Letters

but is also encountered in long standing sympathetic denervation of the pupil,⁶ and can be found in either Horner's or Raeder's syndrome.

This case illustrates the need for thorough investigation of Raeder's and Horner's syndromes.

BRUCE G MATHEW

ROBERT M JONES

MALCOLM J CAMPBELL

Department of Neurosurgery & Neurology,

Frenchay Hospital,

Frenchay,

Bristol BS16 1LE, UK

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Thrombosis of cerebral veins following intravenous application of clomipramine

Sir: Thromboembolic complications, mostly within the veins of the legs, following treatment with antidepressants are well known. We report a patient, who developed a lethal thrombosis of the cerebral veins shortly after the beginning of a treatment with intravenous clomipramine.

A 61 year old woman suffering from an agitated depressed state did not respond to oral applications of different antidepressants and electroconvulsive therapy. She had a history of thrombosis of the veins of the right leg and of cardiac failure treated adequately with digitalis. Physical examination revealed a systolic apical murmur and a resonant chest percussion. A chest radiograph showed an enlargement of the left ventricle. Further investigations (ECG, EEG, laboratory reports) were unremarkable. On the 273rd day after admission to the hospital, treatment with clomipramine infusions (50 mg in 500 ml Ringer's solution) was started. The next day

the patient vomited, then had a seizure, afterwards she became comatose and died at the 275th day of treatment.

CT on the day she died showed haemorrhage in the left hemisphere. Autopsy revealed complete occlusion of the superficial cerebral veins, the superior longitudinal sinus, the right lateral sinus, the right sigmoid sinus, partial occlusion of the left sigmoid sinus and the left lateral sinus, and intracerebral haemorrhage of the left hemisphere with oedema.

Thrombosis of cerebral veins are rare compared with other diseases of the cerebral vessels. We found only one other case report concerning thrombosis of the Galenic system veins after the start of intramuscular injections with amitriptylinepamoate.¹ Besides the slowdown of the blood flow by prolonged bed rest we assume that changes of the blood coagulation and of the vessel endothelium played a part in the pathogenesis of thrombosis in our patient.² This case draws attention to the importance of antithrombotic prophylaxes during treatment with infusions of antidepressants, such as low-dose heparin (5,000 units three times a day), bandaging of the legs, and exercise.³

GISBERT EIKMEIER

RALF KUHLMANN

MARKUS GASTPAR

Department of Psychiatry,

University Hospital Essen,

Hufelandstr. 55

4300 Essen 1

Federal Republik of Germany

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Myotonic dystrophy with unilateral bulbar involvement

Sir: Myotonic dystrophy is a hereditary autosomal dominant multisystem disease

characteristically affecting skeletal muscle, but also involving the heart, gastrointestinal smooth muscle, respiratory system, skull, brain, eyes, and skin.¹

Among the bulbar signs, weakness of the pharyngeal muscles is quite common and not as a late manifestation of the disease.² On the other hand, laryngeal abnormalities have been reported late in the course of the disease but are not common, and are mild and bilateral.³⁻⁵

We present here an unusual case of myotonic dystrophy in which the laryngeal symptoms were the presenting signs combined with unilateral atrophy of the neck and shoulder. A 29 year old unmarried man was admitted to the Ear-Nose-Throat department, because of progressive dysphonia in the past year and dysphagia for 4 months. On examination a unilateral left vocal cord paralysis together with severe weakness of the left pharyngeal muscles were observed. The rest of the examination was normal. Thyroid scan, plain radiographs of the foramen jugularis, and computed tomography of the brain stem and the base of the skull were negative.

He was transferred to the neurological department for further investigation. On examination myopathic facial appearance together with unilateral atrophy of the left sternocleidomastoid and trapezius muscles were observed. Myotonic reaction of both hands, decreased deep tendon reflexes in the arms without any decrement of strength or atrophy of the arms and hands were found. Bilateral posterior polar mild cataract, small testicles and first degree A-V block in an ECG were the additional findings. Chest radiographs were normal and lung function tests showed mild proximal airway obstruction. A barium swallow radiograph showed bilateral pooling of barium in the valleculae, more pronounced on the left side, with normal oesophageal peristalsis. Electromyographic study showed the typical myotonic discharges sounding like a "dive bomber" with normal conduction velocities in peripheral nerves of the limbs.

The diagnosis of jugular foramen syndrome was eliminated by normal CT of the base of the skull, and local disease in the larynx was excluded by 2 years of surveillance without any new finding in an ENT examination.

We have checked nine more siblings and found another brother 40 years old with cardiac pacemaker for 7 years, with advanced myotonic dystrophy. Five more siblings had partial syndrome, with cataract and diabetes mellitus.

