studies of CSF in Sydenham's chorea, the use of qualitative as well as newer quantitative IgG methods would be desirable. Since the incidence of Sydenham's chorea appears to be declining markedly, in those part of the world where the appropriate laboratory facilities are readily available, this may be easier said than done.

Finally, it is noteworthy that circulating immune complexes were not detected in this patient's serum. Citing unpublished observations, Husby et al. recorded the presence of immune complexes in "virtually all acute sera from children with chorea" and questioned the relevance of their occurrence to the means whereby serum antineuronal antibodies might gain access to the brain. Alternative mechanisms of blood brain barrier disruption in Sydenham's chorea have been suggested, with which the findings in the present case would not be at variance.

I wish to thank Dr DJL Van Rooy, Medical Superintendent, Kalafong Hospital for permission to publish details of this patient.

RF GLEDHILL
Neurology Department, Kalafong Hospital, Pretoria 0001, South Africa

References

Brain stem encephalitis in ornithosis

Sir: Psittacosis is caused by Chlamydia psittaci, an obligate intracellular procarciocye parasite which infects several species of birds and mammals. Humans may also be infected. The disease affects many organs but pulmonary symptoms predominate in most human cases. It may occur as a mild influenca-like illness, or take a severe to fulminating course leading to a fatal outcome especially when diagnosed late, or in the elderly. The symptoms are fever, headache, anorexia, chest pain, dry cough, haeomptysis and pneumonia. Some distinguishing features are relative bradycardia or pulse-temperature dissociation as seen in brucellosis or typhoid, a normal white blood cell count and, occasionally, splenomegaly.

Neurological manifestations are much less common than pulmonary symptoms but have been reported. We present a case of unusual neurological involvement in psittacosis, and review the literature.

A 53-year-old Saudi Arabian male developed headache, fever and chills in April 1983 with generalised aches, malaise, anorexia, chest pain and a dry cough. On admission to Riyadh Military Hospital his temperature was 39.5°C, and pulse rate was 120/min. Crepitations were heard in the right lung base; white blood cell count (WBC) was 11500/mm³; erythrocyte sedimentation rate (ESR) was 100 mm/h, SGOT 213 U/L, LDH 911 U/L, alkaline phosphatase 215 U/L. A radiograph of the chest showed a consolidation at the right lower lobe. Chlamydia and mycoplasma complement fixation (CFT) titres were negative on the second and tenth days of hospitalisation. Intravenous penicillin was given for 72 hours. The patient also failed to respond to cefotaxin, gentamicin and erythromycin, and his temperature stayed at 38.5°C. He developed urinary retention on day 9 of hospitalisation. When it became known that he had acquired a parrot one month earlier he was treated with tetracycline and soon became afebrile. Bilateral facial palsy and severe dysaesthesia and weakness in the legs developed during the following two days. He lost 8 kg in weight in the first two weeks of hospitalisation.

He was transferred to King Faisal Specialist Hospital on day 18. On admission he was afebrile with tachycardia, pulse rate 118/min; he had dysarthric speech, bilateral facial palsy, upbeating nystagmus, bilateral brisk reflexes and extensor plantar responses. He was not able to stand unaided. WBC was 9200/mm³, ESR, 57 mm/h, computed tomography (CT) scan of the brain was normal and neither hypodense nor enhancing lesions were seen on thin cuts through the pons. CSF showing elevated protein 200 mg/dl, glucose 86 mg/dl, serum glucose 124 mg/dl and lymphocytes 2/mm³. Tetracycline was continued, with the addition of high dose thiamine and dexamethasone. Serum CFT titre for chlamydia was 1:1024. Early samples of serum were retested for chlamydia CFT using a different make of commercial antigen and gave titres of 1:128 and 1:512. Monospot test was negative; CSF psittacosis CFT titre was 1:16; brucella slide test, viral cultures and blood cultures were negative. Electroencephalogram showed diffuse encephalopathic changes. Brain stem auditory evoked potentials showed a slight prolongation I-V interpeak latency (4.96 ms) with wave V less well defined and of smaller amplitude on the left. Visual evoked potentials were normal. Urodynamic studies showed spincter-detruzer dysynergia. Electrocardiogram and echocardiogram were normal and the tachycardia was attributed to autonomic dysfunction.

