Table *Prevalence (n (%)) of middle ear abnormalities in reading disabled (n=49) and non-reading disabled children (n=913)*

Otological grouping	*Reading disabled*	*Non-reading disabled*
Persistent bilateral otitis media with effusion with hearing loss greater than 25 dB	5 (10·2)	48 (5·3)
Persistent bilateral otitis media without appreciable hearing loss	2 (4·1)	43 (4·7)
Persistent unilateral otitis media with effusion	2 (4·1)	24 (2·6)
Transient unilateral or bilateral otitis media with effusion	8 (16·3)	110 (12·0)
No evidence of otitis media, but scarred tympanic membrane	5 (10·2)	133 (14·6)
C type tympanogram on at least one occasion, but no evidence of otitis media or B type tympanogram	20 (40·8)	429 (47·0)
Bilateral A type tympanograms at every assessment	7 (14·3)	126 (13·8)
Total	49 (100)	913 (100)

disease is unlikely to be an important determinant of specific reading disability. Previous reports of positive findings from cross-sectional studies may be attributable to referral biases, fallible retrospective report data, or unreliable single occasion assessment of reading and middle ear state. Another important point is that in previous studies reporting positive findings the reading disabled children have been largely untreated. It is noteworthy that the only negative finding reported that all reading disabled children studied had been examined and treated by an otolaryngologist.[3] Similarly, all children in the present study identified as having otitis media were examined and treated by an otolaryngologist. Of the 98 children with persistent bilateral otitis media, 83 had tympanostomy tubes inserted on at least one occasion.

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Correspondence to Dr D L Share, The Dunedin Multidisciplinary Health and Development Research Unit, Department of Paediatrics and Child Health, University of Otago Medical School, PO Box 913, Dunedin, New Zealand.

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Cerebral atrophy and nephropathic cystinosis

P COCHAT, R DRACHMAN, M-F GAGNADOUX, D PARIENTE, AND M BROYER

Hôpital Debrousse, Lyon, and Hôpital des Enfants Malades, Paris, France

SUMMARY The management of end stage renal failure in cystinotic children is correlated with a longer survival, sometimes complicated with neurological abnormalities. Cranial computed tomography was performed in 10 patients and showed a significant atrophy; the pathogenesis of this damage remains unclear.

The full description of cystinosis is yet to be completed as until the last 15 years all those patients with cystinosis died early from end stage renal failure; central nervous system involvement was not often reported as a late complication.[1 2] The patients' survival over the second or third decade may be complicated with neurological manifestations, and it has been shown that some patients with nephropathic cystinosis have abnormalities on cranial computed tomography.[3]

Patients and methods

Several types of neurological symptoms were noted in 10 cystinotic patients (group 1; mean (SD) age 14·2 (4·0) years), such as repeated seizures, tremor,

mental retardation, and pseudobulbar or pyramidal syndrome. Ten control patients with another primary renal disease (group 2; mean (SD) age 11·8 (3·7) years) had no cerebral abnormalities.

To decide if these features were related specifically to cystinosis or to severe chronic uraemia and/or its treatment, we studied the cranial computed tomograms of the patients in both groups. Both groups included dialysed or transplanted children without significant difference in length of end stage renal failure management (4·9 (SD 3·9) years in group 1 *v* 4·4 (2·3) years in group 2) or in parathormone and aluminum plasma concentrations. Treatment with steroids is known to induce an appearance on computed tomography of a brain that looks shrunken; such treatment had been used for some subjects in both groups (for transplantation, nephrotic syndrome, etc) without significant difference in cerebral computed tomogram data. Most of the cystinotic patients had mild hypothyroidism controlled by hormonal treatment.

Cerebral atrophy was evaluated by quantitative indexes of the ventricle:brain ratios on two distinct computed tomography sections: A ratio for the largest representation of the lateral ventricles, and B ratio for the largest representation of frontal horns, including Monro's foramen.[4]

Results

Figure 1 shows that the mean (SD) A ratio is 0·27 (0·09) in group 1 and 0·14 (0·04) in group 2 ($p<0·01$); mean (SD) B ratio is 0·15 (0·04) in group 1 and 0·09 (0·03) in group 2 ($p<0·01$). The brain atrophy is confirmed in cystinotic patients by the enlargement of cortical sulci, as only two out of 10 in group 1 versus eight out of 10 in group 2 had no significant measured widening (Figures 2 and 3).

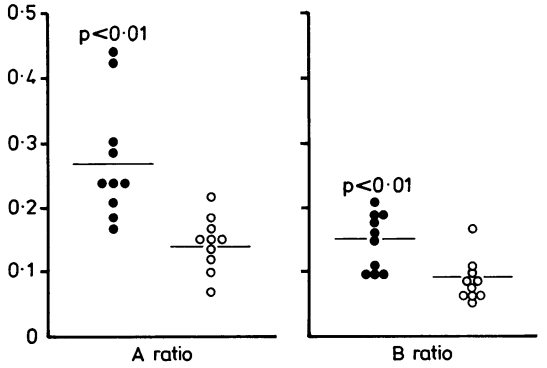

Fig. 1 *Comparison of the ventricle:brain ratios in group 1 (10 cystinotic patients) (●) and group 2 (10 patients with end stage renal failure (○).*

