

The data sheet for acyclovir currently recommends 5 mg/kg eight hourly for normal patients and 10 mg/kg for those who are immunocompromised. After our experience with the first patient the second and third were given a higher dose. The fourth patient may have avoided pneumonitis through its early use. Varicella zoster is known to be less sensitive than herpes simplex virus. Reversible impairment of renal function is the most important adverse effect, resulting from renal deposition when hydration or renal function is inadequate. Acyclovir should be infused slowly with a litre of fluid for each gram of the drug.⁴ Animal studies have not shown adverse effects from acyclovir on the fetus, and treatment of disseminated herpes simplex in pregnancy in humans has been reported with no apparent fetal damage.⁵

Our experience suggests that chickenpox in pregnancy is sufficiently hazardous to merit admission to hospital and early treatment with acyclovir, especially if pulmonary damage is suspected. Possible harmful effects to the fetus must be considered but do not seem to have occurred in our patients, who received treatment in the second and third trimesters.

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Failure of salt to increase starch digestibility and glycaemic response

The hypothesis that dietary starch is digested more rapidly when it is taken together with salt is plausible but rests only on a report that six normal subjects tended to have higher plasma glucose or insulin concentrations when they breakfasted on lentils or bread with added salt.¹ Another small study found no difference in glycaemia when salt was added to glucose or macaroni test meals.² We tested this hypothesis in a larger group of subjects, measuring insulin as well as glucose responses and also estimating the escape of undigested starch from the small intestine. The test meal was designed to be a realistic, mixed meal.

Subjects, methods, and results

Thirteen healthy non-obese volunteers (aged 20-69) took part in the study. Four were male medical students and nine (four men) had normally functioning ileostomies after a curative colectomy for ulcerative colitis performed at least one year beforehand. No subject was taking any medication.

Test meals (52.4 g carbohydrate, 17.1 g protein, 17.1 g fat) consisted of 21 g Cheddar cheese, 8 g butter, 300 ml tea, 50 g milk, and a baked wholewheat bread scone containing 50 g carbohydrate. The scones consisted of 76 g finely ground wholewheat flour, 0.75 g sodium bicarbonate, 1.5 g cream of tartar, and 50 g water; the salted scones contained in addition 4.25 g of sodium chloride. The total sodium contents of the salted and unsalted meals were 87 mmol and 16 mmol respectively. All the scones were baked undisturbed at 210°C for 15 minutes in the same oven. After baking they were cooled then stored at -18°C. When required they were thawed overnight.

After a 12 hour overnight fast venous blood samples were taken in the fasting state and 10, 20, 30, 40, 50, 60, 75, 90, 120, and 180 minutes after the test meal (except in one subject with an ileostomy in whom venous access was not possible). Ileostomy effluent was collected in hourly samples for eight hours after the test meal and immediately stored at -20°C until the time of assay. Test meals were administered in random order and were consumed steadily over 20 minutes under supervision. Plasma glucose concentrations were measured in an autoanalyser by the glucose oxidase method. Plasma insulin was estimated by radioimmunoassay.³ Starch in the homogenised ileostomy effluent was measured as the glucose

released during incubation with amyloglucosidase.⁴ Areas under the insulin and glucose concentration curves were estimated as those above the zero time point. Student's paired *t* test was used in comparing the response to the salted and unsalted meals. Results are expressed as means (and standard errors). The study was approved by the Bristol and Weston health district ethical committee.

The meals containing unsalted and salted bread produced no significant differences in plasma glucose concentration at any postprandial time point or in area under the glucose concentration curve (table). In addition neither individual postprandial plasma insulin values nor area under the insulin concentration curve differed significantly between the two meals (table). If it had any effect, salt tended to reduce the metabolic response to starch.

Plasma glucose and insulin responses to mixed meals based on bread with and without added salt in 12 subjects. Results are means (and standard errors)

	No salt	Salt
Fasting glucose (mmol/l)	3.85 (0.15)	3.97 (0.14)
Peak glucose (mmol/l)	5.95 (0.26)	5.90 (0.20)
Area under glucose curve (mmol/l min)	145 (22)	105 (17)
Fasting insulin (mU/l)	5.00 (1.00)	6.00 (1.00)
Peak insulin (mU/l)	49 (5)	49 (6)
Area under insulin curve (mU/l min)	3728 (411)	3059 (287)

Comment

These findings make it unlikely that salt increases the rate of starch digestion to any important extent, at least in the context of a normal mixed meal.

