TUBERCULOSIS OF BRAIN : PRESENTING WITH RECURRENT STROKE AND INTRACRANIAL HAEMORRHAGE

(A Case Report)

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Introduction

Stroke in the young (less than 45 years of age) is more often observed in India and accounts for 15-30% of all strokes as compared to 3% in Western reports [1]. However, transient reversible ischaemic events of abrupt onset or stuttering progression, so typical of cerebrovascular accidents, are not seen in case of stroke-like presentation in CNS tuberculosis [2,3]. Though panarteritic changes and necrotizing lesions have been described earlier, haemorrhagic strokes are considered exceedingly rare [1-4]. We report a young male patient who suffered 2 strokes

during a period of 20 months. The first resulted in right-sided hemiplegia which totally recovered, but the second episode resulted in death. Autopsy showed bilateral intracerebral haemorrhage.

CASE REPORT

BJ, a male aged 30 years, who belonged to Maharashtra developed an episode of tonic-clonic seizure associated with loss of consciousness and bladder incontinence. He was brought to hospital within 2 hours of onset of symptoms and was found to be comatose. Response to pain was elicited from only the left half of the body. His temperature was 102°F (rectal), pulse 114/minute and blood pressure 170/94 mm of Hg. There was external rotation of the right lower limb which displayed brisk deep tendon jerks and extensor plantar response.

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Fig. 1 : Sagittal section of brain showing blood clots in the lateral ventricle.(Lt).

Right sided facial asymmetry was also noted. Pupillary size and reactions were normal. There was no other cranial nerve deficit. Examination of fundus did not show any papilloedema. Examination of the cardiovascular and respiratory systems were non-contributory. Review of his old medical documents showed that he had an earlier episode of right sided hemiplegia with VII nerve palsy 20 months ago. Blood biochemical parameters, electrocardiogram, cerebrospinal fluid picture and radiography of the chest were then normal. He recovered in two weeks. After 6 weeks of convalescence he was reviewed at a neurology centre where clinical evaluation and computerised tomographic (CT) scan of brain were reported to be normal. He was labelled as a case of occlusive stroke involving left middle cerebral artery territory. Subsequently he was reviewed by us twice at 6 monthly intervals and no sequelae of his neurological event could be demonstrated. He did not have any past or family history of diabetes mellitus, hypertension or tuberculosis. The present episode of convulsion and stroke was not preceded by any febrile episode, headache, vomiting or aura. Initial investigation on admission revealed blood ESR 18 mm fall in first hour. Hb was 15.0 g/dL, TLC - 16,400/cumm, DLC - polymorphonuclear leucocytes - 92%, lymphocytes - 6%, monocytes - 1% and eosinophils - 1%. Malarial parasite was not detected in peripheral blood smear. Urine analysis showed a normal profile. Blood urea, random blood sugar and total serum bilirubin were normal. Total serum proteins, albumin, globulin and transaminase levels were also within normal limits. Radiography of the chest was normal. CT scan of brain could not be done as the facilities were not available.

CSF done at admission revealed that pressure was normal. It was slightly turbid with RBC 950/mm³, WBC 20/mm³, predominant cell lymphocyte. Proteins were 90 mg/dL with increased globulins. Sugar was 82 mg/dL. Gram as well as Ziehl-Neelsen stain of CSF did not show any bacteria.

A 12-lead electrocardiogram showed sinus tachycardia. After admission he continued to be febrile. He was presumptively treated with antitubercular drugs,

Fig. 2 : Microscopic section of 'mid brain' showing epitheloid granuloma with giant cells (x 200).

glucocorticoids and intravenous quinine. On the second day of admission, his coma deepened, plantars became extensor bilaterally and fever persisted. On third day of admission, response to painful stimuli was diminished. He died less than 60 hours after onset of symptoms. Repeat CSF study done immediately after death showed CSF to be uniformly mixed with blood.

Post-mortem examination showed all viscera including heart and lungs to be normal. There was no evidence of significant atherosclerosis in the aorta, carotids or other major vessels. The brain and meninges weighed 1400 g. Left cerebral hemisphere in the temporal region showed a bulge with softening. On cut section, both the cerebral hemispheres showed massive intracerebral bleeding with clots being more marked on the left side. Large areas of left temporal region were replaced by blood clots. Blood clots were also present in both lateral ventricles (Fig 1).

Cerebellum also showed areas of haemorrhage with clots. Lymph node and spleen were normal. Adrenals were congested.

Microscopic examination of the brain showed small- and medium-sized vessels, especially leptomeningeal ones, with aneurysmal dilatations, many showing intimal roughening and medial fragmentation. Leptomeninges showed mononuclear infiltration. Section examined from brain stem (pons, midbrain and medulla) showed areas of haemorrhage, microscopic granuloma, evidence of necrosis, and infiltration with round cells, epitheloid cells and fair number of multinucleated giant cells. A few of them were Langhan's type (Fig 2). Ziehl-Neelsen stain of the tissue did not show any acid-fast bacillus in brain or any other organ.

Discussion

Neurotuberculosis is always caused by secondary dissemination from a primary focus elsewhere in the body (3-5). In the CNS, tuberculosis can affect either the meninges or the brain parenchyma. CNS involvement

seems to be as high as 45% in paediatric patients with tuberculosis and it also accounts for 21% of all intracranial space occupying lesions as reported by Dastur and Lalitha [5]. Fortunately the incidence also seems to be declining [5]. However, tuberculosis as a cause of stroke is a rarity as mentioned in most of the Western textbooks [10]. As far as focal neurological deficit is concerned, hemiparesis was detected in 19% of cases of tubercular meningitis (TBM) by Thomas et al [7]. The pathogenesis of this vasculopathy in TBM is considered to be the periadventitial inflammation and proliferative subintimal fibrotic lesions consequent to chronic inflammatory processes [7,8]. Possibly there also exists strangulatory effect of **basal exudates which produce ischaemic softening of cerebral tissue causing sensory motor deficit [3,5]. It is more common to find involvement of middle cerebral territory than vertebrobasilar system.**

It is interesting to note that in most cases of cerebral arteritis and cerebral softening as described by Dastur and Lalitha [5], and by Dalal and Dalal [3], there have been some features of pulmonary tuberculosis or TBM to start with. We could not find a single report where reversible ischaemic neurological deficit was followed by recurrence of stroke or haemorrhagic event in 1¹/₂ to 2 years time, which has happened in our patient. The rapid progression of the second episode, which proved to be fatal, can only be explained by a rapidly progressive immunologic damage in an already sensitised individual. As in TBM, tuberculosis of brain parenchyma also has been attributed to an immunologic process [3,5,8,9]. Such antigen - antibody mediated Arthus-type reaction has been postulated in

many clinical settings [3,5] as well as in experimental study in monkeys [9]. This case highlights the importance of tuberculosis as a possible cause of stroke-in-young in countries like ours.

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