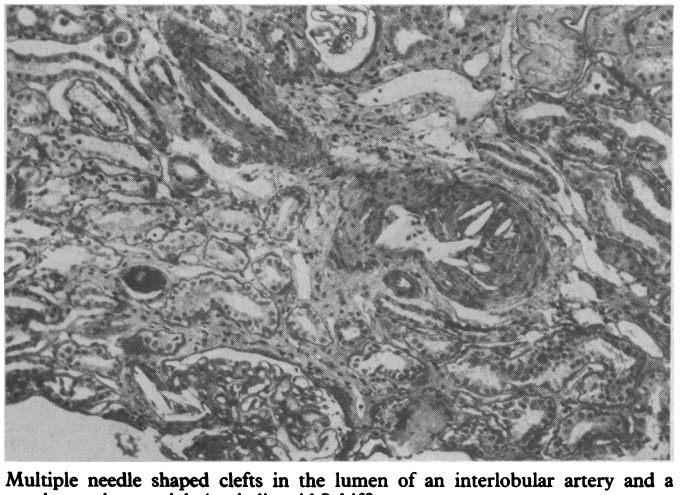
immunosuppressive treatment included azathioprine 100 mg and prednisolone 8 mg daily. Persistent hypertension was treated with a β blocker and enalapril. Exertional angina pectoris had appeared 14 months before admission.

On admission an acute inferior myocardial infarction was diagnosed. Blood pressure was 180/120 mm Hg; serum creatinine concentration was 159 μmol/l. Intravenous fibrinolytic treatment was started with streptokinase as a single one hour perfusion ($1{\cdot}5\times10^6$ IU) and continued with heparin (1000 U/h). Twenty two hours later his serum creatinine concentration had risen to 336 μmol/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 μmol/l, lactate dehydrogenase activity 630 IU/l (normal <300 IU/l), bilirubin concentration 39 μmol/l (indirect 33 μmol/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 μmol/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 μmol/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. Glassock *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, teetotaller 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 depressed 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,

there was a lower index of suspicion for women. In a study of the alcohol consumption patterns of 371 patients admitted to the Maudsley and Bethlem hospitals there was a large measure of agreement between the researchers' diagnosis and the hospital diagnosis.[5] It is unlikely that a primary diagnosis of alcoholism is being missed, but the registrars' failure to attend to other than overt alcohol problems related to alcohol when there is time to investigate such problems represents a valuable opportunity lost to influence a captive audience.

This is a small study of a teaching hospital population, and our data would benefit from comparison with those from a non-teaching hospital. Our findings suggest that the psychiatric registrars who take a quantitative alcohol history perform consistently better than their colleagues who record subjective qualitative comments. Doctors should always quantify the alcohol intake of their patients whatever the dignosis or sex. When this appears excessive then a lifetime drinking history and a daily drinking pattern should be fully documented.

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Severe orthostatic hypotension during treatment of falciparum malaria

Chloroquine is the drug of choice for chloroquine sensitive falciparum malaria and infections caused by *Plasmodium malariae*, *P ovale*, and *P vivax*. A fixed dose combination of sulfadoxine 500 mg and pyrimethamine 25 mg (Fansidar) is the most commonly used treatment for chloroquine resistant malaria. Recently a fixed dose triple combination of mefloquine 250 mg, sulfadoxine 500 mg, and pyrimethamine 25 mg (Fansimef) has also been found to be effective against chloroquine resistant malaria.[1 2] Serious side effects from these drugs are uncommon; severe orthostatic hypotension has not been reported.

Patients, methods, and results

In combined studies 189 male patients aged 12-50 were admitted to hospital with symptomatic falciparum malaria. None was seriously ill or hypotensive (supine blood pressure measured after 10 minutes' rest, erect blood pressure measured after three minutes' standing). Patients received either a single dose of sulfadoxine 1500 mg and pyrimethamine 75 mg (99 patients); a single dose of mefloquine 500 mg, sulfadoxine 1000 mg, and pyrimethamine 50 mg (50 patients); or chloroquine 1500 mg over three days (40 patients). Erect and supine blood pressures were measured regularly after treatment, though at different frequencies in each study (table). In most patients there was complete clearance of asexual parasitaemia and return to normal body temperature within three days of starting treatment.

At some time during their hospital stay, however, 26 patients were found to have unrecordable blood pressures on standing (table). Some patients fainted on standing; others could stand for three minutes before fainting or feeling faint. In every case blood pressure was normal immediately before standing and had returned to normal within five minutes after resuming the supine position. One patient had a sinus bradycardia (ventricular rate 44/min) immediately after an episode of postural hypotension; no other arrhythmias were recorded. Only three of the 26 patients had normal body temperature at the time of their hypotensive episodes.

Orthostatic hypotension was not associated with age, weight, severity of parasitaemia, or pretreatment blood pressure. Two of the patients in whom postural hypotension occurred had further attacks of falciparum malaria about 10 weeks later; both were treated with a further single dose of sulfadoxine 1500 mg and pyrimethamine 75 mg. Only one had a recurrence of his postural hypotension.

Comment

Treating American servicemen, Butler and Weber found that orthostatic hypotension was a common and prominent clinical feature of malaria caused by *P falciparum* and *P vivax*. They attributed it to the relative bradycardia and peripheral dilatation that occurs in malaria. Attempts to improve the orthostatic hypotension by rehydrating the patients before giving them antimalarial drugs were unsuccessful.[3]

The patients in our series who experienced severe orthostatic hypotension did so only after antimalarial treatment had begun. An explanation may be that concentrations of blood histamine increase during attacks of falciparum malaria[4] and pyrimethamine and chloroquine inhibit histamine *N*-methyltransferase,[5] an enzyme responsible for histamine metabolism in the mammalian central nervous system. Together these may produce increased central effects of histamine and hypotension. That only one of 50 patients given the lower doses of sulfadoxine and pyrimethamine developed postural hypotension is consistent with this hypothesis.

Patients prescribed antimalarials for falciparum malaria and people responsible for their care should be aware of the possibility of postural hypotension.

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Incidence and duration of orthostatic hypotension in patients treated for falciparum malaria

Treatment	No of patients	Frequency of blood pressure measurement	No with unrecordable blood pressure on standing	Interval between treatment and onset of hypotension (h)	Duration of hypotension (h)
Sulfadoxine 1500 mg, pyrimethamine 75 mg	50	Daily	10	14-48	24-72
	39	8 Hourly	10	9-44	6-40
	10	Hourly for 24 h, then 8 hourly	2	14, 16	5-24
Mefloquine 500 mg, sulfadoxine 1000 mg, pyrimethamine 50 mg	50	Daily	1	15	24
Chloroquine 1500 mg	40	Hourly for 24 h, then 8 hourly	3	2-4	3-12