HAEMATOCRIT VALUES IN ISCHAEMIC HEART DISEASE

Pages with reference to book, From 34 To 35

Shoaib Tauheed, Shaheen Shoaib (Department of Physiology, Dow Medical College, Karachi.)
Ahmad Kamal (Department of Physiology, Basic Medical Sciences Ingtitute, Jinnah Postgraduate Medical Centre, Karachi.)

ABSTRACT

Haemoglobin (g/dl) and haematocrit (%) values were detefmined in 30 healthy controls and 40 patients with ischaemic heart disease. Both groups wefe 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[1,2]. Haematocrit and plasma fibrinogen are major determinants of blood viscosity. Fibrinogen is raised in patients of IHD[3-6]. Reports regarding the role of haematocrit in the development of IHD are inconsistent. Follow-up data of 16 years from Los Angeles heart study[7] 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[8]. Carter and his colleagues[9] also provided some evidence of a significant relation between haematocrit and IHD. The results of case-control studies are contradictory. Early studies[10,11] report higher haematocrit among patients of IHD than among controls, while Conley et al[12] reported that the mean haematocrit levels of patients with myocardial infarction was lower than those of their normal controls. Nicolaides et al[1] 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[13]. Blood samples were obtained in the morning from resting subjects with minimum stasis from suitable anticubital 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[14]. 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 levels[16]. Cullen et al[17] 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 clotting[18]. Though the study done by Carter et al[9] 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 values[19-22]. 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 Nicolaides[1] who thought smoking might be one of the causes of raised haematocrit in patients with angina. Abuzaid and Chapman[7] 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 stenosis[2]. However, before the conclusion be drawn regarding haematocnt 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. Lowe[23] 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

1. Nicolajdea, A.N., Horboume, T., Bowers, R., Kinder, P.H. and Besterman, E.M. Blood viscosity, red cells flexibility, baematocrit and plasma fibrinogen in patients with angina. Lancet. 1977;2:943-45.
2. Lowe, G.D., Drummond, M.M., Lorimer, A.R., Hutton, I., Forbes, CD.. Prentice, C.R. and Barbenel, J.C, Relation between extent of coronary artery disease and blood viscosity. Br.Med.J., 1980;1:673-74.
3. Meade, T.W., Chakrabarty, R., Haines, A.P., North, W.R., Sterling. Y. and Thompson. S.G.

Haemostatic function and cardiovascular death. Early results of prospective atudy. Lancet, 1980;1:1050-53.
4. Wilhelmsen, L, Svardsudd, K., Bengaten, K.K., Lorson, B., Welin, L and T,bblin, 0. Fibrinogen as a risk, factor for stroke and myocardial infarction. N. Engl.J.Med., 1984;311:501-5.
5. Stone, MC. andThorpe,J.M. Plasma fibrinogen a major coronary risk factor. J.R.Coll. Gen. Prac., 1985;35:565-69.
6. Tauheed, S. and Shoaib, S. Importance, of plasma fibrinogen in ischaemie heart disease. Ann. Dow Med. Coll, 1991;1:53-55.
7. Abu-Zeid, HA. and Chsmpman, J,M.The relation between hsemoglobin levels and risk of ischaemic heart disease. A prospective study. J.Chronic Dis., 1975;29:395-403.
8. Sorlie, P.D., Garcia-Palmieri, M.R., Castillo-Stab, MJ., Costas, R. Jr. and Havlik, RJ. Haemstocrit and risk ofcoronsryhesrt disease; the Puerto Rico Heart Health Program. Am. HeartJ., 1981; 101:456-61.
9. Carter, C., McGee, D., Reed, 0., Yano, K. and Stemmermann, 0. Haematocrit and the risk of coronary heart disease. 'The Honolulu Heart Program. Am. Heart J., 1982;105:674-78.
10. Burcb, G. and DePasquale, N. The haematocrit in patients with myocardial infarction. JAMA, 1962;180:63-65.
11. Mayer, G.A. Haematocrit and coronary heart disease. Can. Med. Assoc. J., 1965;93:1151-53.
12. Conley, C.L., Russell, R.P., Thomas, C. and Tumulty, PA. Haematocrit values in coronary heart disease Arch. Intern. Med., 1964;113:170-76.
13. Reports of the Joint International Society and Federation of Cardiology/World Health Organisation Task Force on Standardization of Clinical Nomenclature. Nomenclature and criteria for diagnosis of ischemic heart disease. Circulation, 1979; 59:607-9.
14. Richardson, TO. and Guyton, A.C., Effects of polycythaemia and anaemia on cardiac output and other circulatory factors. Am.3. Physiol., 19S9;197:1167-70.
15. Guyton, A.C. and Richardson, T.O. Effect of haematocnt on venous return. Cir. Res., 1961;9:157-64.
16, Burch, 0. and DePasquale, N. Erytbrocytosis and ischaemic myocardiall disease. Am. Heart.J., 1961;62139-40.
17. Cullen, K.J., Stenhouse, N.S. and Wearne, K.L., Raised haemoglobin and risk of cardiovascular disease. Lancet, 1981;2:1288-89.
18. Kannel, W.B., Gordon, T., Wolf. P.A., McNamara, P.M. Haemoglobin and risk of cerebral infarction. The Framingham Study. Stroke, 1972;3:409-20.
19. Eisen, M.E. and Hammond, E.C., The effectof smokingon packed cell volume, red blood cell counts, haemoglobin and platelet counts. Can. Med. Assoc. J., 1956;75:520-23.
20. Gales, 0. and Davidson, R.J. Haematological and hsemorbeological changes associated with cigarette smoking. J. Clin. Pathol., 1985;38:978-81.
21. Ernst, E., Matrai, A., Sehomoizi, C. and Magyarosy, I. Doseeffect relationship between smoking and blood rheology. Br.J. Haematol., 1987;65:485.87.
22. Tsuheed, S.,Shoaib, S. and Kamal, A. Haemoglobin, haematocrit and plasma fibrinogen in cigarette smokers. J.Pak.Med.Assoc., 1992;42:162-63.
23. Lowe, G.D.O. Blood rheology and byperviscoaity syndromes, in Bailliere's clinical baematology. London, Bailliere, Tindal, 1987, p. 10.