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A CASE REPORT OF MIDDLE CEREBRAL ARTERY INFARCT AND MULTIFATORIAL ETIOLOGIES ASSOCIATED WITH IT.

Paediatrics

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ABSTRACT

Pediatric stroke is rare and devastating condition. Any patient presenting with acute neurological deficit should raise a suspicion of stroke. This is a case of 16 year old female with acute onset of left sided hemiparesis , left upper motor neuron facial palsy and speech difficulty since 2 days. The patient also had a history of weight loss and cervical lymphadenopathy, Neuroimaging confirmed an acute ischemic infarct in right middle cerebral artery. Fine needle aspiration cytology from cervical lymph nodes revealed granulomatous lymphadenitis suggestive of tuberculosis. This case highlights that there is incease in probability of stroke in tuberculosis and Sickle cell disease, hence emphasizing the need for early recognition and intervention.

KEYWORDS

Stroke, tuberculosis, sickle cell disease, ischemic, hemiplegia

INTRODUCTION:

Paediatric stroke is a clinical syndrome consisting of rapidly developing clinical signs of focal disturbance of cerebral function lasting more than 24 hours or leading to death with no apparent cause except vascular origin. [1] Majority of the cases presents with Clinical features like focal neurological deficits like hemiparesis, dysarthria, aphasia, gaze palsies, facial nerve palsy etc.

The prevalence is estimated to be 1-2 in 100,000 children in western countries.[1] Among different modalities MRI Brain with MR Angiography is investigation of choice. Management includes supportive care along with anticoagulant therapy like heparin, low molecular weight heparin, anti-platelet therapy like aspirin.[2]

Out of many etiologies of stroke , most common cause includes Sickle Cell Disease and Tuberculosis. In India Sickle Cell Disease is estimated to be 89.6 per one lakh population and Tuberculosis incidence is 211 per one lakh population.[3,4] As per studies about 46 out of 120 tuberculosis patients suffered from stroke, out of 15.8% patients died having tuberculosis , 10% were suffering from stroke. [5]

Case Report:

A 16 year-old female was admitted to the hospital with complaints of weakness of left upper and left lower limb, speech difficulty and fever for 2 days. At presentation Heart rate, Respiratory rate, Blood Pressure were normal with SpO2 of 97% on room air, Glasgow Coma Scale was normal, Pupils bilateral reactive to light. On central nervous system examination, the patient's memory was intact but her speech was slurred, sensory functions were preserved. Motor system examination of right upper and lower limb was normal with only flickering movement, muted deep tendon reflexes and absent planter reflex of left upper and lower limb.

On cranial nerve examination, the patient had features of 7th cranial nerve upper motor neuron palsy, evidenced by left sided facial weakness with sparing of forehead muscles, loss of nasolabial fold, deviation of mouth to right side.

All other cranial nerves were intact. There were no signs of meningeal involvement. Fundus examination results were unremarkable. On general examination, the patient was lean, pallor was present, multiple lymph nodes were palpable in the cervical region. No lymph nodes were palpable in the axillary or inguinal region.

Baseline investigations revealed moderate anaemia with thrombocytosis, erythrocyte sedimentation rate was elevated, sickling test was positive, hence high performance liquid chromatography was advised suggestive of sickle cell disease, other baseline investigations like white blood cell count, prothrombin time and activated partial thromboplastin time, renal function, liver function tests, septic screen were within normal limits, To rule out etiologies of stroke some investigations were done which revaled homocysteine level was mildly elevated, serum ferritin and lactate dehydrogenase was elevated, while ANA profile was negative, Complement levels, lipid profile, urine protein/ creatinine ratio, cerebrospinal fluid analysis were normal. 2D echocardiography with agitated saline contrast revealed no structural defects or patent foramen ovale.

Considering history of weight loss with cervical lymphadenopathy, tuberculosis workup was advised which revealed sputum Ziehl Neelsen stain was positive, while Mantoux test, gram stain were negative, Chest radiograph was normal.

Ultrasonogrophy of neck revealed conglomerated lymph nodes seen in left submandibular region and multiple sub-centimetric and enlarged lymph nodes in the bilateral cervical region suggestive of cervical lymphadenitis. Fine needle aspiration cytology of right and left cervical lymph nodes revealed well formed Epitheloid Cell Granulomas in the background of abundant necrosis suggestive of Granulomatous lymphadenitis.

CT scan was done revealed patchy hypodense areas involving right corona radiata and right gangliocapsular region suggestive of Acute ischaemic Infarcts, no evidence of Intraparenchymal extra-axial, intra ventricular haemorrhage is noted.

MRI brain & neck with angiography revealed acute and subacute infarct involving right MCA inferior division territory, lenticulostriate territory, thinned out middle cerebral artery and visualized right anterior cerebral artery with paucity of flow related to enhancement, multiple subcentimeter and conglomerated peripherally enhancing lymph nodes in bilateral cervical region.

The patient was managed conservatively with supportive care, aspirin, Injection enoxaparin . anti-tubercular therapy was given to patient. At the end of hospital stay of around one and half months, notable neurological improvement was noted in patient with improvement in tone, power from 1/5 to 4/5 left upper and lower limb ,and deep tendon reflex +2 in left upper and lower limb.

