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CASE REPORT **OPEN ACCESS**

Encephalitis as a result of severe roundworm infestation in a malnourished child

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A B S T R A C T

The clinical presentation of ascariasis depends on the intensity of infection and the organs involved. Although most individuals are asymptomatic, few can present acutely with intestinal obstruction and extra-intestinal complications. Extra-intestinal complications can involve the pulmonary, hepato-biliary, renal and central nervous systems. [1-3] Encephalopathy as a presenting feature of ascariasis is unusual and rarely described.

We encounter an 8-year-old girl with severe acute malnutrition with a rare presentation of encephalitis with multiple episodes of convulsion because of heavy infestation with roundworms.

Keywords: SAM. Ascariasis, Encephalopathy

INTRODUCTION

Case report: 8-year female child brought by parents with h/o fever x 10 days, convulsion 3-episode x 10 days, Cough cold x 3 days and altered sensorium x 2 days. The patient was all right 10 days back. When she developed a fever high grade with chills, rigours she had one episode of convulsion, which was a generalised tonic-clonic type and lasted for 10 min with post-ictal drowsiness of 30 min, treated as febrile convulsions in a private hospital for two days. The patient had another two episodes of convulsion with loss of consciousness, for which she was admitted to our centre. Between 2 episodes, the patient had a fever on and off low grade with a cough and cold three days before hospitalization. The patient had altered sensorium through decreased playfulness and became excessively sleepy. History of recent weight loss present. Past History was not significant;

The patient was living with grandparents, with subnormal living conditions and had a habit of playing with soil.

On admission, the patient was drowsy with GCS 10, spo2 88 %, tachycardia, and tachypnea with crepitation on the right side of the chest; the patient was pale and malnourished and had grade 3 PEM, according to IAP.CNS examination suggested hypertonia with brisk reflexes and no signs of meningitis. We kept a diagnosis of viral encephalitis/ cerebral malaria with aspiration pneumonitis. We had started treatment with Antibiotics, antiepileptics, and mannitol given because of status epilepticus. Inj. Dexamethasone, inj. Acyclovir and artesunate started with supportive care.

Investigations s/o moderate anemia with eosinophilia.CSF study suggestive of proteins 50mg/dl and sugar 55mg/dl with lymphocytic predominance. Chest x-ray s/o parahilor upper lobe consolidation of the right lung (fig 1). LFT, electrolytes, urine examination, HIV and Peripheral smear for malarial parasite were normal was 15, and ESR 30 mm at the end of 1 hour. The urine examination was normal. Neuroimaging was suggestive of mild cerebral and cerebellar atrophy and degenerative changes. Inj. methylprednisolone was started.

The patient became vitally stable, but the sensorium did not improve and had cortical blindness, too. We ruled out all possible infection's endemic to our area, like tuberculosis, scrub typhus, dengue, post-COVID MIS and HIV status. All reports came out to be negative. After 72 hours, a 10 to 12-cm worm came out through the external Nare, and the histopathologist confirmed it as female ascariasis. (Fig.2)

Stool examination revealed many fertilized and unfertilized eggs of roundworms. (fig.3).Ultrasonography of abdomen s/o echogenic tubular structure in the small intestine.
Treatment started with syrup albendazole through a Ryles tube and a 10% dextrose enema. Piperazine citrates 75mg per kg for two doses were given.
Many worms came out through the anus. (fig .4)
After five days, the patient's sensorium started improving and became oriented. The patient was discharged with proper diet management, parental education on a high-protein diet, and hygiene. On follow-up, the patient recovered entirely with adequate weight gain.

DISCUSSION

Extraintestinal complications of ascariasis involve the pulmonary, hepato-biliary, and central nervous system. Ascariasis is also known to mediate an immunological response. The clinical features of ascariasis relate to both larval and adult stages and depend on the intensity of infection. Migration of larvae through the lungs, particularly in heavy infestation, may cause a severe immune-mediated inflammatory response resulting in pneumonitis, liver enlargement and generalised toxicity (1).

The raccoon roundworm, Baylisascarisprocyonis, causes zoonotic visceral, ocular, andneural larva migrans. It can lead to devastating encephalitis in young children. Infection occurs mainly at raccoon latrines, and risk factors include contact with raccoon latrines, pica/geophagia, age under 4 years, and being male. This infection is prevalent in the United States, where raccoons are found. (7)

Toxic cerebral symptoms simulating encephalopathy or meningitis have been reportedearlier. Thenervous symptomatology so often associated with ascariasis has been attributed to either the adverse effects of toxins produced by larval or adult worms ortheir metabolites like acetaldehyde(3). Another extended hypothesis is an allergic orimmune mechanism involving an antigen-antibody type of reaction in hypersensitivenervous tissue(3). However, the exact mechanism still remains far from clear.

