Tuberculoma Brain

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History

The first operated case was reported by Macewen in 1883[1]. In 1893 Starr reviewed that in 300 cases of brain tumours, 52% cases were tuberculomas[9]. Cushing at the turn of century reported 30-40% incidence[3].

Incidence

In large Neurosurgical centres of the world, in first half of this century, incidence reported is 1.7%.

Olivercrona in 1967, in his series of 5250 cases of space occupying lesions, reported only 0.9% as tuberculoma[7].

So there is no doubt that tuberculoma brain are decreasing in most countries. In Madrid 5400 cases were operated for brain tumours in the past 30 years, and the incidence of tuberculoma was one percent.

In India in 1968, the incidence was reported as 20%. In the past five years the incidence has dropped to 12 percent.

Between 1987-1989, eleven cases were seen of T.B. brain in LRH, Peshawar and in the past two years only 5 cases have been operated and confirmed as tuberculoma brain. In a total tumour population this incidence has been in the region of 16%. This might seen high, but it is attributed to the influx of Afghan population in which incidence of tuberculosis is generally high.

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Etiology/Pathology

Haematogenous spread from other parts of the body is the source. Wilson suggests that if there is no evidence of T.B., any where in the body, then search is incomplete.

At Surgery, the lesion is usually nodular, hard, avascular, beneath the cortex and varies in size. Cerebral oedema is always present.

Multiplicity of the lesions is often seen; and in our series lesions were present (more than one) in posterior fossa, and in the cerebral cortex. Posterior fossa lesion presented with hydrocephalus, and few of them were brought in a state where they required immediate ventricular tapping and ventricular drain was put in, with subsequent shunting. Some tuberculoma may even remain symptomless and disappear with chemotherapy.

Tubercular meningitis with intra-cranial tuberculoma is present in 3% cases.

Histology

Tuberculoma gives a usual picture of granulomatous lesion, with necrotic areas and Langhans cells. There is central caseation, rarely forming abscess in brain.

Diagnosis

In about half the patients operated, there is history of tuberculosis else-where in the body; and usually presentation of intracranial lesion is the first symptom. Intracranial tuberculoma effects young children and early adult. In our experience about 80% of the children were below 13 years of age, 70% in the posterior fossa and 30% in the cerebrum.

Like any other intracranial tumour, signs and symptoms of increased intracranial pressure and localization are present. Epilepsy appears in 70-85% cases; hemispheric disturbances in 50- 70 percent cases and cerebellar signs

in 70-80% cases. Tuberculous bacteremia is common and can be seen in half the cases. Calcification on X-Rays skull is seen in only 6% cases. C.S.F. is normal in majority of the cases, and is not done these days in most of the centres.

Carotid angiography is very useful. Rammamurthi has suggested positive identification on carotid angiography[8].

CAT scanning has revolutionized the diagnosis as it can demonstrate one or more lesion. The most common features are unenhanced; CT shows an area of low attenuation, but after enhancement, the capsule is seen. Occasionally calcification can be seen in the lesion.

Treatment

Cushing at the turn of the century said that if tuberculoma is surgically removed, T.B. meningitis occurs in 3 months[3]. With the advent of modern chemotherapeutic agents this statement is not valid. Operative removal should be followed by chemotherapy for 3-5 months. The prognosis in cerebellar tuberculoma is poor, if the lesions are multiple and causing hydrocephalus.

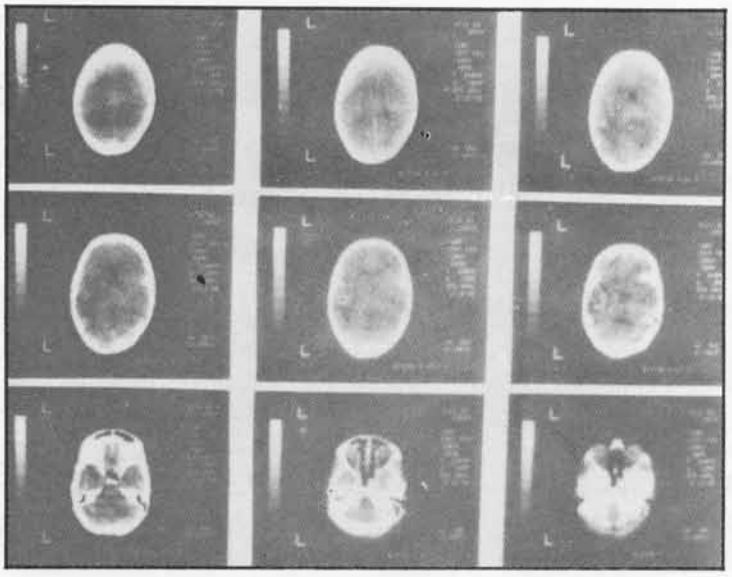
Multiple tuberculoma are treated with chemotherapy.

