PILOT STUDY OF BLOOD COAGULATION IN GOUT PATIENTS

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Gout patients have certain vascular risk factors such as hyperlipidaemia, hypertension, 'type A' personality and, possibly, hyperuricaemia, obesity and glucose intolerance.

Uric acid activates clotting Factor XII and hyperuricaemia in rats is associated with increased platelet aggregation (1) with increased adenosine diphosphate-induced pulmonary platelet thrombosis. Platelet survival is decreased and platelet turnover correspondingly increased in some primary gout patients and this abnormality is reversed during uricosuric therapy with sulfinpyrazone (2,3).

Studies of coagulation and of platelet function in gout have yielded variable results to date (2,4,5) but, in recent years, tests have become increasingly sophisticated and the present study was designed to use modern methods to seek any abnormalities of coagulation in patients with primary gout.

METHOD

Twelve, fasting, male patients with primary gout were investigated. Ten of the twelve patients were untreated, one had ceased allopurinol therapy two weeks before the tests and one was taking 400 mgs cimetidine daily. All patients were requested not to take any medication and, in particular, any aspirin for the two weeks preceding the tests. Every attempt was made to bleed the patients when they were relaxed, resting, after a fast of at least fourteen hours and at the same time in the morning to minimise effects on coagulation of emotion, exercise, food and diurnal variation. No patient was included within three months of trauma and/or surgery and an atraumatic venepuncture technique was used with a large needle.
Assays were performed to fibrinopeptide A (FpA) (6) with modifications (7), to fibrinopeptide B 1-42 (FpB 1-42) (8) with modifications (7) and to β-thromboglobulin (βTG) (9) with modifications (7). Serum cholesterol and triglycerides were measured directly (10) while high density lipoprotein cholesterol (H.D.L.C.) was estimated after differential precipitation of β and pre β-lipoproteins (11) followed by measurement of the cholesterol component of the α lipoprotein (10).

RESULTS

Data from gout patients (Table I) were compared with control data from normal subjects in the same age range (Table II).

No significant differences between gouty patients and controls were found for any of the tests performed although the FpA assay revealed an elevation among gouty patients which might have achieved significance with larger numbers of subjects (0.1 > p > 0.05).

Since one patient was taking cimetidine and another had only ceased allopurinol therapy two weeks before the tests all data were reanalysed after excluding these patients but results were unchanged.

Spearman Rank correlation coefficients were calculated between FpA, FpB 1-42 and βTG and lipid levels, alcohol consumption and weight since the latter data tend not to be distributed normally. Results are shown in Table III. Once again, inclusion of the two treated patients had no effect upon the correlation results.

It was not possible to calculate actual r values for the correlation with smoking since most patients were non-smokers, but simple assessment of the data did not suggest any relationship with smoking.

Correlation results must be considered with care since this was a pilot study on small numbers of patients. Furthermore, it must be remembered that abnormalities of coagulation detected in vitro are not necessarily present in vivo nor do they necessarily have a causal relationship with thrombosis.

In the Framingham Study (12) Hall found an increased risk of vascular disease in hyperuricaemic patients although our own study of vascular mortality among gout patients (13) did not confirm the relationship.

Certainly, in the present study, it was concluded that modern methods to assess coagulation and platelet function did not reveal any significant abnormalities in a pilot study among patients with primary gout.