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Cerebellar Syndrome in Malaria

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Cerebellar syndrome, an unusual manifestation of malaria, is already reported by different authors(1-3). The diagnostic triad of such cerebellar syndrome is characterized by cerebellar signs with fever, presence of malarial parasite in blood and response to antimalarial therapy(1). Recently, we came across five cases of malaria with cerebellar signs which presented in different manner in contrast to previous reports. So, we would like to present the case reports to highlight our point.

Case Reports

An alarming rise in incidence of cerebral malaria is noted in Surat and surrounding areas of South Gujarat in last few years. Cerebral malaria constitutes 2-5% of admissions in Pediatric Ward of Government Medical College, Surat (Unpublished data). We encountered all five cases in last three months from July 1991 to September 1991.

The age group of patients varied from 3-10 years. None received prior treatment before admission. Only one patient admitted with isolated cerebellar signs with behavioral changes and presentation of remaining patients fulfilled the criteria of working definition of cerebral malaria. Bilateral cerebellar signs with unsteady gait were present on admission in two cases and appeared after third day onwards in remaining cases. The average duration of cerebellar signs was one week except in one case. The peripheral smear for malarial parasite was positive in four patients. All patients were treated with quinine (10 mg/kg/d × 10 days) considering the high incidence of chloroquine resistant malaria in our area (Unpublished data). The clinical recovery was complete in all cases (*Table*).

Discussion

The classical malarial syndrome now occurs in only 50-70% cases of malaria, the rest being atypical presentation like abdominal symptoms, bleeding disorders and combination of neurological defects(4,5). The cerebellar involvement in malaria was reported by Deaderic in 1909 and later on, Ringdon *et al.* in their pathological study demonstrated a definite involvement of cerebellum in patients who died of cerebral malaria as well as in experimental animals(6).

The pathogenesis of cerebellar signs is similar like cerebral malaria which results from obstruction of micro-circulation due to sludging of parasitized RBCs and direct

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TABLE—*Summary of the Cases*

	Cases				
	1	2	3	4	5
History					
Age (yrs)	8	4	10	5	3
Sex	Male	Female	Female	Male	Male
Prior treatment	–	–	–	–	–
Clinical presentation					
Fever	+	+	+	+	+
Splenomegaly	+	+	+	+	–
Altered sensorium	–	+	+	+	–
Convulsion	+	+	–	+	–
Behavioral changes	+	–	–	–	+
Focal neuro. deficit	–	–	–	–	–
Cerebellar signs					
A. Onset	O/A	3rd day	4th day	4th day	O/A
B. Duration	6	8	14	7	5
Laboratory investigations					
PS for MP	*P. falciparum*	*P. vivax*	–	*P. vivax*	*P. falciparum*
TLC	8100	6700	7000	8400	5000
CSF	N	N	N	N	N
X-ray chest	N	N	N	N	N
Fundus	N	N	N	N	N
Treatment					
Quinine	+	+	+	+	+
Duration of hospitalization	8	14	20	12	8

O/A—On admission.

malarial vasculopathy, causing extensive damage to purkinje cells of cerebellum(4,6).

The clinical presentations in our cases differ in following aspects from previous published reports(1-3): (*i*) all cases were well documented evidence of cerebral malaria except one case which was associated with hyperkinetic behavioral changes in contrast to isolated presentation of cerebellar syndrome, reported by previous studies. (*ii*) Cerebellar signs were seen in three cases from third day onward, after apparent recovery of altered sensorium,

following treatment. As quinine is not reported to cause cerebellar ataxia either as a side effect or toxicity, it cannot be implicated as a etiological agent for these cases. (*iii*) We had two cases of cerebral malaria, caused by *P. vivax* with the cerebellar signs in the present study. The previous studies have shown the association of cerebellar syndrome with *P. falciparum* only. The difference can be explained by changing epidemiological pattern of cerebral malaria. (*iv*) In our study, cerebellar signs, once appeared, lasted for an average of one week and disappeared gradually with treatment. Previous reports have mentioned a complete recovery within 48 hours with antimalarials. Such prompt response was not seen but recovery was virtually complete in all our cases.

The knowledge regarding the spectrum of neurologic symptoms of malaria is increasing with passage of time which stretches now from altered sensorium and convulsion to behavioral changes and even frank psychosis. So, we would like to coin the broad terminology like "Neuromalaria" instead of using restricted terms like "Cerebral or Cerebellar Malaria", to justify the same pathology having varied presentations.

So, we conclude that cerebellar syndrome, although an uncommon manifestation, can be seen in cases of cerebral malaria as well, as in isolated cases. Cerebellar signs can follow an unusual prolonged course even after institution of antimalarial therapy but clinical recovery is always complete.

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Antithymocyte Globulin in Aplastic Anemia

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Antithymocyte globulin (ATG) is at present the best available treatment for patients with severe aplastic anemia (SAA) who are not eligible for bone marrow transplantation (BMT)(1). Clinical trials of this drug in India particularly in children are limited(2). We report a case of plastic anemia treated successfully with ATG.

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