Starch output in the subjects who had had ileostomies tended to be higher after the meals containing salted bread, not lower as predicted by the hypothesis, but this difference was not significant (502 (216) mg glucose *v* 397 (164) mg glucose, *n*=9). Clearly, salt does not reduce the physiological malabsorption of starch which occurs in healthy subjects.^{4,5} This again makes it unlikely that salt affects starch digestion to an important extent in real life. In the light of these data, salt restriction is unlikely to help control postprandial glycaemia in diabetics.¹

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Cholesterol embolism in a renal graft after treatment with streptokinase

The clinical features of cholesterol embolism in the kidney have been well delineated.¹ We report a case of acute renal graft failure due to cholesterol emboli. We further suggest that fibrinolytic treatment caused the disease and draw attention to this potential complication of an increasingly popular treatment of myocardial infarction.^{2,3}

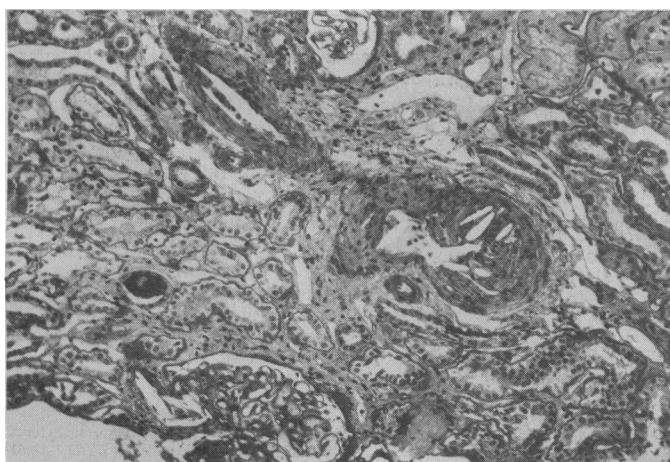
Case report

A 56 year old man was admitted to hospital with chest pain that had lasted four hours. Nineteen years earlier he had received a cadaver kidney transplant after a 20 year history of chronic glomerulonephritis with hypertension. No rejection occurred. Serum creatinine concentration was stable at 150 µmol/l. Maintenance

immunosuppressive treatment included azathioprine 100 mg and prednisolone 8 mg daily. Persistent hypertension was treated with a β blocker and enalapril. Exertional angina pectoris had appeared 14 months before admission.

On admission an acute inferior myocardial infarction was diagnosed. Blood pressure was 180/120 mm Hg; serum creatinine concentration was 159 $\mu\text{mol/l}$. Intravenous fibrinolytic treatment was started with streptokinase as a single one hour perfusion (1.5×10^6 IU) and continued with heparin (1000 U/h). Twenty two hours later his serum creatinine concentration had risen to 336 $\mu\text{mol/l}$. A few hours later a gastric haemorrhage led to transient hypotension: the heparin infusion was stopped and packed red cells were given. His subsequent clinical course was uneventful: blood pressure remained normal without hypotensive drugs. Nevertheless, renal function continued slowly to deteriorate; a persistent mild haemolysis was documented.

Seventeen days after infarction the serum creatinine concentration was 504 $\mu\text{mol/l}$, lactate dehydrogenase activity 630 IU/l (normal <300 IU/l), bilirubin concentration 39 $\mu\text{mol/l}$ (indirect 33 $\mu\text{mol/l}$), and haptoglobin concentration 150 g/l. A graft biopsy specimen obtained on the same day disclosed numerous cholesterol clefts in the lumen of arcuate and interlobular arteries, preglomerular arterioles, and glomerular capillaries (figure). In several vessels an inflammatory reaction (including macrophages) and fibrosis surrounded cholesterol crystals, obliterating the lumen; a few subendothelial fibrin deposits were seen in some glomeruli. Funduscopy showed no cholesterol crystals. Ultrasonography showed severe atherosclerosis of the abdominal aorta with some plaques protruding into the lumen. Extensive three vessel disease was documented 10 days later by coronarography; only medical treatment (aspirin and β blocker) was continued.



Multiple needle shaped clefts in the lumen of an interlobular artery and a preglomerular arteriole (periodic acid Schiff).

After reaching a peak of 558 $\mu\text{mol/l}$ on day 21 the serum creatinine concentration decreased slowly; haemolysis disappeared. Seven months later the patient was asymptomatic and his serum creatinine concentration was 195 $\mu\text{mol/l}$.

