HAEMATOCRIT VALUES IN ISCHAEMIC HEART DISEASE

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ABSTRACT
Haemoglobin (g/dl) and haematocrit (%) values were determined in 30 healthy controls and 40 patients with ischaemic heart disease. Both groups were non-smokers. Haemoglobin and haematocrit values (mean ±SE) in patients were 15.65±0.18 and 46.45±0.49 and in controls 14.30±0.14 and 41.50±0.23. Significantly higher (P<0.001) haematocrit value by influencing the blood viscosity may impose extra burden on the ischaemic heart (JPMA 43: 34, 1993).

INTRODUCTION
There are studies which show that blood viscosity is increased in ischaemic heart disease. Haematocrit and plasma fibrinogen are major determinants of blood viscosity. Fibrinogen is raised in patients of IHD. Reports regarding the role of haematocrit in the development of IHD are inconsistent. Follow-up data of 16 years from Los Angeles heart study shows no significant relationship between haematocrit and development of IHD. However, haematocrit is found significantly related to IHD outcome both as an independent risk factor and after adjusting for known confounding variables. Carter and his colleagues also provided some evidence of a significant relation between haematocrit and IHD. The results of case-control studies are contradictory. Early studies report higher haematocrit among patients of IHD than among controls, while Conley et al reported that the mean haematocrit levels of patients with myocardial infarction was lower than those of their normal controls. Nicolaides et al have also found significantly higher values of haematocrit in 22 patients of angina. Therefore, this study was aimed to evaluate the levels of haemoglobin and haematocrit in patients of IHD.

PATIENTS AND METHODS
Seventy male non-smokers (30 healthy subjects and 40 patients with ischaemic heart disease) were included in this study. After a careful history and review of medical records none were found to have bleeding disorders, respiratory disease, valvular heart disease, kidney diseases, hypertension and diabetes mellitus. Patients of ischaemic heart disease were diagnosed according to WHO criteria. Blood samples were obtained in the morning from resting subjects with minimum stasis from suitable antecubital veins and the tests were performed in duplicate within two hours of sample collection; the results were then averaged. Haemoglobin was determined by Cyanmet Hb method. Haematocrit values were estimated by microhaematocrit method on Hermle (West Germany) machine. The microhaematocrit tubes were filled upto 75% and the samples were run in duplicate for ten minutes, Chi square and Students ‘t’ test was used for statistical analysis.

RESULTS
The age (mean± SE) of 40 patients with ischaemic heart disease was 50.4± 1.07 and 30 controls 47.3±
1.4 years. Haemoglobin (g/dl) and haematocrit (%) values (mean±SE) of patients were 15.65±0.18 and 46.45±0.49 and of controls 14.30±0.14 and 41.50±0.23 respectively. The difference in the values of two groups was statistically significant (P <0.001).

DISCUSSION

For many years, resistance to flow in systemic circulation, specially in the veins, influences venous return very greatly, has been known. Since viscosity is one of the factors that determine resistance to flow, it is logical to believe that viscous changes in blood resulting from high or low haematocrit could greatly influence the various return to the heart and in this way also affects cardiac output. Cardiac output is decreased by increasing haematocrit, the maximum number of red cells present for oxygen transport to the tissue are near the mean normal haematocrit of 40%15. In certain patients, moderate erythrocytosis exists in association with coronary artery disease but it is usually ignored. In the cardiac patients it is probably wise to consider the erythrocyte concentration and to reduce even a moderately elevated haematocrit to mean normal levels16. Cullen et al17 showed a stepwise relation between increase in haemoglobin and a raised risk of both cardiovascular disease and ischaemic heart disease: The raised blood viscosity with a high haematocrit adversely affects collateral blood flow, the risk of thrombosis being related to the levels of the haematocrit. An increased circulating red cells mass increases the viscosity of blood, which may impair coronary perfusion. Both the haemoglobin-content and the percent of packed red blood cells influence the blood’s oxygen carrying capacity as well as the dynamic of flow and potential for clotting18. Though the study done by Carter et al9 showed significant relationship between HCl and IHD incidence and mortality, it failed to provide evidence for a significant relation when other risk factors were considered simultaneously, most influential being diastolic blood pressure, S. cholesterol and smoking. Cigarette smoking also increases the haematocrit values19-22. To exclude the effect of smoking, non-smoking controls as well as non-smoking cases were studied. This criteria was not considered by previous workers. The rise of haematocrit in angina was questionable to Nicolaides1 who thought smoking might be one of the causes of raised haematocrit in patients with angina. Abuzaid and Chapman7 showed no significant relation for haematocrit and development of IHD. However they stated that the rise of haemoglobin after the development of IHD may be speculated as being a compensatory mechanism to provide the ischaemic heart with blood of better oxygen carrying capacity. It is questionable how much the compensatory increase in haematocrit level with stenosed coronary vessels is beneficial to ischaemic heart which, on the other hand, also increases the blood viscosity. Increase in haematocrit is also related to the extent of coronary artery stenosis2. However, before the conclusion be drawn regarding haematocrit as an additional risk factor for IHD, further work is required to completely elucidate the role of haematocrit in the pathogenesis and/or therapeutics of IHD. The constituents of blood can be modified easily compared to relatively irreversible changes of blood vessels. Lowe23 also hoped to stimulate clinicians, dealing with circulatory problems, to think about the contribution of blood, which may be therapeutically modifiable when the heart or vessels are not.

REFERENCES


