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Case Report

A Case Report of Stroke-like Syndrome: Tuberculous Encephalopathy

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Keywords

- Stroke-like syndrome, Tuberculosis, Pericarditis and Encephalopathy

Abstract

Background: Stroke-like syndrome is a stroke mimic, usually common in the young and accurate diagnosis has considerable impact in terms of treatment choices and outcomes.

Objectives: To emphasise the need for high index of suspicion and the need for multidisciplinary approach and accurate diagnosis of stroke-like syndrome.

Case Report: 32 years old woman who presented with recurrent headache of a year duration with right upper limb weakness of 3 months that later involved lower limb 1month and inability to talk 2 days to presentation. Examination revealed a young woman, conscious but aphasic, emaciated, afebrile, anicteric and nil pedal oedema. Neurological findings includes expressive aphasia, right facioparesis and right hemiparesis. Other systemic examinations were essentially normal. A diagnosis of left hemispheric ischaemic stroke was initially entertained. Brain computerized tomography revealed infarct involving the left frontal and temporal lobes but Echocardiography revealed Tuberculous pericarditis, a suspicion of stroke mimic of infective origin was entertained. Diagnosis was confirmed by 'trial of treatment' of anti-tuberculosis, which resulted in dramatic resolution of symptoms within 4 weeks of anti-tuberculosis drugs.

Conclusion: There is need for high index of suspicion, accurate diagnosis and multidisciplinary approach for appropriate treatment choices and outcomes.

INTRODUCTION

A 32year old woman who presented with complaints of recurrent headache of a year duration, 3months history of right upper limb weakness, 1months history of lower limb weakness and inability to talk of 2 days duration. Nil associated history of altered sensorium, vomiting, seizure. No history of palpitations, chest pain, no family history of cardiac disease and stroke. She is not a previously diagnosed hypertensive, diabetic patient.

She does not take illicit drug or use un-prescribed medication, not a known retroviral disease patient. No history of cough or fever but had significant weight loss and she is of low socioeconomic status. Her genotype is AA, not a known peptic ulcers disease, seizure disorder patient. She is para 1+0. Her last confinement was 4 years ago and there is irregular menses and she has never been on any contraception. She doesn't smoke or use tobacco in any form, she does not drink alcohol. Examination revealed a young woman conscious but aphasic, afebrile, anicteric, not pale, acyanosed, but emaciated and there was no pedal oedema. Central nervous system showed right facioparesis and hemiparesis with power of 3/5 in the upper limb and 4/5 in the lower limb. There was no sign of meningeal irritation, pupils were 3mm round, equal and react briskly to both consensual and direct light. Muscle bulk is reduced but tone and reflexes were essentially normal and planter response were flexors. Other systemic examinations were essentially normal. Fasting lipid profile, Fasting blood sugar, Electrolytes, urea and creatinine were essentially normal; 12 leads resting ECG, Chest radiograph, Carotid and veterbral arteries doppler were normal; full blood count showed PCV of 40%, ESR 55mm/hour, WBC 5200/mm^3 with normal differentials; HbsAg, anti-HCV and Retroviral screening were non-reactive. Computed tomography scan done 3 days into admission revealed an area of infarct involving the left frontal and temporal lobe necessitated the initial diagnosis of left hemispheric ischemic cerebrovascular disease (CVD). Serum ANA, serum double stranded DNA antibody, Rheumatoid factor, protein C and S, NOTCH 3 gene assay were normal. Genotype is AA and blood group is A+ve. Echocardiography was done about 18months into this index illness revealed: thickened pericardium with mild pericardial effusion, dilated right atrium and right ventricle, mild tricuspid regurgitation while systolic and diastolic functions were normal.

Patient was on admission for 11months with no improvement in her clinical condition despite optimal treatment for stroke including adequate physiotherapy and speech therapy. She was discharged home and being seen on outpatient. She presented at 18months of treatment for echocardiography which revealed thickened pericarditis, mild pericardial effusion, dilated right atrium and ventricle with normal systolic and diastolic functions. 'Trial of treatment of anti- tuberculosis was started on the

suspicion of a stroke-like syndrome of infective origin possibly disseminated tuberculosis.

We were impressed by the dramatic improvement of total resolution of hemiparesis and facioparesis and patient started speaking within 4weeks of anti-tuberculosis.

There has been steady weight gain and restoration of her normal regular menstrual cycle. Weight on commencement of anti-tuberculosis was 50kg and she was last seen in November 2022 with weight of 59kg. Patient has completed regular anti-tuberculosis drugs for 12 months with progressive and sustained clinical improvement and has been advised to complete 12 months (Figures 1,2).

She repeated ESR and Brain computerized tomography in February 2023. ESR was 08mm/hour and Brain CT (contrast enhanced) revealed Scanogram shows reduction in the areas of infarcts. Serial pre and post contrast axial and reformatted images revealed scattered non enhancing hypodensities of different sizes and shapes in the parietal lobe of the right cerebral cortex. Some of the vessels in the area of hypodensities are prominent in pre-contrast images and shows significant enhancement in the contrast study giving middle cerebral artery sign. The right cerebral cortex and ventricle, cerebellum, pons, medulla and brain stem are normal. The paranasal sinuses are normal. Bone window reveals no bony abnormalities. Conclusion: Right cerebral infarction most likely infective process.

