in the disease^{2,5}.

Disseminated Neurocysticercosis with starry sky neuro-radiologic appearance is uncommon. PubMed search revealed 22 cases reported by Wadia et al and an additional 16 cases reported². Basu et al described only 2 starry sky appearances in a study of 124 pediatric Neurocysticercosis cases⁶. Th Shanti Devi et al described a case of disseminated Neurocysticercosis with cyst in subretinal space and in right lateral rectus muscle⁷. Bhalla et al reported an immunocompetent patient with disseminated cysticercosis involving the brain, subcutaneous tissues, skeletal muscles, right orbit and thyroid gland².

Ischemic cerebrovascular disease is a relatively under-recognized complication of Neurocysticercosis usually caused by inflammatory occlusion of the arteries at the base of the brain secondary to cysticercotic arachnoiditis⁸. In most cases small vessels are involved leading to lacunar infarction⁹. However, large infarcts related to the occlusion of the middle cerebral or internal carotid artery can also happen. In one study besides occlusive endarteritis, some blood vessels of large diameter showed occlusion of their lumens by atheroma-like deposits that resulted from disruption of the endothelium secondary to the invasion of the vessel wall by a severe inflammatory reaction induced by cysticerci⁸. In our case the cause of infarction is the extensive brain parenchymal involvement extending to the meningeal and subarachnoid space of the brain leading to arachnoiditis or large vessel involvement by the above mentioned mechanisms.

In one study of 28 patients of subarachnoid cysticercosis, 15 patients had angiographic evidence of cerebral arteritis (53%); 12 of the 15 had a stroke syndrome, Eight of the 15 patients (53%) with cerebral arteritis had evidence of cerebral infarction on MRI¹⁰.

In both cysticercus meningitis and tubercular meningitis, the arteries at the base of the brain are surrounded by a dense exudate causing inflammatory changes in the entire wall of perforating blood vessels¹⁰. Neither CT nor cerebral angiography permit the differentiation between tuberculous and cysticercotic meningitis as they show similar changes in both conditions. So Del Brutto suggests that the diagnosis of cysticercosis-induced cerebral infarction should be established only in patients who have no other risk factors for stroke and who show CT evidence of a meningeal cyst adjacent to the infarction or CSF findings compatible with active arachnoiditis⁸.

Those patients with cysticercus angitis should be started on steroid therapy as soon as the diagnosis is established to ameliorate the subarachnoid inflammatory reaction which may cause recurrent cerebral infarcts unlike in patients with atherosclerotic ischaemic stroke, where steroids have proved to be harmful. The diagnosis of NCC should be included in the list of causes of stroke in the young, particularly in endemic areas.

Interestingly highly active brain parenchymal cysticidal drugs like Praziquantel and albendazole can trigger intense host inflammatory reaction in response to the acute destruction of the parasites within the subarachnoid space, may enhance the process of angiitis in the neighbouring blood vessels favouring their occlusion and subsequent cerebral infarction. One patient with a suprasellar cysticercus reportedly developed a cerebral infarct during a course of praziquantel¹¹, There is another report of cerebral infarction in a Korean Neurocysticercosis patient receiving Praziquantel¹².

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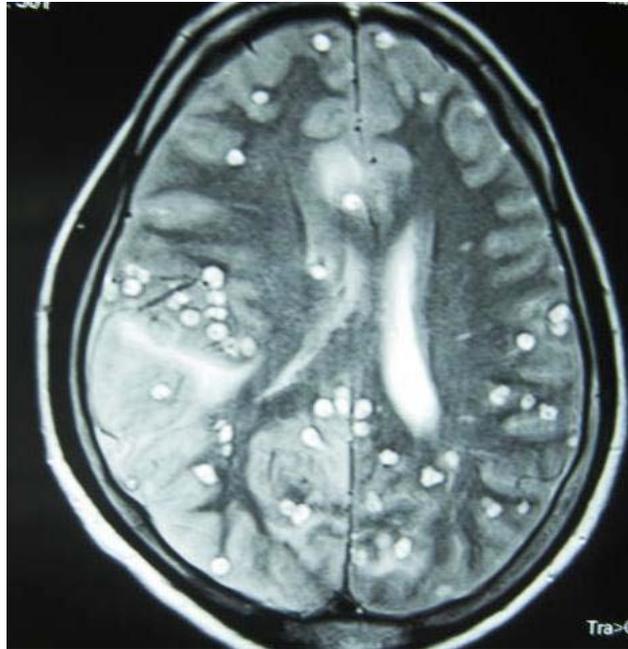


Figure 1: MRI brain (axial view) showing multiple Neurocysticercosis lesions(mainly transitional stage cysts) all over the cerebral hemisphere, lateral ventricle compression, Right parietal lobe edema



Figure 2: T2W MRI brain (Sagittal view) showing starry sky appearance, anterior+ posterior soft tissue neck involvement