The laryngeal involvement in myotonic dystrophy is well accepted even though it is

not common.²⁻⁶ Early in the course of the disease it is rare to find any laryngeal abnormality. The voice is sometimes flat, expressionless and monotonous with tendency to improve as talking continues. On examination at this stage of the disease no abnormality can be detected.^{2,3}

Our case presented with unilateral vocal cord paralysis. To the best of our knowledge such a clinical picture has never been reported before. The pronounced asymmetry with left pharyngeal weakness and unilateral atrophy of the left neck and shoulder, is also unusual in a disease where the symmetry of appearance is one of the typical features. Early diagnosis of myotonic dystrophy is important in the light of genetic counselling.

N GILADI
I BAZAK
D HAREL

Department of Neurology,
Lady Davis Carmel Hospital,
7 Michal Street,
34362 Haifa,
Israel.

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Sixth cranial nerve palsy complicating psittacosis

Sir: Neurological complications in psittacosis are uncommon. We report the occurrence of an acute VIth cranial nerve palsy complicating psittacosis.

A previously healthy 49 year old English lady presented with a 2 week history of malaise, sweats, fever, chills and severe, intermittent headache. Three days preceding

admission she developed a non-productive cough. She did not own any pets. On examination she was acutely ill, febrile at 39.5 °C, with a blood pressure of 130/90 mm Hg. There were crackles in the left mid-zone posteriorly. She was fully conscious, orientated, with no neurological signs. Eye examination was normal. Initial investigations showed a haemoglobin of 11.8 g/dl, increased white cell count at $16.1 \times 10^9/l$ (neutrophils 89%, lymphocytes 8%, monocytes 3%), and an elevated plasma viscosity at 2.33 cp. The urea and electrolyte levels, liver function tests and serum electrophoresis were within normal limits. A chest radiograph showed patchy left mid-zone shadowing. A diagnosis of an atypical pneumonia was made and the patient commenced on erythromycin 750 mgm 6 hourly by mouth. Two days after admission, when she felt significantly better, the patient developed acute diplopia. Examination revealed an isolated, complete, left VIth nerve palsy, with no papilloedema or other neurological signs. CT of the brain, skull radiograph and CSF findings were normal. Subsequent results showed a positive immunofluorescence *Chlamydia psittaci* titre at 1/256 and a CFT titre at 1/80. Upon close questioning, a history of incidental contact with two parrots and a sick budgerigar was obtained. Serological tests for mycoplasma, legionella and coxiella were negative. Erythromycin was continued for 10 days.

The patient became extremely distressed by the diplopia and was unwilling to use alleviating measures with an eye patch or special spectacles. With no precedence to guide us, the prognosis was guarded. Recovery of the VIth nerve palsy commenced 8 days after its onset and was complete by the fifth week. The patient remains well 3 months later.

Cranial nerve involvement complicating infection with *Chlamydia psittaci* is unusual.¹ IInd,² supranuclear VIIth,³ and XIIth⁴ cranial nerve palsies have been reported in severe encephalitic cases of psittacosis. Unlike these, there were no signs of encephalitis or raised intracranial pressure in our patient. Rare as it may be, the prognosis appears excellent.

A ZUMLA
G LIPSCOMB
D LEWIS
Infectious Diseases Unit,
Rush Green Hospital,
Romford,
Essex RM7 0YA
UK.

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Motor axon loop as a source of error in the measurement of motor nerve conduction velocity

Sir: In 1984 Roth and Egloff-Baer described the motor axon loop, a new kind of response of the motor unit to stimulation of the peripheral motor nerve.¹ We would like to report a case in which a motor axon loop may have caused an error in the motor nerve conduction velocity measurement.

A 72 year old man developed weakness and atrophy in his right hand muscles in January, 1985. The weakness and atrophy slowly progressed, and in September, 1986 when he visited our outpatient clinic, he had severe weakness and atrophy in his right upper extremity muscles and moderate weakness and atrophy in his left hand muscles. Sensation was intact. Electromyography revealed neurogenic abnormalities in the facial, sternocleidomastoid, tongue, and upper and lower limb muscles. A diagnosis of amyotrophic lateral sclerosis was made. A motor nerve conduction study was carried out on the right median nerve. Recording was made with surface electrodes placed on the belly of the thenar muscle (cathode) and the base of the thumb (anode). Electrical pulses of 0.2 ms in duration were delivered. Only one motor unit potential (MUP) was evoked in the thenar muscle, in an "all-or-none" manner. When the median nerve was stimulated at the elbow, the latency of the MUP shortened abruptly by increasing the intensity of the stimulus to a critical level (figure inset). Serial stimulation from the wrist to the upper arm in 3 cm increments revealed that this shortening phenomenon occurred only between 16.5 and 25.5 cm from the wrist, the difference between the latencies of the late and brief responses being constant. When the stimulus was delivered outside this segment, the latency of the MUP did not show any change by increasing the stimulus intensity. No F-waves were observed. When the latencies of the MUP were plotted