

maquine (total 210 mg). He became asymptomatic in about five days. On the eleventh day following onset of fever, the patient developed tingling sensations in both the lower limbs from the feet up to the knees and both hands. The next day he noticed weakness of both the lower limbs, he could not get up from the squatting position and could not climb stairs. In the night he developed retention of urine. On the following day he was bed-bound and could not move his fingers adequately and could not raise his arm above the bed. He was admitted to the hospital where he developed difficulty in speaking, swallowing and had nasal regurgitation. He was not dyspnoeic at that stage. There was no history of any other illness preceding or following malaria and there was no history of recent immunization.

On examination, his speech volume was low. There were bilateral VII, IX and X nerve palsies. Secretion accumulated in the throat. There was hypotonia in all the muscles of all four limbs. Muscle power was 2/5 in the upper limbs and 0/5 in the lower limbs. The sensory system was normal. Superficial reflexes were normal and all deep tendon jerks were absent. Chest expansion was 5 cm on deep inspiration and single breath count was 26. Chest examination was normal. Peripheral smear examination showed no malarial parasite. Falciparum antigen test was negative and urinary porphobilinogen was absent. CSF study revealed: cells –3/cmm (all lymphocytes), sugar 78mg%, protein 208 mg%. Conduction studies in both median, ulnar, peroneal and posterior tibial nerves revealed gross reduction in motor nerve conduction velocity (MNCV) and CMAP amplitude in all the nerves; distal latency grossly prolonged in all the nerves; F waves absent in all the nerves; temporal dispersion seen in all four limbs; H-reflexes absent in both sides; and SNAP absent in both median, ulnar and sural nerves.

The weakness progressed and the patient developed respiratory difficulty. He was ventilated. He was given IV immunoglobulin 24g/day for five days. The patient started improving 2 days after IVIG was completed and could be weaned off the ventilator after one week. He went home after about one month and could walk unaided.

AIDP / GBS following malaria is rare. It is important to rule out other neurological syndromes that may be unmasked by a febrile episode. A review of 12 cases of GBS (11 previously reported and the present one) revealed that eight patients had preceding falciparum malaria and four had vivax infections. All but three patients (including the present one) had distal symmetric sensory deficits. Paralysis was mild in seven cases (three due to P. Vivax and four due to P. Falciparum) and recovered completely in 2-6 weeks without any specific treatment. Four patients with falciparum malaria developed severe paralysis with respiratory failure, and three patients died. This appears to be the first case report of severe GBS following P. Vivax malaria requiring ventilatory support and IVIG therapy.

The pathogenesis of GBS following malaria infection is not known. This is likely to be immunogenic like that occurring after viral or bacterial infections. Other mechanisms suggested for the development of polyneuropathy following a parasitic infection include parasitic emboli obstructing vasa nervosum, release of neurotoxins, associated metabolic and nutritional disturbance, immune-mediated capillary damage, release of free radicals and tumor necrosis factor.<sup>2</sup>

**A. Chakravarty, B. Ghosh, R. Bhattacharyya,  
S. Sengupta, S. Mukherjee**

*Division of Neurology, Vivekananda Institute of Medical Sciences  
and B. R. Singh Hospital and Research Centre, Calcutta, India.  
E-mail: saschakra@yahoo.com*

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## A rare cause for mononeuritis multiplex

Sir,

Paraneoplastic peripheral neuropathy is a well-described entity.<sup>1,2</sup> Amongst the various manifestations, mononeuritis multiplex is relatively unusual and is usually associated with hematological malignancies.

A 72-year-old man presented with eight months history of progressive symmetric burning paraesthesias over both legs. Later he noticed tingling paraesthesias over the dorsum of the right hand. Four months later he noticed weakness in the left foot in wearing slippers and clearing the ground, which gradually worsened over two months. After one month, he noticed weakness of the right hand, with difficulty in writing, holding objects and buttoning his shirt. Eight months back he developed edema of the feet. He was not a known hypertensive or diabetic. On examination, he had bilateral pedal edema and non-tender hepatomegaly extending 2 cm from the costal margin. There were no hypoesthetic patches. In the right upper limb, he had clawing of the medial two fingers, wasting of first dorsal interosseous muscle and weakness of the adductor pollicis, interossei, lumbricals and opponens digiti minimi. In the left leg he had weakness of the dorsiflexors of the ankle, evertors of subtalar joints and extensors of toes. Both ankle jerks were absent. He had sensory loss over the right little finger and medial aspect of the palm, the right medial forearm, and the lateral aspect of the left leg and dor-