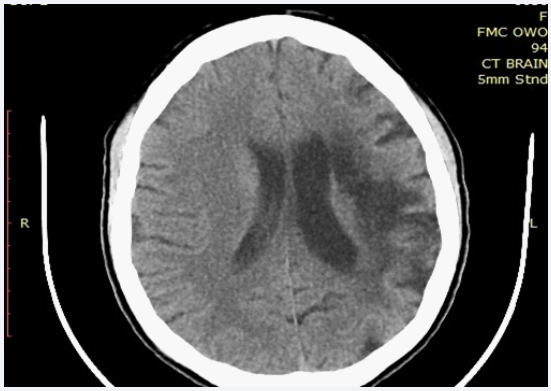

Figure 1 Brain CT before trial of treatment of Anti tuberculosis.

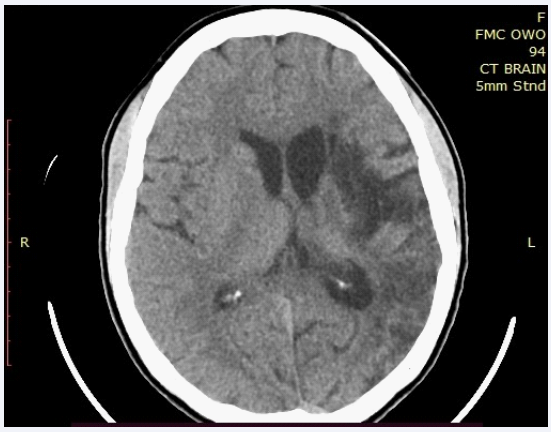

Figure 2 Brain CT after 12 months Treatment of Anti tuberculosis.

DISCUSSION

Tuberculosis is a chronic granulomatous disease caused by *Mycobacterium tuberculosis.* It is a multisystemic disease[1] with numerous presentation and manifestation. Classical symptoms associated with pulmonary tuberculosis are fever, cough, chest pain, hemoptysis, night sweat, weight loss and anorexia. Our patient is a case of extrapulmonary Tuberculousis with symptoms of headache and weight loss. Extrapulmonary tuberculosis [1,2] is common in immunodeficient case of HIV/AIDs [3], Diabetes, Cancer or patient on steroid and Chemotherapy. Our patient is immunocompetent with normal serology for HIV and Hepatitis. Full blood count and blood sugar were normal. Diagnosis is mostly by sputum smear and culture of Acid fast bacilli with Ziehl Neelsen stain and increasingly by rapid molecular based diagnostic test, particularly the GeneXpert PCR-based molecular testing launched in 2004 [4].

Chest radiograph and diagnostic imaging are important supportive diagnostic tools. It is interesting that the diagnosis of TB was not initially considered. Patient has no cough or sputum and facility for GeneXpert is not consistently available in our centre. Chest radiograph was entirely normal. Patient did computerized tomography of the brain, which was wrongly interpreted, in line with initial diagnosis of Ischaemic CVD and subsequently could not immediately do other investigations due to financial difficulty.

Echocardiography done 18months in the treatment revealed Thickened pericardium, mild pericardial effusion, dilated atrium and ventricle with Normal systolic and Diastolic functions. This confirmed the suspicion of TB pericarditis [2]. Although pericardial fluid smear or pericardial biopsy for Acid-fast bacilli detection were not done for logistic reasons. Facility for Adenosine diaminase assay was not available. We were left with the option of 'trial of treatment' of anti-tuberculosis, which is both diagnostic and therapeutic. Other investigations done by the patient were essentially normal with exception of Erythrocyte Sedimentation Rate (ESR) which was 55mm/hour. There is evidence that where diagnostic facilities are limited, a trial of anti-tuberculosis treatment [5] with drugs specific for Mycobacterium tuberculosis may be a useful way of diagnosing disseminated TB. We are conversant with the case for and against 'a trial of treatment' however there is still more work to be done whether this can be incorporated into the case finding package of a National TB control Programme. A closer comparison of the first brain CT and repeat Brain CT after 12 months course of anti tuberculous drugs revealed the same findings but a significant reduction in the area of hypodense lesion and first CT shows associated mass effect as evident by compression of different parts of the ipsilateral ventricle. Magnetic resource imaging might have given a better diagnostic report initially but this not

available in our centre. MRI exist in few centres in our country at a prohibitive cost. There is also a reasonable reduction in ESR from 55 to 08mm/hr

CONCLUSION

Tuberculosis is highly endemic in Africa and Nigeria particularly. Extrapulmonary presentation is commonly seen in immunodeficient patient [3]. We reported this rare case of stroke mimic which turn out to be extrapulmonary TB in an immunocompetent patient, although she of low socioeconomic class. There is evidence of pericarditis and encephalopathy without pulmonary or meningeal involvement. In a resource constraint and diagnostic deficient areas like our there is need for high index of suspicion, availability of rapid diagnostic tools and incorporation of 'trial of treatment' of anti-tuberculosis.

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