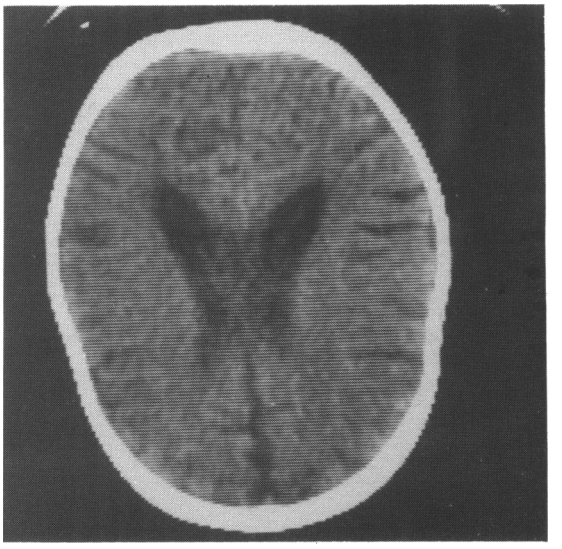

Fig. 2 *Cranial computed tomogram of a cystinotic patient, showing cerebral atrophy.*

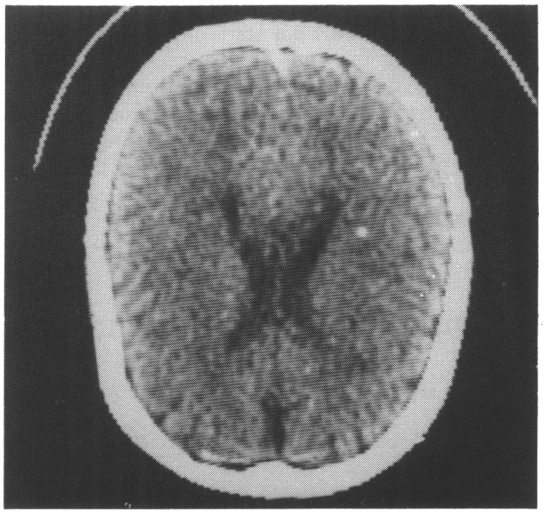

Fig. 3 *Cranial computed tomogram of a control patient without significant cerebral abnormality.*

Discussion

The pathogenesis of this progressive cerebral damage remains unclear. An inbalance in cerebrospinal fluid is a possibility, as cystine crystal deposits have been found in choroidal plexi and meninges at

autopsy;[3 5] cerebral cell atrophy in relation to the metabolic disorder is also a possibility.

We conclude that children with nephropathic cystinosis have cortical and subcortical atrophy after 10 to 20 years of age, and this feature suggests specific central nervous system complications, which are not yet fully understood.

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Correspondence to Dr P Cochat, Hôpital Debrousse, 29 rue Soeur Bouvier, 69005 Lyon, France.

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Myocarditis after triple immunisation

S G AMSEL, A HANUKOGLU, D FRIED, AND M WOLYVOVICS

Department of Paediatrics and Heart Institute, Edith Wolfson Hospital, Holon, and Sackler School of Medicine, Tel-Aviv University, Israel

SUMMARY We describe a 3 month old infant who developed myocarditis several hours after diphtheria, tetanus, and pertussis vaccination. The time of occurrence of symptoms, the clinical course, and the negative virological studies suggest a possible cardiogenic adverse reaction to the vaccine.

Severe systemic adverse reactions after diphtheria, tetanus, and pertussis vaccination are uncommon and include extreme irritability, collapse or shock like episodes, convulsions, and encephalopathy.[1 2] Cardiac side effects associated with this vaccination have been reported rarely and are most probably related to the pertussis component of the vaccine.[3 4] To the best of our knowledge, myocarditis after the vaccination has not been reported. We describe a case of acute myocarditis that developed a few hours after diphtheria, tetanus, and pertussis vaccination.

Case report

A 3 month old infant was admitted to our hospital 24 hours after receiving his second diphtheria, tetanus, and pertussis and oral polio vaccination because of severe respiratory distress and cyanosis. The infant was delivered at term, weighing 3250 g, and had had an uneventful neonatal period. The first diphtheria, tetanus, and pertussis and trivalent oral polio virus immunisation was administered at 6 weeks without any adverse reaction. The parents stated that the child had been normal and playful until 12 hours after the second administration of the vaccines, when irritability and mild respiratory difficulties appeared.

On admission, physical examination revealed a 3 month old, well nourished, well developed, acutely ill infant. The temperature was 38·5°C, pulse rate 200 beats/minute, and respiration rate 150/minute. His colour was ashen, and a mild oedema of the extremities was evident. On auscultation the heart sounds were of poor quality, occasionally a gallop rhythm was heard, and no murmurs were audible. The lungs were clear, and the liver edge was palpated 5 cm below the right costal margin. All the peripheral pulses were weak. The capillary blood gases showed metabolic acidosis with pH 6·7, carbon dioxide tension 28 mm Hg, oxygen tension 67 mm Hg, base excess −28 mM/l, and bicarbonate 5 mM/l. White blood count was $20{\cdot}6\times10^9$/l, with 70 per cent lymphocytes; haemoglobin 93 g/l; serum urea nitrogen and creatinine 14 mmol/l and 90 mmol/l, respectively. The sodium, potassium, and calcium concentrations were normal.

The serum creatine phosphokinase activity rose abruptly to 348 IU/l (normal is 5–80 IU/l) on the second day after admission, reflecting myocardial damage. An electrocardiogram disclosed low voltage QRS complexes on limb leads and non-specific ST changes and T wave flattening on precordial leads. *X* ray film showed generalised cardiac en-