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Tuberculous Arachnoiditis

—A CASE REPORT FROM CENTRAL AFRICA AND A BRIEF REVIEW OF THE DISEASE

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INTRODUCTION

In most parts of Africa, where tuberculosis is endemic, tuberculous arachnoiditis remains a rare disease. This case is presented to illustrate the polymorphous course of the disease which probably due to its rarity is very scantily described in standard textbooks.

CASE REPORT

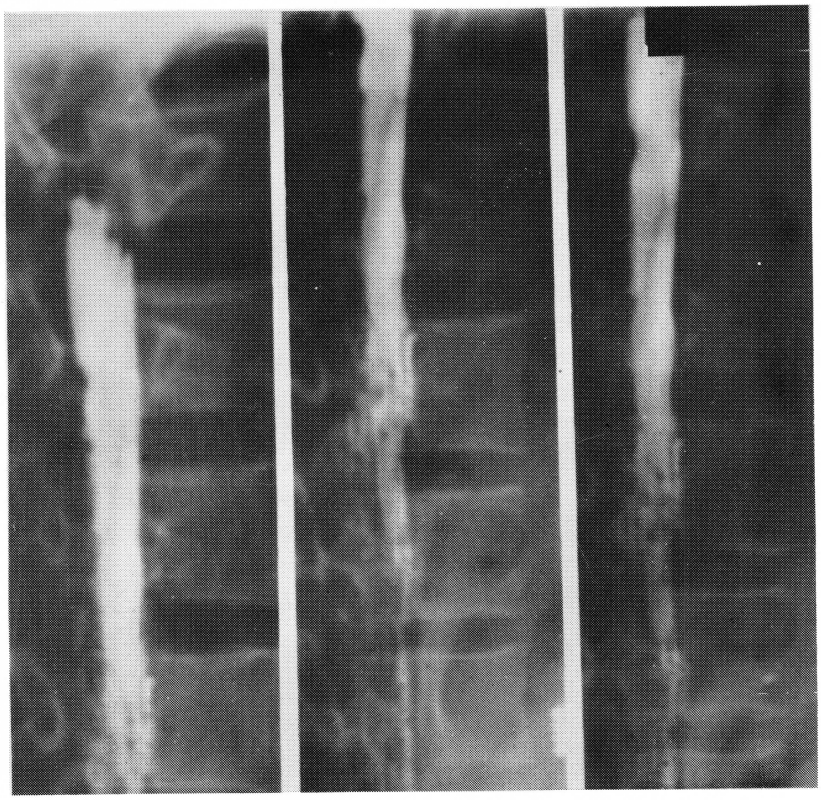
A young Zambian African female aged 21 years presented in a peripheral hospital with a history of acute and sudden onset of headache, three days prior to hospitalisation. The headache was accompanied by twitching of the right side of the face and vomiting. On examination she was found to have neck stiffness and flaccid weakness of the lower limbs. The cerebro spinal fluid was xanthochromic and protein content was 200mg%. A provisional diagnosis of subarachnoid haemorrhage was made and the patient was transferred to the University Teaching Hospital Lusaka, two days later. On arrival she was drowsy, disorientated and had marked neck rigidity and flaccid paralysis of both lower limbs. There were no signs of cerebellar or cranial nerve involvement and the sensory system as well as funduscopy were normal. The cerebrospinal fluid was still xanthochromic; proteins were now 4g%, sugar 48mg% and white blood cells 8/cu. mm.

The chest film showed miliary tuberculosis. At myelography several punctures were made before about three mililiters or cerebrospinal fluid was obtained and this congealed immediately (Froin's syndrome). At fluoroscopy, the column of contrast was sluggish and broke into linear fragments before obstruction, which was complete at the level of D10.

but found no direct evidence to incriminate tuberculosis.

In West Africa, the condition is rare. Osuntokun described 8 cases of arachnoiditis from the University Teaching Hospital Ibadan, during the period 1957 to 1970. It is not clear if any of these were due to tuberculosis. Handdock in Korle Bu hospital, Accra

FIG.



The fiture is a lateral film sequency of the myeogram showing *static appearances of the myodil column in the lumbodorsal region. The column is irregular with guttering and round tranradiant shadows. In the lower dorsal region the contrast column was sluggish and finally obstructed at the level of D10. These are features of spinal arachnoiditis.*

DISCUSSION

Tuberculous arachnoiditis has been most widely described from the Indian subcontinent [Ramamurthi (1961); Wadia & Dastur (1969)]. The disease seems to be much less prevalent in the other parts of the world.

Harris in a review of 750 patients with neurological disorders in a five year period in Kenya, described only one case and two others of a non-tuberculous origin.

In Uganda Billinghurst recognised spinal arachnoiditis as one of the commoner causes of myelopathy found no cases during the period 1968–1969. Cardozo et al in their review of tuberculous meningitis in Lusaka Zambia (1976) also found no case of a arachnoiditis.

Different authors describe different patterns of presentation of the disease. Kocen & Parsons observed that paraplegia normally complicates antecedent clinical symptoms by a period of months rather than weeks or days. In our patient both signs of meningeal disease and paralpegia occurred simultaneously. The onset was both acute and sudden differing from the insidious illness described by other workers (Cardozo

et al). The lowest cell count in the CFS in Cardozo's series was 100/cu mm. contrasting with the findings in our patient who had only 8 cells/cu mm. The low cell count in this patient however collaborates the findings of Taylor et al, who described a case with widespread miliary tuberculous involvement of the brain, but showed no pleocytosis over a period of 8 weeks. Their comment that the cell count correlates with the degree of meningeal involvement cannot be however relevant in this case.

We had regarded the rapid onset and progression of the disease, the co-existence of meningeal disease and paraplegia and the absence of significant plecocytosis as unusual in this condition. From Wadia's account however tuberculous arachnoiditis shows a polymorphous course and the spectrum of presentation includes a rapid clinical course, a subacute progressive variety simulating a myelitis, and a slow form which resembles a tumour.

The CSF cellular response is often not remarkable, and the sugar content is not reduced in all cases. The protein content is however normally high.

Radiology

Myelography is the confirmatory investigation. The myelographic features need not however be specific but many features are quite characteriestic and taken together usually diagnostic.

(a) As in our case the lumbar puncture may be dry and when some CSF fluid is drawn, only a small amount may be obtained.

(b) The contrast Column on fluoroscopy usually breaks into longitudinal stringly columns likened by Elington to the guttering of a candle.

(c) The contrast columns may show small irregular or round defects and moves very slowly in the involved segment.

(d) Partial or complete block with an ill defined edge (contrasting with a well defined margin in mechanical obstructions due to tumour, disc etc) is very often present.

Anciliary Investigations

Careful examination of the chest and spine for tuberculous process is advisable, and in the secondary form of the disease this may reveal the primary focus **e.g.** miliary tuberculosis of the lungs (as in our case) or spinal tuberculosis (which is more common).

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