cultured from his central venous line. Within a few hours of this episode muscle enzyme values rose sharply (table) followed two days later by a drop in urinary output. The infection was controlled by the administration of netilmicin and the muscle enzyme values returned to normal. Late hypercalcaemia (2·63 mmol/l (10·5 mg/100 ml)) was noted as the patient's renal function improved.

Case 3—A 52 year old man was admitted with a severe chest infection. He gave a one-week history of fever, nausea, vomiting, severe headaches, and myalgias. Within a few hours of admission he developed acute respiratory failure necessitating artificial ventilation. Chest *x*-ray films showed bilateral lung infiltrates predominantly affecting the left upper lobe. He was treated with intravenous erythromycin as an atypical pneumonia was suspected. A few days later the muscle enzyme values rose with a rapid decline in renal function and numerous pigmented granular casts in the urine. Acute renal failure secondary to rhabdomyolysis with myoglobinuria was diagnosed and haemodialysis started. As the chest infection slowly improved a diagnosis of *Legionella pneumophila* infection was made by serology (titre < 1/8 rising to 1/32). Muscle enzyme values returned to normal 10 days after the onset of rhabdomyolysis.

Comments

Myalgias are frequently part of viral and bacterial infections. Rhabdomyolysis, however, has only recently been associated with systemic diseases; some patients with *Legionella pneumonia* infection have presented with severe rhabdomyolysis leading to acute tubular necrosis.[3] The mechanisms underlying muscle damage during infections are poorly understood and are likely to be multiple. Severely ill patients with systemic infections are often hypoxic, dehydrated, and acidotic; this together with concomitant electrolyte disorders and hypophosphataemia[4] can predispose to muscle damage and subsequent acute renal failure.

We believe that rhabdomyolysis is underdiagnosed during infections; the recognition of this association could lead to prompt rehydration, correction of acidosis, and perhaps forced alkaline diuresis.[5]

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(*Accepted 9 September 1982*)

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Urethral gonococcal infection by co-existing beta-lactamase and "resistant" strains of gonococci

Detection of more than one strain of gonococcus from different sites in one person has been reported many times.[1] We report a case of two strains of gonococci isolated from the same site at the same time in one person.

Case report

A 34 year old divorced miner took a week's holiday in Bangkok, during which time he indulged in sexual intercourse with "quite a few" consorts. On 16 August 1982 he attended this department with a three day history of urethral discharge. Gonorrhoea was diagnosed clinically on the basis of a Gram film showing typical intracellular Gram negative diplococci. On epidemiological grounds he was treated with 2 g intramuscular spectinomycin dihydrocholoride.[2 3]

Subsequent laboratory culture of the urethral discharge showed the presence of two distinct strains of gonococci from the single site. One had a minimal inhibitory concentration of 0·3 mg/l to penicillin and was sensitive to all the usual range of antibiotics except co-trimoxazole; the other strain was a β-lactamase producer resistant to penicillin G, trimethoprim, ampicillin, and amoxycillin. Both strains were sensitive to erythromycin, clindamycin, tetracycline, cefuroxime, spectinomycin, streptomycin, kanamycin, and acrosoxacin. The β-lactamase strain was confirmed by the venereal diseases reference laboratory at the London Hospital.

Comment

We found no report of two differing strains of gonococci being isolated from the same site at the same time in one person. The two strains in our patient may have been contracted at different times from more than one partner or, more improbably, from one partner only. The penicillin-sensitive strain may have been undergoing mutation into a β-lactamase strain. Had treatment with penicillin been used initially in this case and full sensitivity tests not carried out, a partial improvement might have been noted clinically and the subsequent presence of gonococci diagnosed as a recurrence. Out of a total of 89 separate isolates of β-lactamase-producing strains in our laboratories, this is the first time that two distinct strains have been isolated from one site at the same time in one patient. This serves to reinforce the utmost importance of carrying out sensitivity tests on all gonococcal isolates.