In conclusion, ascariasis should be considered as a differential diagnosis for a variety of obstructive hepato-biliary pathologies and unexplained encephalopathy in a tropical country.(6) Worm encephalitis should be considered as one of the differential diagnoses for unexplained encephalitis, multisystem involvement, particularly in tropical countries, after ruling out endemic infection.

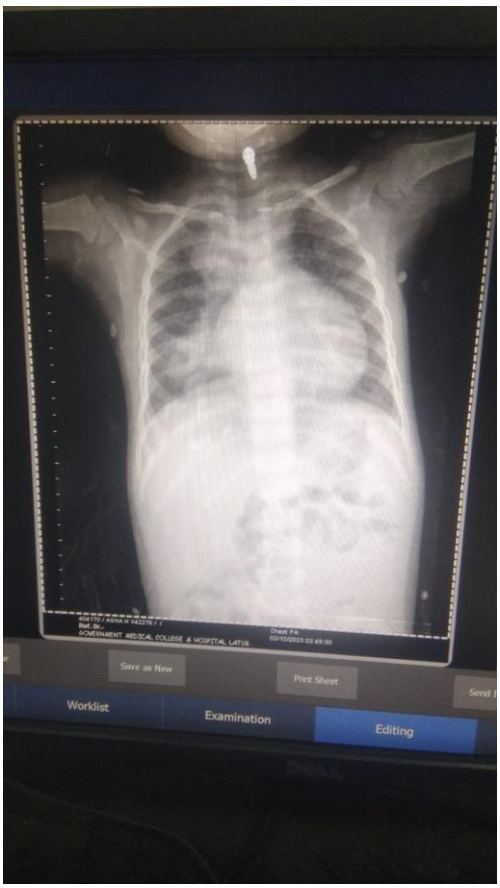

Fig 1: Pneumonitis right lung

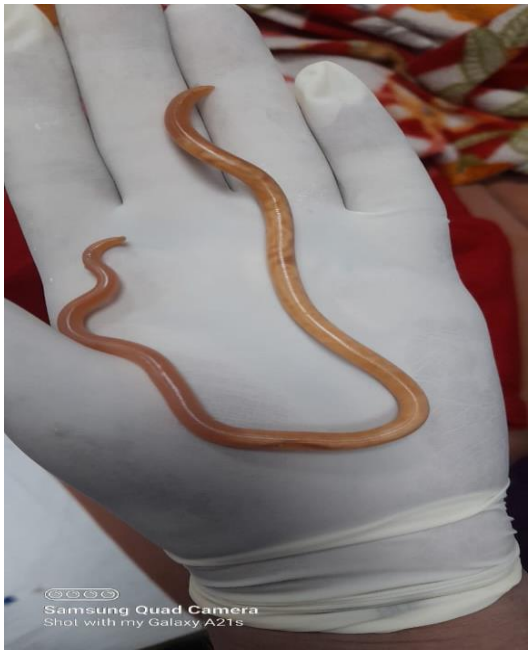

Fig 2: Ascaris lumbricoid (10 -12 cm worm)


Fig 3: fertilized and unfertilized eggs of Ascaris lumbricoid

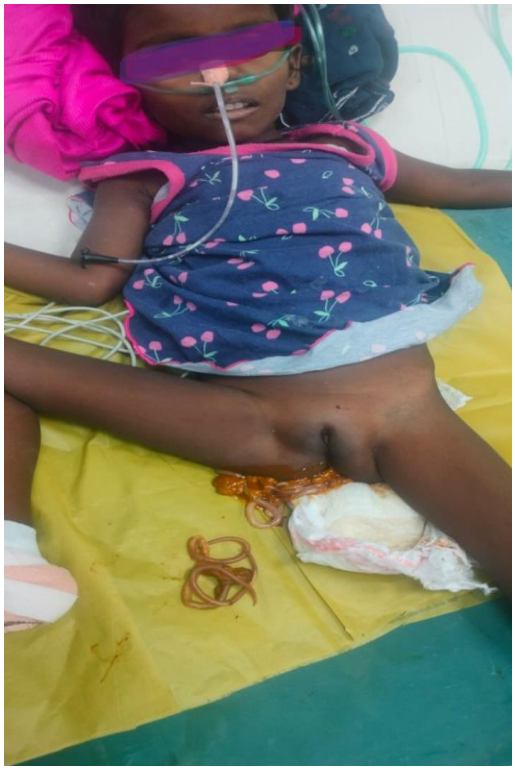

Fig 4: worms coming out after dextrose enema.

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