

TEACHING FILE

LOWER MOTOR NEURON FACIAL PALSY IN CNS TUBERCULOMAS

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Clinical Problem : An 8 years old boy presented with fever for 19 days with left sided upper limb monoparesis and left lower motor neuron (LMN) facial palsy for 1 day. Cerebrospinal fluid (CSF) showed 25 mg/dl proteins, 75 mg/dl sugar and no cells. MRI brain showed multiple granulomas in right thalamus, left frontal cortex, right temporal lobe and right side of medulla and pons. There was no infarct. Chest X-Ray was normal. Mantoux test was positive. He was started on antituberculous therapy (ATT) along with steroids. A month later, repeat MRI showed regression in size of parenchymal granulomas. Steroids were tapered and stopped in 4 months. After one year of therapy, his facial palsy and monoparesis had resolved and MRI showed no change as compared to previous scan. He was continued on same ATT.

Question : How common is LMN facial palsy with CNS tuberculomas?

Expert Opinion: Central nervous system (CNS) manifestations of tuberculosis (TB) include TB meningitis (TBM) and tuberculomas in the brain and the spine. (1) Tuberculomas may be single or multiple and usually are found in the frontal and parietal lobes. (2) The clinical manifestation of tuberculoma depends on its location. Lower motor neuron (LMN) facial palsy occurs commonly in children due to infection and trauma but mostly a cause cannot be ascertained and it is categorized as Bell's Palsy. (3) A few rare cases of facial palsy caused by a tuberculomas have been reported in children mostly occurring in disseminated TB. (4,5) Amongst the infections causing facial nerve paralysis in children, the most common infections include herpes simplex, otitis media and chicken pox. (4) TB has however been listed as one of the infections that could cause facial palsy usually in the form of tuberculous otitis media. (3) A very rare case of a tuberculoma in the mastoid bone leading to facial palsy has been reported. (6) A brainstem tuberculoma has been reported in a preschool going child which presented as eight and a half syndrome that is paralysis of the facial nerve along with one and a half syndrome. (2) Acute central paralysis of the left facial nerve along with progressive hemiplegia and severe ataxia due to a cerebral tuberculomas was reported in a 16 year old boy as a late clinical manifestation of military tuberculosis. (3) His condition resolved with continuous ATT therapy and steroids. A case of a pontine tuberculoma presenting as Foville's syndrome-contralateral hemiplegia, homo-lateral VI and VII nerve palsies (7) thus suggesting that rarely does facial paralysis and hemiparesis occur as sole clinical manifestation of tuberculomas and usually presents with other mixed symptoms. A space occupying lesion or tumour occurring in the cerebropontine region has been reported as a known cause of facial palsy. (4) Thus, the left sided facial palsy and monoparesis found in our patient could have been caused by the tuberculoma in the right pons.

In cases of strokes, central facial paralysis are usually caused when infarcts occur either in the pontine arteries or a lacunar infarct affecting fibres of the internal capsule going to the facial nucleus. (8) However, no sign of infarcts were seen in our patient.

Studies have shown that prompt treatment with ATT and corticosteroids has proven to be a successful non surgical treatment for intracranial tuberculomas. (9) The facial palsy and monoparesis in our patient resolved with ATT and steroids.

Thus it can be concluded that though extremely rare intracranial tuberculomas can present in the form of facial palsy in children. Prompt diagnosis and treatment is required to prevent the tuberculoma from becoming life threatening especially if the brainstem is involved.

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