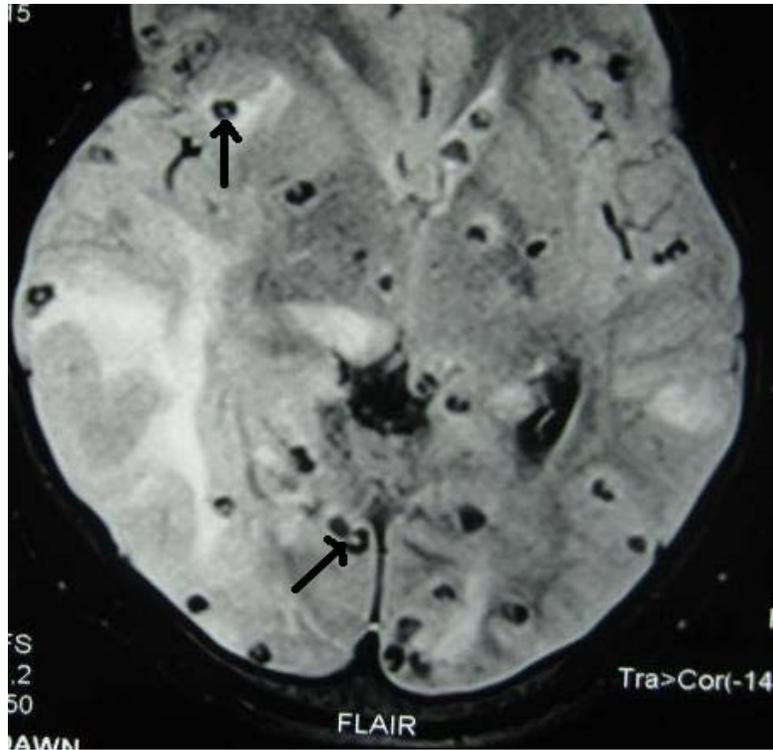


Figure 3: MRI brain (FLAIR) showing numerous lesions with scolex inside in the form of eccentric dots (black arrow)

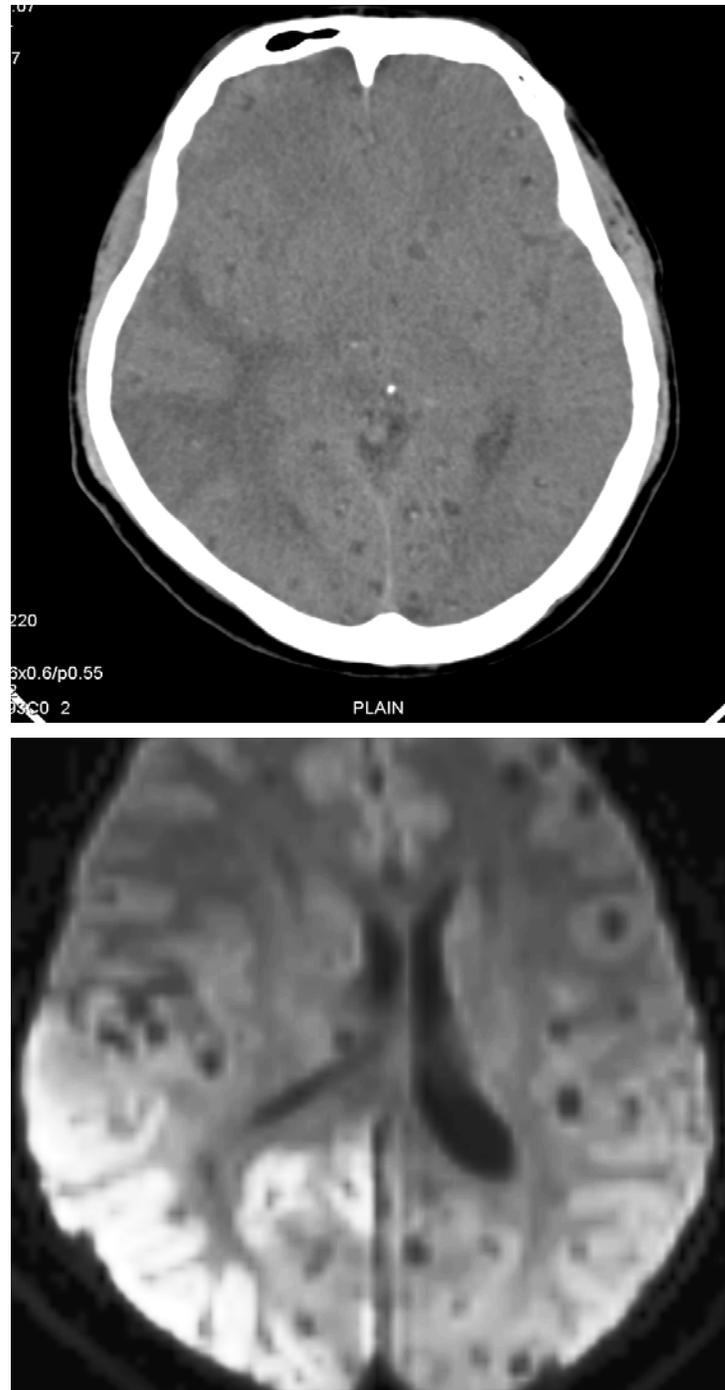


Figure 4a(upper image): CT brain plain shows right parieto-temporal infarct

Figure 4b(Lower Image) :DW MRI showing Right parieto- temporo- occipital hyperintensity suggestive of infarct.