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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Case Report

A 10-year-old male child was admitted with the complaints of progressive pallor of 5 months duration. Prior to this he had suffered with fever for 15 days and was treated by a private practitioner. The nature of drug given was not known. There was no history of bleeding from any sites. He had already received 3 blood transfusions, hematinics and androgenic steriods, without any relief. On admission he had severe pallor. There was no lymphadenopathy, hepatosplenomegaly and evidence of bleeding. Systemic examination was also normal. The hemogram showed Hb 4.5 g/dl, TLC 1400/mm^3, absolute neutrophil count 224/mm^3, reticulocyte count 0.1% and platelet count 30,000/mm^3. Bone marrow biopsy showed 10% cellularity, marked increase in fat spaces, reduction in myeloid and erythroid cells, no mega-karyocytes and only small islands of cells mainly lymphocytes, plasma cells and erythrocytes, thus confirming the diagnosis of SAA(1). Since he had already received androgens earlier, initially short term methylprednisolone (MPN) and later on long term methylprednisolone therapy was given, but there was no response. Finally, ATG alongwith adjuvant therapy was tried. The details of treatment are summarized in the *Table*.

TABLE–*Treatment Schedule*

Drugs	Dose	Duration	Response	Blood Requirement
A. *Prior to Admission*				
Adroyds	3 mg/kg/day	2 months	No response	2-3 weekly
B. *After Admission*				
Day 3, MPN	1 g alternate day IV	Inj	Transient increase in Hb, neutrophils platelets and reticulocytes.	2-3 weekly
Day 35 MPN	30 mg/kg/day IV	3 days	Transient improvement in platelets & reticulocytes. No consistent effect on Hb, neutrophils.	2-3 weekly
	10 mg/kg/day	7 days		
	10 mg/kg/day	7 days		
	5 mg/kg/day	7 days		
	2 mg/kg/day	7 days		
Day 180, ATG (ATGAM, Up John USA)	10 mg/kg/day IV	12 days	Hb, neutrophils, platelets & reticulocytes. Consistent and sustained rise.	Nil
MPN	15 mg/kg/day IV	3 days		
	10 mg/kg/day IV	3 days		
	5 mg/kg/day IV	3 days		
	2 mg/kg/day IV	3 days		
Day 194, Adroyds	2.5 mg/kg/day oral	3 months		

During and after the treatment with ATG no side-effects were encountered. Hemogram report 1½ year after the treatment showed Hb 11 g/dl, TLC 7200/mm^3, neutrophils 2736/mm^3, reticulocyte count 2% and platelet 1.5 lac/mm^3. Bone marrow biopsy shows a cellularity of 20% with a few megakaryocytes and slightly bigger foci of hemopoiesis. As per the criteria for response to ATG therapy(3,4), our patient can be said to have attainted complete response.

Discussion

The exact pathogenesis of SAA is not known but the concept of alteration in the immunological regulation is strongly believed. The role of immunosuppressive therapy with ATG in the management of SAA is now well established in the West(2), the clinical response ranging from 14 to 85%(5,6). In India the drug is still on experimental therapy, its preliminary experience, particularly in children is not encouraging(2). Our patient has responded well and after attaining complete response within 3 months of treatment, has maintained normal blood counts without any transfusion for 18 months after stopping therapy. This could be due to absence of poor prognostic factors like infection and bleeding. Desired response to ATG may also not be observed if the defect is in the stem cell rather than in the microenvironment influenced by immunological dysregulation. We did not encounter any side effect during or after the therapy probably due to simultaneous use of IV methylprednisolone as adjuvant therapy(1).

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A Satellited Metacentric Marker Chromosome in a Phenotypically Normal Male (Transsexual)

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There have been reports of karyotypes including an extra small metacentric chromosome. Most of these chromosomes have been found in patients with congenital malformations and/or mental retarda-

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