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Lean, dry gout patients

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Hypertriglyceridaemia is common in gout.^{1,2} Obesity may increase triglyceride concentrations, as may alcohol,³ but it remains uncertain whether obesity and alcohol, alone or in combination, are sufficient to explain the hyperlipidaemia in all cases. We looked for abnormal lipid concentrations in non-obese patients with gout who drank little or no alcohol to determine whether the hyperprebetalipoproteinaemia associated with gout occurred in such a lean and abstemious group.

All patients were of desirable weight or less for their age and frame⁴ and no patient drank more than one pint of beer per day or its equivalent. Such patients are rare, and only seven were found in four years from a busy clinic.

Fasting concentrations of lipid and lipoprotein were measured in serum at

a laboratory with its own control population.⁵ Serum uric acid concentrations were determined for the patients with gout but unfortunately data for the control population were not available.

Readings for serum cholesterol, triglyceride, β -lipoprotein and prebeta lipoprotein concentrations in the patients with gout lay within 2 standard deviations of the corresponding mean for controls. This means that they were within the 95% confidence limits for the control population and it is therefore unlikely that there is any real difference between the patients with gout and control populations.

In spite of the small numbers of these, 'lean, dry' patients, the results revealed no intrinsic hyperlipidaemia in subjects with gout when obesity and

an excess of alcohol were removed as causes of hypertriglyceridaemia.

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Cardiovascular disease and gout: a function of sex and age?

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Hypertriglyceridaemia is common in patients with gout and hyperuricaemia but it is still not known whether this results from a link between purine and lipoprotein metabolism or whether they occur together due to other associated facts, particularly obesity and high alcohol intake, both of which are commonly found in patients with

hyperuricaemia.¹ Nor is it firmly established whether patients with gout are, in fact, predisposed to premature cardiovascular disease, and, if so, whether the raised serum uric acid concentration operates as an independent risk factor, or only via its association with hypertriglyceridaemia and hypertension, which

are established as cardiovascular risk factors in their own right.² Another possibility—namely, that a raised serum uric acid concentration causes platelet hyperaggregability and hence thrombosis—has recently been investigated in our unit with negative results.³

Over a period of two and half years

60 patients have presented to our rheumatology department with gout: 51 men, mean age 55.2 years and mean age of onset 46.6 years and nine women, mean age 80 years and mean age of onset of 78.6 years. We studied the prevalence of hyperlipidaemia, hypertension, obesity, and cardiovascular disease in these two groups.

Eighteen men but none of the women had a significant hyperlipidaemia. Triglycerides were by far the most common lipids (type IV) to be raised, being found in 13 of the 18. Four had hypercholesterolaemia: two type IIa and two type IIb (serum triglycerides also raised). One patient had a pronounced rise of serum triglyceride and cholesterol due to accumulation in serum of intermediate density lipoproteins, type III hyperlipidaemia or broad β -disease.

Six men described themselves as teetotal, but three of these had a hyperlipidaemia. One had type IIa and had had a myocardial infarction at the age of 42, the other two had type IV. One of these, the hospital's appliance officer, was within the range of his ideal body weight and therefore

corresponded to those patients described by Dr Darlington (p. 90) with lean, dry gout. The other was massively overweight at 144 kg. None of the women drank on a regular basis but several did admit to the occasional sherry at Christmas, or the odd glass of Guinness as a 'tonic'. Only one of the women was above her ideal body weight, the remainder tending to be thin and frail.

Twenty men and two women had hypertension, defined as a resting diastolic blood pressure above 100 mmHg on two or more occasions, or receiving established treatment for hypertension. The patients were questioned about history of myocardial infarction and symptoms of angina. They were examined for signs of peripheral vascular disease and all underwent electrocardiography. On this basis 10 men and three women had appreciable cardiovascular disease. This did not correlate with hyperlipidaemia except in the two patients with type IIa, and nor did it correlate with hypertension.

These observations are on a small number of patients and the study was uncontrolled, but the interesting fact

that emerges is the great disparity in the ages of the men and women presenting with gout and the virtual absence of associated factors such as obesity, hypertension, and hyperlipidaemia in the elderly women. This could be interpreted as implying two quite separate disease processes in the two groups. It may be significant in this regard that all the women were receiving potent diuretic treatment. An alternative explanation could be that women are much more prone to cardiovascular disease in association with gout and many had died at a younger age; this, however, is contrary to clinical experience.

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Study of blood coagulation in gout patients

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Patients with gout have vascular risk factors such as hyperlipidaemia, hypertension, 'type A' personality, and, possibly, hyperuricaemia, obesity, and glucose intolerance. We used a modern method to seek abnormalities of coagulation in patients with gout.

Twelve fasting men with primary gout were studied. Patients were asked to take no medication, particularly aspirin, for the two weeks preceding the tests. No patients were included within three months of trauma or surgery and an atraumatic venepuncture technique was used.

Assays were performed to fibrinopeptide A (FpA)¹ with

Table 1 Comparison of mean data from patients with gout and from controls

	Controls	Patients with gout	Significance
FpA (pmol/l):			
No.	13	10	
Mean	0.98	1.53	NS
SD	0.29	0.77	
FpB β 1-42 (pmol/l)			
No.	10	9	
Mean	1.65	2.92	NS
SD	0.63	1.85	
β TG (pmol/l)			
No.	9	12	
Mean	1.00	1.58	NS
SD	0.28	1.81	

NS = Not significant.