The patient felt progressively better; he remained afebrile and gained weight. His gait improved gradually. Urinary retention resolved after treatment with phenoxybenzamine and baclofen with intermittent catheterisation for a few days. Serum psit-
Psittacosis CFT titre remained high. Liver enzymes became normal; WBC remained normal; brain stem evoked potential interpeak latency I-V decreased slightly to 4-8 ms. The patient was discharged after 25 days in King Faisal Specialist Hospital, able to walk independently but with difficulty.

On follow up the patient gradually returned to normal; mild subjective dysaesthesia in the feet persisted at one year. Psittacosis CFT titres on eight persons at risk in the household were negative. The parrot was sacrificed and disposed of without necropsy.

The criteria for the diagnosis of psittacosis are met only by the history of exposure to a parrot, onset with pulmonary symptoms, a compatible chest radiograph, normal WBC, high serum CFT titres, and a dramatic response to the appropriate antibiotic after failure of potent but non-specific antibiotics. Unfortunately confirmation by necropsy of the bird was denied.

It is known that psittacosis affects many organs including liver, heart, kidneys, skeletal muscles and the CNS. The neurological signs in our patient were those of brain stem involvement, the nature of which remains speculative in the absence of pathological confirmation. One may argue that this is a form of central pontine myelinolysis caused by nutritional derangement or fluctuating electrolytes during the early days of the illness. But the CT scan did not show the unenhancing radiolucency usually seen in this condition; the near complete recovery argues against pontine myelinolysis which is usually fatal or has significant neurological sequelae. The time was too short for a previously healthy patient to have been depleted of thiamine, and no significant hypotraemia or rapid electrolyte correction had occurred during his illness.

The psittacosis surveillance reported by the United States Center for disease control in the 1970s showed that CNS manifestations occurred in 5–9% of cases. Symptoms are variable. Encephalitis, polyradiculitis and cerebellar symptoms have all been reported in psittacosis, and in other infections such as Mycoplasma pneumoniae, legionellosis and mononucleosis. Neuropathological study of cases of "psittacosis encephalopathy" showed mainly vascular changes including congestion, hyaline thrombi and perivascular monocyteic infiltrate with small foci of necrosis or demyelination. Invasion of the CNS as evidenced by the presence of intracytoplasmic inclusions (LCL bodies) had been found only rarely in cases of meningitis. The term brain stem encephalitis was first used by Bickerstaffe to describe neurological manifestations predominately referable to the brain stem and occurring in the course of an acute or subacute infection. The term had been suggested to include a variety of symptoms, such as those of Fisher's syndrome. The non-specificity of the initial infection suggests that the underlying pathophysiology involves the immune system or causes changes in the blood vessels which lead to the symptoms of brain stem involvement. The absence of an immunological event is not inconsistent with the diagnosis as concluded by Waxman et al who reported subacute brain stem encephalitis leading to respiratory death without an obvious antecedent infection, immunological event or neoplastic disease. In our case, intense activation of the immune system early in the course of the infection may have led to a focal vasculitis involving the brain stem. Although vascular changes have been reported in psittacosis, the cause of preferential involvement of the stem vasculature requires further study.

References


Relapsing Fisher's syndrome

Sir: Fisher's syndrome, characterised by the triad of ophthalmpoplegia, areflexia, and ataxia, is considered by many neurologists to be a variant of the Guillain-Barré syndrome. Approximately 3% of patients with Guillain-Barré syndrome will have one or more relapses, but the relapse rate for Fisher's syndrome is unknown. We wish to report a patient with a relapsing illness which fits the classical description of Fisher's syndrome.

A 58-year-old woman was first evaluated in November 1977 for the complaints of dizziness and unsteadiness. She had a 'flu-like illness four weeks previously, but was otherwise in good health. Her past history was unremarkable for prior neurological illness. The unsteadiness increased over the ensuing two days, and parasthesias of the feet and horizontal diplopia developed. General examination, including vital signs, was normal. On neurological examination she was anxious. The visual fields were normal. The pupils were 5–6 mm and reacted minimally to light, although they did not constrict during attempted convergence. Horizontal gaze and upgaze was absent, and on attempted downgaze the eyes would move only a few degrees. There was no
Brain stem encephalitis in ornithosis.

M Z Al-Kawi and M M Madkour

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