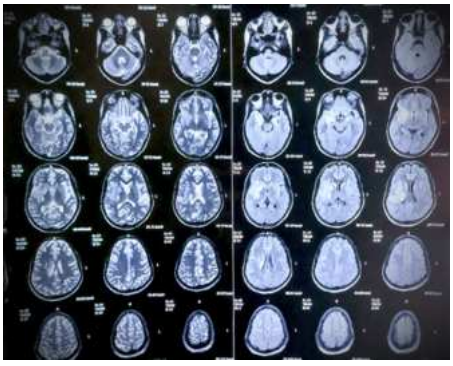

Figure (1): Axial T2 (right) and FLAIR (left) images of the 16 year-old girl showed well defined hyperintensities in right insular cortex, caudate and lentiform nuclei. Additionally sulcal spaces are more prominent than expected for age, suggesting chronic microvascular insults.

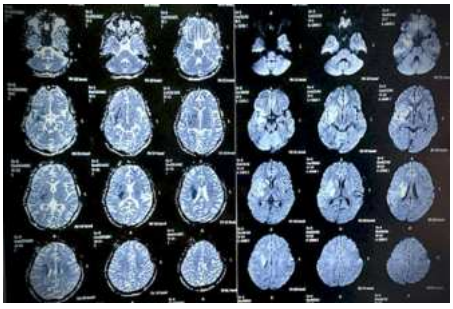

Figure 2: Axial T1 and sagittal weighted images of the 16-year-old girl. The regions corresponding to T2/FLAIR hyperintensities are hypointense on T1 weighted images. Sagittal T2 imaes show concordant findings.

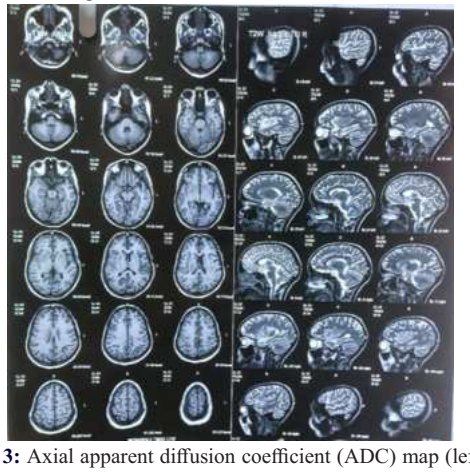

Figure 3: Axial apparent diffusion coefficient (ADC) map (left) and diffusion weight images (DWI) of the 16-year old girl. The regions of T2/FLAIR hyperintensities are bright on DWI and dark on ADC map, suggesting true diffusion restriction, as expected for acute infarct.

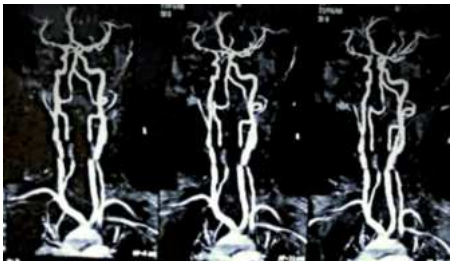

Figure 4: Three dimensional time of flight (3D-TOF) MR angiography shows narrowing of mainly right M1 MCA and also of distal branches, corresponding to the territory of acute infarct.

DISCUSSION :

Stroke is a devastating complication of sickle cell disease, pathogenesis involves chronic hemolysis, endothelial dysfunction, nitric oxide depletion, hypercoagulability and inflammatory activation predispose to cerebrovascular disease [6] Stroke due to sickle cell disease are typically due to large artery vasculopathy affecting intracranial internal carotid arteries and proximal middle cerebral artery and anterior cerebral artery, silent stroke due to sickle cell disease affect territory of penetrating arteries.[7]

Tuberculosis especially extrapulmonary forms causes prolonged immune activation with increased level of pro-inflammatory cytokines such as TNF-alpha, Interleukin-1 and interferon gamma which potential endothelial injury. [8] Systematic tuberculosis also causes reactive thrombocytosis and secondary antiphospholipid antibodies which are recognized risk factor of stroke. [9] Central nervous system tuberculosis commonly lead to meningitis and vasculitis resulting in basal ganglia infracts and middle cerebral artery was the most common involved intracranial blood vessel.

This case illustrates complex interplay between sickle cell disease and tubercular lymphadenitis contributing an acute ischemic stroke in middle cerebral artery. In this case combination of chronic inflammation, anemia induced hypoxia, thrombocytosis predisposed in case sickle cell disease. Management of tuberculosis in sickle cell disease is challenging, isoniazid and rifampicin may alter cytochrome P450 metabolism affecting mechanism of hydroxyurea and anti-epileptic drugs. Additionally, close monitoring is necessary in Sicke cell disease patients, as rifampicin induced hemolysis may exaggerate anemia in these patients. [9]Tuberculosis and stroke individually carry significant morbidity, their co-occurrence needs proactive screening, early neuroimaging and multidisciplinary intervention.

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