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Selective increase in cerebrospinal fluid immunoglobulin G in a patient with Sydenham's chorea

Sir: Evidence exists to suggest that immunological mechanisms may be involved in the pathogenesis of Sydenham's chorea.[1–6] This evidence is led by the demonstration of tissue specific immunoglobulin G (IgG) antibodies to neurons of normal human caudate and subthalamic nuclei in the serum of 14 of 30 children with Sydenham's chorea.[2] Moreover, antibody titres correlated with disease activity and the antibody was absorbed out with membranes of group A streptococci, infection with which is a recognised antecedent to the development of Sydenham's chorea.[7] The case for antibody mediated responses playing a primary role in Sydenham's chorea would be strengthened were there evidence of excess IgG present in the cerebrospinal fluid (CSF) of these patients.[8] Such a finding is not recorded in either a contemporary authoritative text[9] or review article[10] on the CSF, or in recent accounts in which the CSF of patients with Sydenham's chorea was studied (for example refs 11, 12). These observations prompted the following case report.

A 15-year-old black female was admitted to Kalafong Hospital on 12 August, 1985 with a 6-day history of abnormal movements. Her past medical history was remarkable for recurrent sore throats, the most recent of which occurred approximately 3 weeks before admission. There was no known family history of chorea and she denied drug ingestion or treatment with hormonal contraceptives. Examination revealed emotional lability, distractibility, restlessness, occasional snorts and grunts, mild dysarthria and generalised involuntary movements typical of Sydenham's chorea.[13] The limb muscles were hypotonic and the patellar reflexes were "hung up". Voluntary movements were exaggerated and she was unable to walk without aid. The temperature was 38°C and pulse rate 110 beats per min. In the opinion of a cardiologist, the heart was clinically normal.

Results of pertinent investigations were as follows: Haemoglobin 12·7 g/dl, erythrocyte sedimentation rate 35 mm/first h, total white blood cells $7{\cdot}7 \times 10^9$/l, serum IgG 29·6 g/l (normal 8·00–18·00), antistreptolysin 0 titre 400 Todds units (< 200), streptokinase haemagglutination antibody 1: 2560 (< 1: 1280) and rapid plasma reagin (RPR) 1: 128. The *Treponema pallidum* haemagglutination antibody (TPHA) and fluorescent treponemal antibody absorbtion (FTA-ABS) tests were positive in the blood, the IgG FTA-ABS test reactivity being 3 + (max 4+) and the IGM 1+. (These results prompted appropriate therapy with parenteral penicillin). An echocardiogram revealed evidence of stenosis and incompetence of the mitral valve. Normal or negative results were obtained for the following: Throat swab culture, C-reactive protein, Heller agglutination test, fluorescent antinuclear antibody test, total haemolytic complement, serum C_3, plasma C_4, circulating immune complexes, thyroid function tests, Kayser Fleischer rings, serum copper and ceruloplasmin, somatosensory evoked potentials (median nerves), electrocardiography and CSF RPR, TPHA and FTA-ABS tests.

An electroencephalogram on 26 August showed an excess of 4–7 Hz and intermittent 2–3 Hz activity in a diffuse, bilateral distribution. Contrast enhanced computed tomography of the brain (10 September) was normal. Lumbar CSF on 22 August was acellular with a normal glucose concentration and a total protein of 0·46 g/l (0·15–0·45). The IgG was 0·146 g/l (0·002–0·028), 31·7 per cent (5–12[9]) of the total protein, and CSF/serum IgG ratio was $4{\cdot}9 \times 10^{-3}$ ($1{\cdot}0–3{\cdot}9 \times 10^{-3}$ [14]). Repeat CSF examination on 11 September was unremarkable, the IgG (0·018 g/l) being 4·6% of the total protein (0·39 g/l) and the CSF/serum IgG ratio $0{\cdot}98 \times 10^{-3}$ (serum IgG 18·3 g/l). The patient was discharged on 25 September free of psychologic and neurologic deficits and taking no medication other than oral penicillin.

Of the numerous nervous system diseases associated with a selective increase in CSF IgG,[9 10 15] neurosyphilis has special relevance in this patient since blood test results indicated a recent response to *Treponema pallidum* infection.[16] Results of the RPR, TPHA and FTA-ABS test in the CSF were negative, however, which, together with the absence of cells and normal total protein concentration, to all intents and purposes excludes[16] syphilitic involvement of the nervous system.

Using the IgG index formula, Tourtellotte *et al*[17] reported excess CSF IgG in 9% of asymptomatic normal individuals (n = 56), raising the possibility that the findings in the present case represent a fortuitous example of a normal phenomenon rather than the occurrence of a pathological immunoglobulin fraction. Tourtellotte *et al*[17] did not state whether an elevated IgG index was a persistent finding in their control subjects, precluding any relevant deductions from the presence of a normal IgG concentration in the later sample of CSF in this patient. At first glance, this normalisation of the CSF over a period of 3 weeks could cast doubts on the veracity of the initial result. However, not only were both readings double checked (Dr L Van Niekerk, personal communication), but also the second lumbar puncture was performed when the patient's chorea had virtually subsided, at which time Husby *et al*[2] found that serum antineuronal antibodies had disappeared in cases studied with serial samples. Notwithstanding these circumstances, the observations of Tourtellotte *et al*,[17] together with the constraints pertaining to the study of a single case, make it imprudent to draw inferences on the possible immunopathogenic basis for Sydenham's chorea from the CSF findings reported herein. With respect to further

studies of CSF in Sydenham's chorea, the use of qualitative as well as newer quantitative IgG methods[2 15 17 18] would be desirable. Since the incidence of Sydenham's chorea appears to be declining markedly[19] 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*[4] 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[1 3] with which the findings in the present case would not be at variance.

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Brain stem encephalitis in ornithosis

Sir: Psittacosis is caused by *Chlamydia psittaci*, an obligate intracellular procariocyte 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 influenze-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, haemoptysis 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/mm^3; 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 cefotoxin, 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/mm^3, 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/mm^3. 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 sphincter-detruser dyssynergia. 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-