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(*Accepted 15 October 1982*)

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Antiemetic effect of nonabine in cancer chemotherapy: a double blind study comparing nonabine and chlorpromazine

The psychotropic agent nonabine is a chromenol derivative with antiemetic properties and a similar pharmacological profile in animal models to that of tetrahydrocannabinol.[1] We carried out a study to compare the effects of oral nonabine on nausea and vomiting in patients receiving cancer chemotherapy with the effects of chlorpromazine, a drug of proved efficacy in these circumstances.[2] The study was approved by the hospital ethical committee. In view of the disturbing nature of the symptoms a placebo was not included.

Patients, methods, and results

We chose a randomised, double blind crossover design in which we studied 12 patients with lymphoma (eight men and four women aged 41-82 (mean 59·3) years) receiving standard MOPP (mustine, vincristine, procarbazine, and prednisolone) or CHOP (cyclophosphamide, adriamycin, vincristine, and prednisolone) chemotherapy regimens (four and eight patients respectively). Patients received six courses of chemotherapy (a total of 139 treatments) and one hour before infusion were given by mouth 15 mg nonabine, 99 mg chlorpromazine, or a combination of these drugs. In one patient a combined dose of 15 mg nonabine and 99 mg chlorpromazine produced excessive sedation and was subsequently reduced to 10 mg nonabine and 66 mg chlorpromazine. The same antiemetic was given on days 1 and 8 of a particular course since we had a strong clinical impression that

the second cytotoxic treatment of a pair usually causes less vomiting than the first.

About 18 hours after treatment patients assessed nausea by marking a unipolar visual analogue scale 100 mm long at the point most closely equivalent to their experience, 0 mm being equivalent to no nausea at all and 100 mm the worst nausea imaginable. They recorded the number of vomits with a digital counter, and nausea and vomiting were also graded by a ward observer as none, mild, moderate, or severe. To assess tolerance the occurrence of any side effects was recorded.

Data were analysed by non-parametric statistical methods—namely, visual analogue scores by a split plot analysis of variance after application of an arcsine square root transformation to the raw data, and the total number of patients who did not vomit with the Mann-Whitney test.

The incidence of vomiting with the MOPP regimen was greater than that with the CHOP regimen ($p < 0.05$), but there was no significant difference in these groups between the effects of the three antiemetic treatments. The table shows that all three treatments were similarly effective in preventing vomiting.

Proportions (and %) of patients who did not vomit over 18 hours

Regimen	Antiemetic		
	Chlorpromazine 99 mg	Nonabine 15 mg	Combination
MOPP	9/15 (60)	7/16 (44)	9/16 (56)
CHOP	25/31 (81)	28/32 (88)	24/29 (83)

MOPP = Mustine, vincristine, procarbazine, and prednisolone.
CHOP = Cyclophosphamide, adriamycin, vincristine, and prednisolone.

No patients rated their vomiting as severe, and nausea was rated only mild to moderate. In the patients who received the MOPP regimen the mean visual analogue score was significantly lower on the eighth day than on the first day with all three antiemetic treatments ($p < 0.01$), confirming the impression that the second cytotoxic treatment of a pair causes less nausea than the first. In this group on day 1 nausea was subjectively less after chlorpromazine than after nonabine, but this trend was not significant.

Comment

Both nonabine and chlorpromazine appeared adequate for controlling nausea and vomiting in patients treated with the CHOP regimen but failed to control vomiting in around 40% of those receiving the MOPP regimen. Possibly this figure could be improved by giving higher or repeated doses. Combining nonabine with chlorpromazine did not improve the antiemetic effect and was so sedative that the doses had to be reduced.

Side effects were fairly minor with each antiemetic, several symptoms, including drowsiness, dizziness, dry mouth, and headache, being common to both drugs. Cannabis-type "highs" did not occur, and postural hypotension and vagotonia, which have been reported with cannabis and its analogues, were also absent.[3-5]

This study shows that the antiemetic effects of nonabine and chlorpromazine in the doses used are similar. Side effects were not a serious problem with either drug.

