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Can arterial catheterisation induce autoimmune disease?

Struthers *et al*¹ reported a case of polyarthritis nodosa (PAN) related to angioplasty. However, the absence of cholesterol emboli on muscle biopsy material was not mentioned in that paper. This could be an interesting fact as patients with cholesterol microembolisation, after arterial procedures, have been described as presenting features of PAN.²

We admitted a 66 year old man, who underwent cardiac catheterisation and presented with malaise, fever, livedo reticularis, purpura, distal ischaemic lesions in lower extremities with normal peripheral pulses, six weeks after the invasive procedure. On admission, he developed stupor, renal failure and mesenteric ischaemia, and died of multiorgan failure. The most remarkable laboratory findings were: elevated erythrocyte

sedimentation rate, anaemia, mild leucocytosis with eosinophilia, thrombopenia, increased nitrogen and creatinine, and positive circulating immune complexes, rheumatoid latex and antinuclear antibodies. Antineutrophil cytoplasmic antibodies, antiglomerular basement membrane antibodies, antiphospholipid antibodies and cryoglobulins were negative. Serum complement was normal. Blood cultures and hepatitis B antigens were negative. Muscle biopsy disclosed cholesterol microembolisation in the small vessels and inflammatory vascular infiltrate.

In this patient, histological findings were consistent with multiple embolisation cholesterol disease (MECD), but clinical and biological features strongly suggested autoimmune disease associated with vasculitis of the small and medium arteries. Although the precise pathogenesis of MECD remains uncertain, its close similarity to necrotising vasculitis point to an immunological phenomena probably triggered by a mechanical or ischaemic endothelium damage during invasive procedures. Similar case reports¹⁻³ could be involved in the wide clinical and biological spectrum of the same underlying disease.

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depressed androgen synthesis. It is well established that alcohol, though it may increase libido, lowers men's testosterone concentrations.² Provided that depressed androgen synthesis in patients with RA is a predictor rather than a consequence of the disease, it might be expected that alcohol consumption would be a risk factor for RA.

To test this hypothesis, we studied alcohol consumption for its association with the incidence of seropositive RA in a cohort of Finnish men. In this cohort, the questionnaire measure for total alcohol intake proved to be reliable at an interval of half a year (intraclass correlation coefficient, $r=0.73$) and closely associated with the incidence of severe fall injuries.³ RA cases were identified on the basis of their entitlement to free medication under the sickness insurance covering the entire population of Finland.⁴ In the 9777 men who had neither arthritis nor a previous history of it at the start of the study, 30 incident cases of seropositive RA occurred during 134 083 person years.

In men consuming more than 500g per month of alcohol (table), the age-adjusted relative risk of seropositive RA seemed to be slightly elevated (model 1). There are many factors, however, that correlate closely with alcohol use and could confound this association. Drinking and smoking, in particular, are related habits, and in this cohort smoking was a strong risk factor for seropositive RA in males.⁴ When allowance was made on potential confounders, the association between alcohol consumption and RA incidence even turned to inverse (model 2).

Thus our study does not provide any evidence for the role of alcohol as a risk factor for RA.

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LETTER TO THE EDITOR

Alcohol, androgens and arthritis

As recently discussed by James,¹ patients with rheumatoid arthritis (RA) frequently have

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Table Alcohol intake and the risk of seropositive rheumatoid arthritis (RA)

Alcohol intake (g/month)	Number of men examined	Number of RA cases	Model 1*		Model 2†*	
			Relative risk	95% confidence interval	Relative risk	95% confidence interval
0	2470	7	1.0		1.0	
1-99	1274	4	1.1	0.3-3.6	0.9	0.3-3.2
100-499	3782	10	0.9	0.3-2.3	0.6	0.2-1.6
500-	2251	9	1.3	0.5-3.6	0.8	0.3-2.2

*Cox's life-table regression adjusting for age.

†Adjusting for age, marital status, level of education, smoking, body mass index and physical activity at leisure.