

TITLE: EXTENSIVE VASCULITIS IN TUBERCULOUS MENINGITIS PRESENTING WITH STROKE

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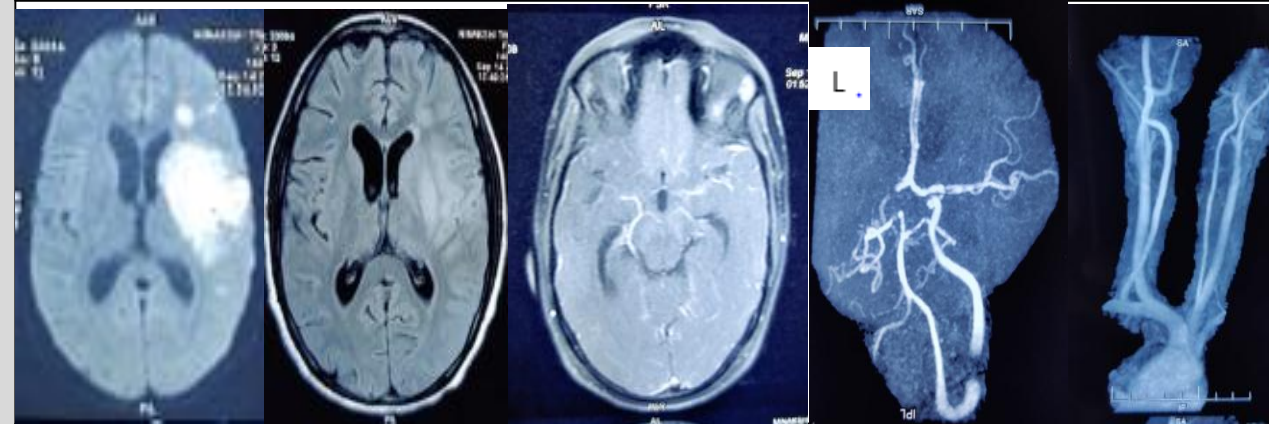
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ABSTRACT: Tuberculous meningitis (TBM) is a common cause of morbidity and mortality in tropical countries. It can complicate with hydrocephalous, vasculitic infarcts, tuberculomas, abscess, optochiasmatic arachnoiditis etc. Vasculitis in TBM is basically at the level of lenticulostriate arteries supplying the basal ganglia and terminal cortical branches.

METHODOLOGY: This 26 year-old post-puerperal day 13 lady presented with fever, persistent headache with intermittent vomiting for 1.5 months, blurring of vision for 1 month and history of altered sensorium for 15 days. There was no history of convulsion, diplopia, dimness of vision, eye pain, weakness, any bowel or bladder symptoms. There is no history of significant weight loss, cough, rash, joint pain, jaundice. There is no history of similar illness in the past, or any history of head injury, high risk behavior, abortion, diabetes, hypertension. Her general examination showed pallor, without icterus, edema, clubbing, lymphadenopathy, rash, joint tenderness, normal thyroid with pulse of 97 beats/min, regular rhythm, normal volume, no delays, all the peripheral pulses were normally palpable, blood pressure 110/70 mm Hg, temperature 100.4 F, respiratory rate 19/min. Nervous system examination revealed initial GCS of E3V4M5 with bilateral grade 2 papilledema with choroid tubercles, normal pupils, normal eye movements, normal power, reflexes with bilateral flexor plantar responses, neck rigidity and Kernig's sign were positive. Her respiratory, cardiovascular and abdominal examination were normal except for palpable uterus in hypogastrium.

Her investigations showed hemoglobin 11.2 g/dL, normal leucocyte and platelet count, differential count N80L15E3M2, normal renal, liver function, blood glucose, electrolytes with negative serology for Hepatitis B, C and HIV. Cerebrospinal fluid examination revealed 180 cells, 10N,90L, protein 1906 mg%, glucose 22 mg% (corresponding blood glucose 154.1 mg%), CBNAAT not detected, Cryptococcal antigen negative, fungal stain negative, malignancy negative. Her MRI brain imaging revealed leptomeningeal enhancement, multiple brain tuberculomas and hydrocephalus and she was treated with anti-tubercular drugs with injectable steroid therapy. Within 5 days, her GCS returned to normalcy, with reduction in signs of meningeal irritation. However on day 6, she developed 4 episodes of generalized tonic-clonic seizure and was treated with levetiracetam injections which eventually controlled her seizures. The next day morning, she noticed sudden onset weakness of right side of body with inability to speak. Her vitals were normal but GCS dropped to E4VAM6 with classical dense hemiplegia of right side with Broca's aphasia. Her CT scan showed acute ischaemic stroke involving left middle cerebral artery (MCA) territory and she was put on antiplatelets. She was further evaluated for evidence of vasculitis, panel was negative. Her MRI brain and neck revealed left MCA territory infarct with reducing hydrocephalous, unchanged leptomeningeal enhancement or new tuberculoma, occlusion of left Internal carotid artery with hypoplastic left vertebral artery. CT angiogram was done to look for evidence of vasculopathy elsewhere in the body which came out to be negative. Her carotid doppler showed the same without thrombus and 2D echocardiography of heart was normal. She was finally diagnosed as extensive vasculopathy in TBM presenting with massive vasculitic infarct. With speech therapy, physiotherapy and medications for TBM & aspirin, she started showing recovery within 4 weeks.

DISCUSSION: The development of infarcts in TBM can lead to a poor outcome. The most common site of involvement is around the basal ganglia via penetrating lenticulostriate arteries and terminal cortical arteries. There has been another concept of classification of infarct by Hsieh et al- TB zone and ischaemic zone. There are multiple mechanisms of such neurological deficits in patients of tuberculous meningitis. First of all, the formation of thick exudates might externally compromise the nearby arterial branches either directly or by reactive vasospasm. Secondly there might be an element of tubercular endarteritis. It is well known that tuberculosis, being a chronic infectious entity can lead to prothrombotic milieu via excessive production of pro-inflammatory cytokines. The element of significantly increased cerebrospinal protein might correlate to the predisposition of extensivity of vasculopathy in TBM.



CONCLUSION: Vasculitic infarcts are an important phenomena in TBM. The formation of exudates in TBM can compress nearby basal arteries directly leading to vascular compromise and infarcts. The involvement of proximal Internal carotid artery in TBM is a rare observation. The exact mechanism of such finding is unknown, however severely elevated protein in cerebrospinal fluid might predispose to such a phenomena in patients of TBM. An extensive study of such association might help in immunopathogenic mechanism of vasculitic infarcts in TBM and role of prophylactic antiplatelets needs to be explored in such patients with severely elevated cerebrospinal fluid protein. The concept of extensive vasculopathy in TBM needs further exploration as the pathological basis of such involvement is not well-known at present. There can be future grading system for predilection of stroke in patients of TBM. In addition, there can be role of prophylactic antiplatelets and cerebral vasodilators in TBM having higher predilection for development of stroke.