We thank Beechams Pharmaceuticals research division, and in particular Dr S Thompson, for supplying of drugs, and Mr D M Rose for the statistical analysis.

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(*Accepted 3 December 1982*)

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Cerebrovascular accident in a 14 year old marathon runner

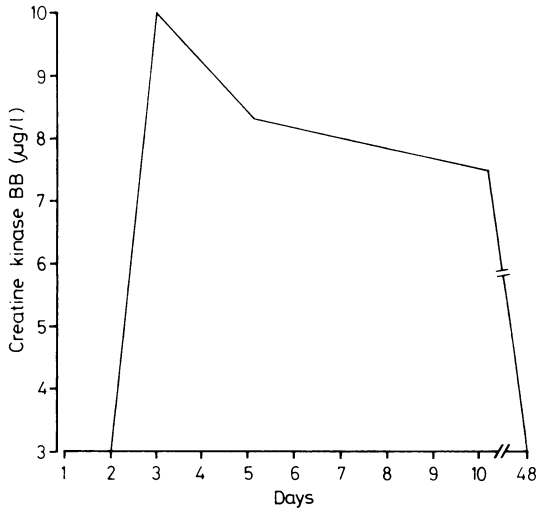
Marathon running is now extremely popular, and the large, publicised national events attract fit, well-acclimatised competitors. Medical directors dissuade ill-prepared or unhealthy people from competing and provide adequate ambulance and medical services during races. None the less, the body's complex physiology is tested by the arduous exertion associated with marathon running, and ill effects may be experienced, even by young, healthy trained competitors. We report here the onset of a cerebrovascular accident in a 14 year old boy during a 13-mile marathon.

Case report

A 14 year old healthy schoolboy, who participated regularly in sporting activities, had trained specifically for a 13-mile run through the streets of Dublin. After 10 miles he felt unwell, developed heaviness in his right leg, and after a further five strides collapsed with right-sided weakness.

On arrival at hospital he was alert but dysphasic with right hemiplegia. He was transferred to our neurosurgical unit, where a computed tomogram of the brain showed several low-density areas in the territory of the left middle cerebral artery, suggesting ischaemia or early infarction. Bilateral carotid angiography after 48 hours showed delayed filling and incomplete opacification of several ascending frontoparietal branches of the left middle cerebral artery. The cervical carotid arteries were smooth and of normal calibre. Conventional and two-dimentional echocardiography showed normal heart and valves. Results of haematological investigations, including clotting times, platelet count, and studies of platelet function, were normal. Blood viscosity, estimated from the packed cell volume and serum globulin and fibrinogen concentrations, was normal. The cerebrospinal fluid contained 11 lymphocytes. The creatine kinase BB level, measured by radioimmunoassay, was raised on days 3, 5, and 10 (figure).

He was treated with bed rest, steroids, and low-molecular-weight intravenous fluids. After five days all neurological signs had improved, and he was discharged at 12 days. There were no abnormalities at six weeks. There was no evidence of a collagen disorder and no history of migraine.



Serum creatine kinase BB measured by radioimmunoassay on days 2, 3, 5, 10, and 48. (Normal range 0-3 µg/l.)

Comment

Neurological complications may be associated with excessive dehydration and an altered packed cell volume. Marathon runners are advised to take adequate fluid replacement during races. It may be important that our patient did not do this. None of his haematological variables indicated dehydration, but most were measured four hours after the race, after oral intake of fluid. The major causes of "collapse" in the 1982 London marathon was dehydration, although most runners who retired did so because of cramp or fatigue.[1] While the physiology of red-cell deformability and serum viscosity during prolonged exertion is complex, a relation exists between increased blood viscosity, decreased cerebral blood flow, and cerebral infarction.[2] Changes in viscosity together with cerebral hypoxia not measurable on routine screening and influenced by inadequate fluid replacement may have contributed to this athlete's temporary neurological complication.