sum of the foot including the first web space. He had left foot drop. The clinical impression was mononeuritis multiplex involving the right ulnar nerve, right medial cutaneous nerve of the forearm and the left common peroneal nerve. Prostatomegaly was evident on ultrasound scan. Nerve conduction study using surface electrodes showed low amplitude Compound muscle action potential (CMAP) of the right ulnar nerve with reduced conduction velocity and absent F waves; CMAPs were absent in bilateral common peroneal and posterior tibial nerves. CMAPs of bilateral median, radial and left ulnar nerves were normal. Bilateral median nerve Sensory nerve action potentials (SNAP) had reduced amplitude while right ulnar and bilateral sural nerve SNAPs were absent. Left ulnar SNAP was normal. Concentric needle EMG in the right abductor digiti minimi and tibialis anterior was suggestive of denervation. Biopsy of the right dorsal cutaneous nerve showed thickened perineurium and axonal breakdown without evidence of vasculitis or Hansen's disease. Doppler scan of legs revealed deep vein thrombosis. Per-rectal fine-needle aspiration of prostate was done and examination of the smear showed adenocarcinoma of the prostate gland. The patient was referred to the oncologist and is on follow-up.

Mononeuritis multiplex is characterized by subacute affection of multiple individual nerves. Common causes for this distinct clinical picture include vasculitis such as Polyarteritis nodosa and Churg-Strauss syndrome, diabetes mellitus and infections such as Hansen's disease, Lyme's disease and HIV infection. Malignancy is an unusual cause for mononeuritis multiplex.<sup>1</sup>

**J. N. Panicker, D. Nagaraja, E. Ratnavalli,  
P. K. Pal**

*Department of Neurology, National Institute of Mental Health and  
Neurosciences, Bangalore - 560029, India.*

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## **Diagnostic dilemma in flaccid paralysis following anti-rabies vaccine**

Sir

Serious neuro-paralytic complications occasionally follow immunization with neural tissue vaccine. The reported inci-

dence of such a complication is 1:220 to 1:12000 vaccines.<sup>1</sup> Patients who receive anti-rabies vaccine (ARV) as post-exposure prophylaxis following a dog bite or exposure to a rabid dog occasionally develop flaccid paralysis. We describe a patient who developed flaccid paralysis following exposure to ARV.

A 24-year-old man was admitted with complaints of tingling sensation in the distal parts of all four limbs for 5 days, difficulty in walking for 4 days, difficulty in lifting his arms above the shoulder for 2 days and inability to close his eyes and whistle, nasal twang of voice and nasal regurgitation for 1 day. His unvaccinated pet dog developed rabies and had died one month ago. Following this the whole family was vaccinated with Semple type ARV. The patient received 11 injections on alternate days till 10 days before onset of symptoms. A day after the last injection he developed dull continuous frontal headache along with corrhiza, which improved completely in 3 days without treatment.

On examination he was found to have bilateral lower motor neuron (LMN) type facial nerve weakness, decreased palatal movement, with central uvula and decreased gag reflex. There was weakness of neck flexors and proximal limb muscles, absent deep tendon reflexes (DTR) with flexor plantar response. Cerebrospinal fluid study revealed no cells, 83mg/dl protein, sugar 97mg/dl (simultaneous blood sugar 120mg/dl) and globulin positive. Magnetic resonance imaging (MRI) of the brain and cervical spine were normal. Motor nerve conduction studies showed decreased conduction with dispersion of compound muscle action potential, low amplitude with prolonged distal latency and decreased nerve conduction velocity. Sensory conduction was normal. Serological examination for HIV was negative. Corneal smear for rabies was negative. After hospitalization, the patient was placed on steroids for two weeks. Limb power returned to normal in 3-4 days. Nasal regurgitation and nasal twang of the voice improved in 4-5 days. Right facial weakness and absent DTR were present at 3 months follow-up. At 6 months follow-up, facial weakness improved almost completely, though reflexes were still absent.

A neuro-paralytic syndrome after Pasteur's post-exposure rabies immunization was first recognized in 1889. Although safer vaccines have been developed, Semple vaccine is still used commonly because of its low cost and easy availability. Semple vaccine is a suspension of phenol or beta- propionolactone killed virus in sheep brain.<sup>2</sup> The incidence of a neurological complication with Semple vaccine is approximately 1 per 220.<sup>2,3</sup> Reported reactions have included encephalomyelitis, transverse myelitis, acute polyradiculoneuropathy and peripheral and cranial neuropathy.<sup>2,4</sup>

Measurement of rabies antibody titer in the serum and cerebrospinal fluid could be of help to differentiate between paralytic rabies and sporadic GB syndrome.<sup>5</sup> In India a serious look into the matter is required as 25,000 people fall victim to

