



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.¹

**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.¹ 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.² The incidence of a neurological complication with Semple vaccine is approximately 1 per 220.^{2,3} Reported reactions have included encephalomyelitis, transverse myelitis, acute polyradiculoneuropathy and peripheral and cranial neuropathy.^{2,4}

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



rabies every year and about half a million people receive anti-rabies vaccine as post-exposure prophylaxis after being bitten by a rabid animal.¹

**A. K. Srivastava, V. Sardana, K. Prasad,
M. Behari**

*Department of Neurology, Neurosciences Center, All India Institute
of Medical Sciences, New Delhi - 110029, India.
E-mail: madhuribehari@hotmail.com*

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Sphenoid wing meningioma presenting as hemi-parkinsonism: Case report

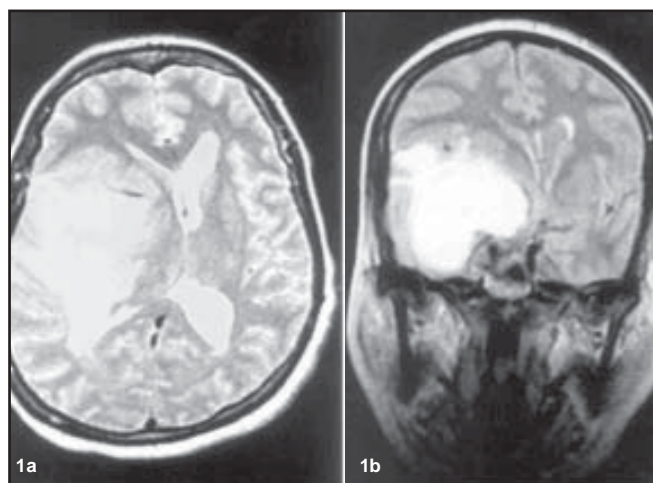
Sir,

Parkinson's disease (PD) has infrequently been described in association with intracranial

tumors.^{1,4} We describe a patient who had a sphenoid wing meningioma and presented with hemi-parkinsonism. Parkinsonism was completely relieved after tumor resection.

A 54-year-old woman presented with a 2-month history of tremor, particularly at rest, in the left upper limb and 2 episodes of generalized seizures. Examination revealed a masked facies, rest tremor in the left hand with a postural tremor of small amplitude, cog-wheel rigidity at the left wrist and elbow and loss of dexterity due to bradykinesia. Fundus showed bilateral papilledema. Gait was normal but slow and left arm swing was diminished. Rest of the neurological examination revealed no abnormality. MRI of the brain showed a large homogenous dural-based mass along the right medial sphenoidal wing with significant perilesional edema and midline shift (Figure 1). She underwent right fronto-temporal craniotomy and near-total excision of the tumor. Postoperatively her tremor and rigidity in the left upper limb disappeared completely. Histologically, the tumor was a meningioma.

The basal ganglia play a major role in the control of posture and movement. Intrinsic basal ganglionic and thalamic lesions



Figures 1a and 1b: Axial (a) T2 weighted and coronal (b) proton density (PD) MR images of the brain showing right medial sphenoid wing dural-based mass. There is mass effect on the right basal ganglia and the thalamus with midline shift to the left

as well as extrinsic compressive lesions have been reported to cause Parkinsonism-like syndrome. Parkinsonism is produced more often by external masses impinging on the basal ganglia than by intrinsic infiltrating lesions. Gliomas, meningiomas, metastatic tumors and even chronic subdural hematoma have been reported to produce symptoms of Parkinsonism.^{2,5}

Direct mechanical pressure and/or torsion of the basal ganglia by a tumor might cause dysfunction of these nuclei. The other proposed mechanism is midbrain compression from upward or downward transtentorial herniation.³ Such herniation may result in the impairment of blood flow to the basal ganglia via the posterior cerebral artery, leading to ischemia of the subthalamic nuclei. In intrinsic tumors, the basal ganglia and thalamus are involved primarily by the tumor, which may lead to internal compression of neurons or pathways.

An intracranial mass as a cause of PD might be missed, especially in elderly patients. Associated symptoms of increased intracranial pressure, focal motor or sensory deficits or seizures (as in our patient) can assist in the diagnosis.

K. C. Shah, V. Rajshekhar

*Department of Neurological Sciences, Christian Medical College
Hospital, Vellore, India.*

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