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ACUTE CEREBELLAR SYNDROME IN A PATIENT OF FALCIPARUM MALARIA

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ABSTRACT

Malaria is a vector-borne disease transmitted by the bite of an infected female anopheles mosquito presents with varied clinical manifestations. Neurological manifestations include headaches, confusion, convulsions, hemiplegia, ataxia, cerebral palsy, cortical blindness, and Guillain-Barre syndrome (GBS). We are presenting a case report of acute cerebellar Syndrome in a 45-year-old male patient who presented with fever and positive for Plasmodium falciparum malaria.

Key words: *Plasmodium falciparum*, Malaria, Cerebellar ataxia.

INTRODUCTION

Plasmodium falciparum malaria is the most common imported parasitic disease in Portugal. Malaria should be suspected in anyone with fever or history of fever who has returned from or previously visited a malaria endemic area, regardless of whether prophylaxis was taken or not. Cerebellar involvement in P. falciparum malaria can occur during the acute stage of fever, as sequels of cerebral malaria in survivors, in the form of delayed cerebellar ataxia (DCA) and as a side effect of drugs [1]. Two syndromes of cerebellar ataxia have been recognized: acute (or early) cerebellar ataxia and delayed (or late) cerebellar ataxia [2].

Acute cerebellar ataxia has been described in patients with cerebral malaria who were found to have cerebellar ataxia on recovery from coma. Cerebral malaria was defined as asexual parasitemia in a febrile patient with an unrousable coma of more than six hours for which no other cause was evident [3]. delayed cerebellar ataxia (DCA) refers to patients who developed cerebellar ataxia shortly after full recovery from an uncomplicated attack of malaria.

Case report

A 45yrs oldman from a malaria endemic area admitted in Maharana Bhupal Government Hospital, Udaipur with following chief complains: -

1) Fever which was high grade, sudden rising, and intermittent type from 6 days.
2) Nausea, vomiting and vertigo from 2 days.
3) Unsteadiness on lying to sitting and walking from 2 days.

There was no history of altered mentation or sensorium, seizure, incontinence, diplopia. Patient had no history of hypertension, diabetes, tuberculosis and any major operative history. No any similar episode of illness experienced by the Patient in past. Patient was vegetarian and having good habits (non smoker, non alcoholic, non tobacco chewer). All family members were healthy. Patient had no history of any drug ingestion or drug abuse.

On admission patients vitals were: - Pulse-96/mint. Regular, BP-130/80 mm of hg, RR-20/mint., Temp-102 degree F (orally). Patient had mild pallor but there was no cyanosis, clubbing, oedema, icterus, and lymphadenopathy. Patient had mild splenomegaly which was firm, smooth, round bordered, and non-tender. Respiratory system and CVS was normal on examination. On neurological examination patient had:- normal mental

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functions, all cranial nerves normal, and normal bulk, tone, power, reflexes. Planter was b/l flexor. Patient had truncal and gait ataxia (unable to sit and walk without support). Patient was positive for cerebellar sign as:- dysarthria, dysmetria, past pointing, dysdiadokinesia, incordination, tremor. Patient had no nystagmus. All sign of meningeal irritation was absent.

Patient was fully investigated:- Hb-10.1 gm/dl,TLC- 10700/mm3, ESR-71, PBF - ncnc with ring stage of pl.falciparum. CARD test and MPQBC were positive for pl.falciparum. Patients urine was clear. RBS-108 mg/dl, Urea- 51.30 mg/dl, Creatinine -1.4 mg/dl, Bilirubin- 2.20 mg/dl, SGOT-375.10 U/L, SGPT-495.70 U/L, Normal electrolytes (Na 134 meq/L , K 3.5 meq/L). Patient ECG and Chest x-ray were normal. Fundus was normal in both eyes. MRI Brain and spine was absolutely normal.

In view of clinical finding, splenomegaly, blood biochemistry, MPQBC and Slide test positivity, our diagnosis was malaria fever with mods, malarial hepatitis, and acute cerebellar syndrome or cerebellitis or cerebellar malaria. Patient was treated according to standard guideline of WHO for malaria. Inj. Quinine and inj.clindamycin and i.v. fluids were given. Patient has started to improve within two days, after full course of treatment. Patient was free from symptoms which were present initially of illness. Repeat liver function test were also improved and patient discharged in a stable condition.

DISCUSSION AND CONCLUSION

Cerebellar syndrome or cerebellar ataxia is a rare complication of *Pl.falciparum* malaria [4]. Cerebellar involvement may occur in complicated as well as of uncomplicated malaria. Dominant cerebellar involvement could be part of cerebral malaria. Some Immunopathologic events occur in patients with malaria include polyclonal activation which leads hypergammaglobulinemia and formation of immune complexes, immunodepression, and release of cytokines. Cytoadherence of the parasitised erythrocyte to vascular endothelium occur in P. falciparum malaria [5]. It may lead to obstruction of blood flow and capillary damage with resultant vascular leakage of protein and fluid, edema, and tissue anoxia in the brain, heart, lung, intestine and kidney. So mechanism of development of cerebellar complication in falciperum malaria, is due to obstruction of microcirculation in cerebellum by sludging of parasitised RBCs and malarial vasculopathy [6]. Severe gait ataxia and truncal ataxia in this patient indicates the involvement of midline cerebellar structures. Purkinje cells are most sensitive part of cerebellum to damage due to pyrexia [7]. Cerebellar syndrome or cerebellar ataxia is a rare complication of *Pl.falciparum* malaria.

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