viscometer. The disposable system consists of a standard 2 ml plastic syringe (BD Plastipak), internal diameter 0.8 cm, and a 19-G serum needle. The Ostwald apparatus was used in the standard fashion, with a water bath to maintain constant temperature. The barrel of the syringe in the disposable system was filled by drawing up undiluted serum through the needle, starting with the plunger at the 1 ml mark. Taken in this fashion, the serum level is just over the 2 ml mark at the moment that the plunger is withdrawn completely from the barrel of the syringe. With the syringe hand held or clamped in a vertical position, the run-through time is started when the bottom of the meniscus passes the 2 ml mark of the barrel and the timing stopped when the meniscus reaches the zero mark on the barrel. Care is taken to ensure that the vertical position of the barrel is maintained, though in practice there is not a significant difference between the measurements obtained by free hand holding of the barrel and those obtained by clamping in the rigid apparatus. Before comparing the syringe needle system against the Ostwald apparatus it was confirmed that different combinations of needles and syringes on single serum samples produced similar results. Measurements were made at three standard temperatures—namely, 37°C (incubation room), 20.5°C (ambient room temperature), and 4°C (cold room). In some instances there was insufficient serum to fill the Ostwald apparatus so that there were more measurements taken with the disposable system at all temperatures. The relative viscosity (RV) of a sample at any given temperature was derived by dividing the run-through time for the serum against that of distilled water at the same temperature.

There was a significant increment in the relative viscosity of normal serum as temperature fell and the scatter of individual values for any given temperature was similar using the two methods of measuring viscosity (see figure).

Relative viscosity of normal serum using Ostwald viscometer (open circles) and disposable system (closed circles). Results compared at three different temperatures.

Comment

The relative viscosity of control serum at the three temperatures using both apparatuses shows that the disposable apparatus produces similar results to the Ostwald apparatus. The needle syringe system has the advantages of being disposable, using smaller volumes of serum, and having a slower run-through time, which almost certainly reduces observer error in timing. Moreover, it is known that the disposable system used at ambient room temperature can be valuable in assessing the viscosity of serum from patients with myeloma.² In a larger study on polymerised IgA paraproteinaemias Chandy *et al*³ have shown that occasionally individuals may show a large increase in viscosity at the lower temperature. Thus measuring the relative viscosity of serum from patients with myeloma at ambient ward temperatures is a valuable procedure and may be carried out using standard disposable ward equipment.

The system may also be of value in various conditions associated with raised plasma protein values and small vessel disease. Data (unpublished) from 30 patients with insulin-dependent diabetes has shown an increase in serum viscosity similar to that observed using absolute values on more advanced apparatus.⁴ Increased blood viscosity, due in part to increases in plasma protein values, is probably

a major contributor to diabetic microangiopathy.^{4,5} Hyperfibrinogenemia is probably an important element,⁵ so that the system should be evaluated using plasma from diabetic patients. The system described is clearly feasible as an outpatient test and thus may be of value in assessing risk factors for the vascular complications of diabetes.

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Nephrogenic diabetes insipidus associated with Dyazide (triamterene-hydrochlorothiazide)

We report a case of nephrogenic diabetes insipidus in an elderly man after two months' treatment with the combination diuretic Dyazide (triamterene 50 mg and hydrochlorothiazide 25 mg).

Case report

A 68-year-old man was admitted complaining of anginal chest pain associated with dyspnoea and secondary complaints of apathy and weight loss. His history included a myocardial infarction eight years before. He smoked 10 cigarettes a day, drank little alcohol, and was not taking drugs. On admission there was clinical and radiological evidence of pulmonary oedema. Electrocardiography showed atrial fibrillation with no recent evidence of myocardial infarction. Plasma urea and electrolyte concentrations, serial cardiac enzyme activities, and thyroid function values were normal. Erythrocyte sedimentation rate was 70 mm in the first hour. He was treated with digoxin, frusemide (40 mg/day), and potassium supplements (600 mg thrice daily) with good response. Before his discharge six days later Dyazide one tablet daily was substituted for the frusemide and potassium supplements. At the time plasma urea and electrolyte concentrations were normal.

At review after one month the patient complained of exertional dyspnoea and bouts of irregular palpitations (presumed to indicate paroxysmal atrial fibrillation). Dyazide was increased to two tablets daily and warfarin instituted, which maintained his prothrombin time ratio between 2.0 and 2.5. One month later he complained of polyuria and polydipsia, anorexia, and symptoms of postural hypotension. He looked dehydrated. He was in sinus rhythm and free of cardiac failure. Investigations showed plasma urea concentration 15 mmol/l (90 mg/100 ml), sodium concentration 133 mmol(mEq)/l, potassium concentration 2.8 mmol(mEq)/l, total CO₂ concentration 27 mmol(mEq)/l, and creatinine concentration 214 μmol/l (2.4 mg/100 ml). Plasma calcium, phosphate, glucose, and urate concentrations, liver function values, and immunoglobulins were all normal with no abnormal bands on plasma protein electrophoresis. Full blood count was normal but the erythrocyte sedimentation rate was 108 mm in the first hour. Plasma digoxin concentration was satisfactory at 1.9 nmol/l (1.5 ng/100 ml). Urine analysis was negative for protein, blood, and glucose with absence of Bence Jones protein. Midstream urine specimen was sterile. An intravenous pyelogram was normal. Urinary volumes were on average 3.5 l/24 h, with an average intake of 2 l/24 h. A 12-hour water-deprivation test showed failure of urinary concentration at 12 hours with a urinary osmolality of 337 mmol(mosmol)/kg and a plasma osmolality of 298 mmol(mosmol)/kg and loss of 2 kg over the period (3.6% of body weight). There was no improvement in urinary osmolality (305 mmol/kg) after 5 μg desmopressin given

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 hypokalaemia¹ 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.

We thank Dr Andrew Doig for valuable advice.

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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 chromatography⁵ 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 mg/kg/day."