FAMILY STUDY OF LIPID AND PURINE LEVELS IN GOUT PATIENTS AND ANALYSIS OF MORTALITY

L. G. Darlington, J. Slack, J. T. Scott

Epsom Rheumatology Unit, Epsom, Surrey.
Institute of Child Health, London, W.C.1
Kennedy Institute of Rheumatology, London, W.6

Many authors have noted an association between hyperlipidaemia and hyperuricaemia and Berkowitz (1964), Feldman and Wallace (1964) and Darlington and Scott (1972) all found raised triglyceride levels in gout.

Hypertriglyceridaemia occurs frequently and was found in 52% of gout patients by Darlington and Scott (1972) and Frank (1974) and in 75% of gouty patients by Berkowitz (1964). This association was not seen in patients with symptomless hyperuricaemia (Frank 1974).

In 1973, Mielants et al. found an increase in pre-β lipoproteins and a reduction in α- and β-lipoproteins.

In 1971, Emmerson and Knowles demonstrated hypertriglyceridaemia in gout patients persisting after correction for body weight and, in 1972, Darlington and Scott also described gout and hypertriglyceridaemia independent of obesity.

In 1974, Gibson and Grahame suggested that obesity, alcohol or both were the main causes of hypertriglyceridaemia in gout.

In 1979, Gibson et al. showed significant reductions in triglycerides in gout patients by reducing either alcohol intake or weight and, in the same year, Darlington and Scott, also found reductions in triglycerides on alcohol abstention although these did not achieve statistical significance.

In 1977, Elkeles and Chalmers raised triglyceride concentrations by fat infusion but did not find any subsequent effect on plasma lipids.
uric acid.

In 1979, Gibson et al. also used a fat tolerance test but did not demonstrate any effect of triglyceride on uric acid or of hyperuricaemia on triglyceride removal.

Unaffected relatives of gout patients may have an increased serum level of uric acid which is thought to be genetically determined (Smyth et al., 1948; Talbott, 1940; Hauge and Harvald, 1955).

In 1970, Rondier et al. concluded that gout does not have special features when associated with hypertriglyceridaemia but a family history is more frequent.

To determine whether the hypertriglyceridaemia of gout patients occurs in their families or is simply the result of the life-style of these usually obese, alcohol-drinking patients who frequently originate from higher social classes a family study was designed to measure lipid and uric acid levels in the blood of gout patients and their first-degree relatives and to compare them with those found in normal controls of the same age and sex.

A pedigree was constructed for 135 male and 9 female gout patients.

Index patients and relatives who agreed to help were weighed and fasting blood samples were taken.

Due to the regional and ethnic (Bronte-Stewart, 1955) differences in lipid concentrations control data was needed from subjects from the same geographical area and ethnic background as the gout group. A suitable control sample was available from a working population in N.W. London (Slack et al., 1977) and this was used to calculate standard deviation scores for lipid and uric acid levels.

The means and standard deviations for index patients and relatives are shown in Table 1 as standard deviation scores and significance was assessed. Triglyceride levels in gout patients were significantly higher than in relatives or controls (p < 0.001).

These changes were, predictably, reflected by changes in the proportion of pre-β-lipoprotein which was significantly higher in the gout patients than in relatives or controls (p < 0.001).

There was a corresponding, significant reduction in the proportion of β-lipoprotein in gout patients when compared with controls (p < 0.001) and relatives (0.01 > p > 0.001) resulting in a raised α:β ratio. There was no significant difference between the