A Typical Case of Tuberculoma Brain.

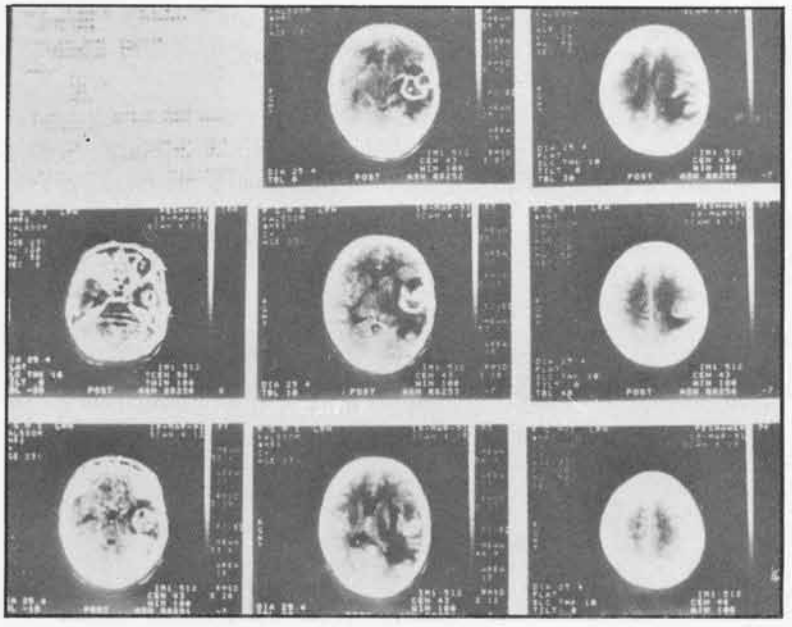
Case Report:

K. Bibi, age 25 years, married with 2 children, from Swabi, N.W.F.P. was admitted to Neuro-Surgical Unit, L.R.H. Peshawar in March 1991 with the following complaints:

Headache	One year
Grand mal Epileptic fits	Six months
Vomiting off and on	



1. C.T. Scan (Six months before)



2. C.T. Scan (Six months after)
Showing lesion increased and causing compression

Headache had recently increased and in past few weeks she had cough and fever at night with occasional dyspnea.

General physical examination was unremarkable.

She had no neurogical defect except early papilloedema. She was slightly anaemic; her HB being 8.8; ESR was 18 mm; chest and skull X-Rays were unremarkable. A CT Scan done in Lahore on 02.12.1990 vide code No 015726 Hk, reported as follows:-

"The contrast CT shows an ill defined non-homogeneously enhancing area in the left temporo-parietal region. Advised left carotid angiography to add more information".

However, angiography was not carried out and the patient reported here in March 1991, and a CAT scan showed a left temporo-parietal well-enhancing lesion with mass effect.

A temporo-parietal craniotomy was done, and a solid hard, relatively avascular lesion, arising from the base of temporal lobe was excised totally.

The whole tissue was sent for histology.

The patient made an uneventful recovery with no residual effect. Her epileptic fits improved after excision of the lesion.

The histology report was:

"Section shows fragments of meningies and brain, containing granulomas composed of epitheloid cells, lymphocytes and Langhans type of giant cells. No malignancy. Consistent with tuberculoma brain."

Conclusion

Tuberculoma brain is still not uncommon in developing countries and particularly those areas of the world harbouring refugee problems with under nourishment and poor hygienic states. The modern chemotherapy and diagnostic facilities have gone a long way to check the intracranial complication.

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