intramuscularly. Nephrogenic diabetes insipidus was diagnosed. Dyazide was withdrawn and potassium supplements given. At review one month later the water-deprivation test gave normal results. After 12 hours of fluid deprivation the urinary osmolality rose to 800 mmol/kg with a plasma osmolality of 280 mmol/kg. The patient remained well with normal plasma urea and electrolyte concentrations and normal erythrocyte sedimentation rate.

Comment

This patient with previously normal renal function developed nephrogenic diabetes insipidus after two months of treatment with Dyazide. We are unaware of any other report of this condition after treatment with Dyazide or its constituents triamterene and hydrochlorothiazide. Probably the impaired urinary concentrating ability resulted from the hypokalaemia[1] induced by Dyazide. Stimulation of the thirst centre owing to potassium depletion[1 2] may have been an additional factor in this patient's symptoms. Though he was also receiving warfarin and digoxin we do not know of any documented association between these drugs and renal impairment in water reabsorption.[3] There was no evidence of any other cause of nephrogenic diabetes insipidus, such as hypercalcaemia, renal structural lesions, or myeloma.[2] Withdrawal of Dyazide and correction of the potassium and sodium depletion abolished the abnormality in renal tubular function. Though thiazide diuretics inhibit potassium reabsorption in the proximal part of the distal renal tubule, appreciable urinary potassium loss seldom occurs during Dyazide treatment because of the potassium-retaining effect of triamterene in the distal part of the distal renal tubule.[4] In this case, however, hypokalaemia and features of nephrogenic diabetes insipidus developed only two months after starting treatment with this combination diuretic.

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Status epilepticus caused by solvent abuse

Glue sniffing may present in various ways,[1 2] and the condition may be fatal.[3 4] We describe an adolescent with severe status epilepticus due to solvent abuse.

Case report

A 15-year-old previously healthy boy presented after a grand mal seizure in the school gymnasium followed by severe uncontrolled status epilepticus. He had had two febrile convulsions in early childhood. On admission he was in coma but was localising painful stimuli; there were no focal signs, and general examination showed no abnormality. Results of routine biochemical, haematological, and bacteriological investigations were normal, as were chest and skull radiographs and cerebrospinal fluid. Computed tomography showed small ventricles and no focal abnormality. On the third day the frontal lobe was sampled for biopsy. Histologically there was no evidence of encephalitis. A few neurones showed features of recent necrosis, but whether this was a focal infarct or part of a diffuse disorder could not be defined. Viral serological and immunofluorescent antibody studies excluded herpes simplex infection. An anonymous telephone call then suggested that the boy might have been sniffing glue. An aliquot of the brain biopsy specimen was therefore sent for analysis for solvents by gas chromatography[5] and showed a toluene concentration of 14·5 μg/g tissue.

Despite therapeutic serum concentrations of various anticonvulsant combinations the patient continued to have frequent seizures. He was therefore also treated with intermittent positive-pressure ventilation, muscle relaxants, and sedation and required a tracheostomy. Continuous electroencephalographic monitoring indicated seizure activity at around six/hour. After two weeks he began to improve. The frequency of seizures decreased without any major change in treatment. Mechanical ventilation was discontinued. He began to obey commands, and after removal of the tracheostomy tube he began speaking at 10 weeks, at which time computed tomography showed dilatation of the lateral and third ventricles and several areas of low density resembling multiple infarcts. He began to walk 15 weeks after onset and was discharged home after five months. Follow-up two years later showed no focal neurological deficit, and psychometry indicated no definite impairment. He continued to have one or two seizures a month, however, and exhibited major behavioural problems.

Comment

This case presented major difficulties in diagnosis, and many aetiological factors were investigated. Though hypoxia may have influenced the neurological disturbance, solvent intoxication probably initiated the illness and contributed to its severity. A latent predisposition to convulsions may also have been significant in view of the patient's reaction to fever in childhood. Blood concentrations of toluene usually encountered in cases of solvent abuse range from 0·4 to 1·0 μg/g in samples taken within 24 hours after abuse and from 1·0 to 10·0 μg/g in samples taken when the abuser was obviously intoxicated. Two subjects who hanged themselves when under the influence of toluene had blood concentrations of 52 and 39 μg/g.[5] The concentration found in the brain tissue was above that found in sniffers who are acutely intoxicated, presumably because the solvent was concentrated in lipids. The true concentration was probably higher, as there would be loss of solvent owing to the small size of the sample, transport in a container of relatively large volume (32 ml), and the delay of seven days between biopsy and analysis.

Without laboratory confirmation it would have been difficult to establish that glue sniffing had been an important aetiological factor in this boy's life-threatening illness. The diagnosis of solvent intoxication should be considered in cases of epilepsy or encephalopathy of undetermined aetiology arising for the first time in the adolescent age group.

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Correction

Herpes-zoster myelitis treated successfully with vidarabine

An error occurred in this paper by Dr R N Corston and others (12 September, p 698). The dose of vidarabine given in line 8 of the third paragraph of the case report should have read "15 mg/kg/day," not "5 m/kg/day."