On the ninth day, the CSF contained a protein level of 133 mg/dl and one lymphocyte/mm³. Electrophysiological study was performed on the twenty first hospital day. Motor nerve conduction of median nerve was 34 ms⁻¹ with a distal motor latency of 4.5 ms (normal, up to 4.2). F responses were not obtained. Distal sensory nerve conduction velocity of median nerve was 53 m/s, with sensory action potential being small (1.5 ìV). The extensor digitorum brevis muscle was denervated and motor conduction along the peroneal nerve could not be performed. Latencies from the caput medusae to the tibialis anterior was slightly prolonged, but the M-wave was very decreased (0.1 mV).

Electromyography of the deltoid, abductor pollicis brevis, and extensor digitorum anterior muscles revealed a pattern of discrete activity with an increase in mean duration of individual motor unit potentials. Fibrillation potentials and positive waves were only observed in the deltoid muscle. Six months after the onset of illness the patient still had pronounced proximal (MRC 2 to 3) and distal (MRC 3 to 4) weakness.

The diagnosis of GBS was strongly supported by the reduced number of circulating platelets, increased number of megakaryocytes, absence of splenomegaly and the response to prednisone. Specific IgG antibodies directed to platelet membrane glycoprotein antigens have been demonstrated in patients with chronic ITP, confirming the autoimmune nature of this disease. In acute ITP following a viral infection, the thrombocytopenic factors, be it specific platelet IgM antibodies or immune complexes, have not been defined. Failure to detect antibodies on platelets and in the serum of our patient might be the result of limited sensitivity of the immunofluorescence test.

This patient also fulfilled the diagnostic criteria for GBS. This is an inflammatory demyelinating neuropathy in which both protein and lipid antigens in peripheral nerve myelin are the target of immune attack, but the pathogenesis is still debatable. Experimental allergic neuritis induced by immunization with the P2 protein has been considered a disease of cellular immunity and the experimental model of GBS. Alternatively, during acute-phase illness the serum of patients with GBS contains complement-fixing antineural platelet IgM antibodies. These antibodies bind to a neutral glycolipid in the myelin that has yet to be completely identified. Circulating immune complexes may constitute an additional type of pathogenetic mechanism to produce demyelination.

GBS rarely occurs concurrently with another autoimmune disorder. Coexistence of antibodies to factor VIII and peripheral nerve myelin has been demonstrated in a patient with chronic inflammatory polyradiculoneuropathy complicated by a coagulation disorder. The present association of GBS and the bleeding disorder due to ITP, as described in our case, has only been reported once previously.

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Isolated cerebellar syndrome: an atypical form of cerebral malaria

The neurological manifestations of malaria are usually associated with the febrile attack, their outstanding features being the seizures and an impaired consciousness. We report a case of isolated cerebellar syndrome, a more benign complication of malaria, not related to the febrile attack.

A 31 year old French man, with no past medical history, was admitted to our hospital on May 21 1989 at another hospital for a rapidly progressive ataxia. Four months previously, the patient worked as a member of the French Cooperation Group in Burkina Faso, and did not take regular prophylactic anti-malaria drugs. In January 1989, the patient had an attack of fever (40°C), headaches, vomiting and diarrhoea, which was diagnosed as malaria and treated successfully with chloroquine. On 15 March 1989, he experienced rapidly progressive dysarthria and an unsteadiness on walking. A week later, the patient was referred to our hospital.

On admission, he was afebrile, but appeared chronically ill and complained of severe fatigue. There was a recent history of weight loss. Cardio-pulmonary and abdominal examinations were normal. There was no rash or lymphadenopathy. Neurological examination revealed a cerebellar syndrome interfering with a normal gait, and a less severe bilateral cerebellar ataxia. There was no abnormality of cranial nerves, neither was there any nystagmus, or motor or sensory deficit. Tendon reflexes were present bilaterally, but were more prominent on the right side.

The following laboratory studies were normal: complete blood count, erythrocyte sedimentation rate, electrolytes, glucose, blood urea nitrogen, liver function tests, electrophoresis of plasma proteins and amylase. Serological tests for HIV1, HIV2, syphilis, Epstein Barr virus, cytomegalovirus, hepatitis B surface antigen, herpes simplex virus I were negative. A radiograph of the thorax, and the cerebral CT scan and MRI were normal. Blood and urine cultures were negative. The CSF was clear, under normal pressure, with six lymphocytes, and a normal glucose concentration. Protein was 0.7 g/l and the gamma globulin count 15% with polyclonal banding. Bacteriological studies of the CSF were negative. The EEG showed diffuse slow waves, suggesting an encephalopathy.

During the following days, while there was no fever, the patient became icteric, and developed a hepatosplenomegaly confirmed by echography. There was also a pancytopenia. Peripheral blood films were positive for plasmodium parasites and an indirect immunofluorescent test was positive at the optical interface of falciparum species. Treatment by mefloquine produced a rapid and complete recovery of the hepatosplenomegaly, pancytopenia and icterus. The cerebellar ataxia improved at the same time. The gait returned to normal one month later. Subsequent blood films for plasmodium were negative.

Even though the so called "cerebellar syndrome" is a well known but uncommon clinical presentation of malaria, there are few reported cases in the neurological literature. Lemeriset al found a transient cerebellar syndrome in two of three patients presenting with severe febrile attacks, but they were usually less prominent than the other general and neurological signs of the attack. Nevertheless, the authors insisted on the frequency of the lesions involving the cerebellum, or its connections on neurophysiological examination. Our patient had some common features with the 12 cases from Sri Lanka reported by Senanayake:1 cerebellar ataxia, sometimes associated with nystagmus, occurring in a febrile patient during the febrile attack. We report a febrile attack of falciparum malaria. Gametocytes were present in the blood of these patients and the cerebellar signs subsided one to three months after the anti-malarial treatment was begun. In 12 patients the delayed onset of the neurological deficit, the absence of general signs (splenomegaly was present in one patient) and the presence of gametocytes on repeated blood smears (four out of 12 patients) suggested an immuno-allergic mechanism rather than a direct toxic event due to the plasmodium, even though there was a complete neurological recovery.

Our patient had different clinical features from those of the cases reported from Sri Lanka, but were similar to those reported by Girard et al and Garin et al. Clinical and laboratory findings in our patient make a viral or toxic cause unlikely.