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## Cyproheptadine in Severe Anorexia

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Tuberculous meningitis (TBM) is known to give rise to many complications and sequelae, including hypothalamic damage. We report a case of TBM with marked anorexia and its management.

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## Case Report

A 4-year-old girl was diagnosed to have TBM on the basis of history, abnormal CSF findings and a CT scan showing moderate hydrocephalus and basal exudates. She was treated with a combination of streptomycin, isoniazid, rifampicin and pyrazinamide. She also received oral phenobarbitone for convulsions and mannitol and acetazolamide. The sensorium improved on the 6th day, only after a ventriculoperitoneal shunt was inserted. Her neurologic status and mentation was normal within 30 days of therapy. However, she had to be discharged on tube feeds since she had severe anorexia.

A month later, during the second admission for drug induced hepatitis she was noticed to have signs of undernutrition, anemia, xerosis and edema feet. Oral feeding was unsuccessfully attempted again after improvement in liver functions. There was no evidence of shunt dysfunction at anytime. A repeat lumbar puncture showed considerable improvement in the CSF. A psychiatry opinion ruled out maternal neglect or disturbed parent-child relationship.

Eight weeks after the first admission, oral cyproheptadine was started in a dose of

0.5 mg/kg/24 h and continued for 4 weeks. The child developed a voracious appetite and showed an average weight gain of 250 g/day over the next 6 days, the trend continuing even after stopping cyproheptadine. She gained 4.4 kg over 12 weeks and a total of 6 kg over 6 months.

### Discussion

Appetite is controlled by two hypothalamic centers, one in the lateral area of tuber cinerium which is the center for hunger or appetite; the other, the ventromedial nucleus of thalamus is concerned with satiety. Neurotuberculosis can damage both these centers. A lesion in the lateral nucleus can produce total loss of appetite resulting in emaciation(1).

Cyproheptadine, an antihistaminic with predominant antiserotonergic action is often misused for its effect of producing weight gain. Its mechanism of appetite stimulation is not known. The drug is said to interact with cortisol and insulin biosynthesis and interferes with regulation of growth hormone secretion(2). A loss of appetite due to damage to lateral hypothalamic nucleus alongwith unopposed activity of the satiety center probably resulted in the extreme anorexia in our patient. The

antiserotonergic action of cyproheptadine possibly suppressed this center(3), resulting in improved appetite in this case.

In conclusion, we suggest the use of drugs like cyproheptadine only in severe cases of anorexia, secondary to a possible hypothalamic lesion as in cases of TBM or encephalitis.

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