Comment

The episode of acute renal failure observed in this kidney transplant recipient with long term stable renal function was clearly due to histologically proved cholesterol embolism. To the best of our knowledge this complication has not been reported in a kidney graft recipient.

The risk of renal cholesterol embolism is related to the severity of abdominal aortic atherosclerosis.¹ The high prevalence of atherosclerosis in renal transplant recipients together with the steadily growing number of long term survivors suggests that cholesterol embolism should be added to the causes of potentially reversible renal graft failure.

Cholesterol embolism in our patient was most probably due to fibrinolytic treatment. He had not undergone aortic surgery or arterial catheterisation. Although cholesterol crystal release in the circulation may occur spontaneously, in our patient renal failure developed within 24 hours of the start of streptokinase treatment. Glasscock *et al* have recently discussed a similar case of cholesterol emboli developing after streptokinase administration and reviewed the evidence that anticoagulation treatment triggers the disease.⁴ Streptokinase lyses thrombi, including those covering atherosclerotic plaques, and might thus release cholesterol debris into the blood stream. As intravenous streptokinase has become a routine treatment for early acute myocardial infarction^{2,3} this potential, though probably rare, complication should not be ignored in severely atherosclerotic patients.

The spontaneous recovery of this patient is noteworthy. Renal failure due to cholesterol embolism was previously regarded as irreversible. Recently, however, recovery of renal function, even after temporary dialysis, has been reported in several patients.^{1,5}

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Do psychiatric registrars take a proper drinking history?

Much attention has been focused on the failure of hospital doctors to detect excessive drinking in patients admitted to general wards.^{1,2} Alcohol has been estimated to be the cause, directly or indirectly, of about 27% of acute medical admissions.³ Research on early intervention has shown that a single session of counselling to problem drinkers in medical wards results in those individuals drinking less 12 months later than controls.⁴ A survey of patients admitted to a psychiatric hospital showed that nearly a fifth drink over eight units a day.⁵ These surveys have been conducted mainly by psychiatrists, who claim a greater sensitivity in detecting alcohol related problems. This study aimed at assessing whether psychiatric registrars working in a teaching hospital took an adequate drinking history on admission.

Patients, methods, and results

The case notes of 100 consecutive new admissions to the Maudsley Hospital were studied. Fourteen admissions to the alcohol treatment unit were excluded, leaving 46 men and 40 women (mean age 34.5). The main categories of diagnosis were affective psychoses, neurotic depression, schizophrenic psychoses, personality disorder, anorexia nervosa, and miscellaneous psychiatric disorder (ICD 9). Drinking and smoking histories were checked. All the histories were taken and recorded by psychiatric registrars and were classified according to the adequacy of the drinking history: (a) no mention, (b) qualitative comment, such as "social drinker," (c) quantitative assessment—for example, teetotaler or five pints of beer a night, etc.

A quantitative drinking history was obtained in only 26, while 42 had a qualitative comment and 18 had no mention of alcohol. The 86 histories were completed by 35 psychiatric registrars, of whom only 15 recorded quantitative histories. This group of registrars recorded 42 of the cases and on only two occasions did they omit to mention alcohol. The 17 registrars who recorded qualitative comments only were more likely to omit any mention of alcohol. Twelve registrars failed to record an alcohol history on some occasion.

There was no mention of alcohol in 12 (30%) of the women compared with 6 (13%) of the men but this difference did not reach statistical significance. The drinking histories of the different diagnostic groups were broadly comparable except in the affective disorder category. While all 14 of the depressed men were asked about their alcohol consumption, there was no reference to drinking in two (13%) of the 15 depressed women. Seven of the depressive men, however, had a quantitative history, compared with only one of the depressive women.

Of the six patients with a history of alcohol abuse only one had a detailed breakdown of the typical drinking day or a lifetime drinking history.

Smoking was not mentioned in the histories of 36 subjects, five had a qualitative comment, and 45 had a quantitative assessment.

Comment

The failure of the psychiatric registrars to make any comment on alcohol consumption in 21% of their patients is only slightly better than the failure of junior hospital doctors to document alcohol consumption in 39% of their patients.¹ The quantitative assessment of alcohol consumption by psychiatrists was even poorer than that of housemen (30% v 37%).¹ This is evidence of an attitude to alcohol abuse that is shared across medical specialties. Despite the rising level of alcohol consumption among women,