Increased Red Cell Volume in Impaired Glucose Tolerance: A Further Evidence of Hematologic Sequelae of Altered Glucose Metabolism

The occurrence of an increased mean red cell volume (MCV) in diabetes has been reported.[1] Moreover, we have suggested that metabolic control may influence MCV in diabetes.[2] Since the measurement of HbA_1 has been reported to be a quick, accurate, and simple screening test to reveal patients with impaired glucose tolerance,[3] we have evaluated plasma glucose, HbA_1, and MCV in 150 male blood donors (age 22–40 yr), who had no family history of diabetes.

Our normal range of HbA_1 is 5–7%.[4] All subjects had normal fasting plasma glucose (4.84 ± 0.44 mmol/L; mean ± SD).

When the subjects were divided according to their HbA_1 values, those with levels of HbA_1 about 7% (35 subjects) presented higher values of MCV (89.74 ± 4.21 femtoliters) than those subjects with HbA_1 below 7% (115 subjects) (MCV = 86.47 ± 3.44 femtoliters; $P < 0.001$). There was no correlation between HbA_1 and MCV values.

These data show that impaired glucose tolerance may be associated with increased MCV.[5–8]

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REFERENCES

[1] Davidson, R. J. L., Evan-Wong, L. A., and Stowers, J. M.: The mean red cell volume in diabetes mellitus. Diabetologia 1981; 20:583–84.

[2] Ceriello, A., Dello Russo, P., Curcio, F., Balsamo, C., and Pietrantuono, C.: Red blood cell volume and glycaemic control in diabetes. Diabetologia 1983; 24:397.

[3] Hall, P. M., Cook, J. G. H., Sheldon, J., Rutheford, S. M., and Gould, B. J.: Glycosylated hemoglobins and glycosylated plasma proteins in the diagnosis of diabetes mellitus and impaired glucose tolerance. Diabetes Care 1984; 7:147–50.

[4] Ceriello, A., Giugliano, D., Dello Russo, P., and Passariello, N.: Hypomagnesemia in relation to diabetic retinopathy. Diabetes Care 1982; 5:558–59.

[5] Graham, J. J., Ryall, R. G., and Wise, P. H.: Glycosylated haemoglobin and relative polycythaemia in diabetes mellitus. Diabetologia 1980; 18:205–207.

[6] Ceriello, A., Dello Russo, P., Sgambato, S., and Giugliano, D.: Glycosylated haemoglobin and reticulocyte count in diabetes. Diabetologia 1982; 22:223.

[7] Peterson, G. M., Jones, R. L., Koening, R. S., Helvin, E. T., and Lehrman, M. L.: Reversible hematologic sequelae of diabetes mellitus. Ann. Intern. Med. 1977; 86:425–28.

[8] Dello Russo, P., Ceriello, A., and Curcio, F.: Reticulocyte counts and haemoglobin glycosylation in diabetes. In press. Diabete Metab. 1984.

Hypertension and Diabetes

Like many of my colleagues, I am delighted to finally see the new and long-awaited report by the Joint National Committee on the detection, evaluation, and treatment of high blood pressure, which included a review of earlier recommendations. (Arch. Intern. Med. 1984; 144:1045–57.) Yet, like a number of physicians, especially diabetologists, I am concerned and rather disappointed by the failure of this report to specifically address the issue of hypertension in the diabetic patient, unequivocally, and in a more forthcoming and detailed fashion.

Evidence is now mounting that the use of diuretics is associated with potential cardiovascular risks; namely, hypokalemia, resulting in serious cardiac arrhythmia; hypertriglyceridemia; hypercholesterolemia; hyperuricemia; and decreased HDL, all of which contribute to the process of atherosclerosis in diabetic patients who already have compromised vascular status, let alone impairment of diabetes control, associated with these agents. Indeed, our European colleagues have long abandoned the use of diuretics as first-step agents, especially in the hypertensive diabetic population. And, although it could be argued by some that the dose of diuretics should not be pushed to the maximum to avoid possible hypokalemia and that the potassium level could be monitored in these patients, many studies indicate that abnormal glucose metabolism may occur without changes in the potassium level.[1–3] Furthermore, it can be argued that a limited rise in lipids is sufficient to increase the risk of atherosclerosis and coronary heart disease.[3,4] This is a fact that has also been recently suported by the results of the LRC study.[5] Add to this the economic impact caused by frequent office visits and lab tests to monitor potassium level, lipids, glucose, and uric acid in a patient whose blood pressure may remain uncontrolled, requiring the use of additional antihypertensive agents.[6]

The MRFIT Study,[7] the Morgan Study,[8] and the Oslo Study[9] provide us with at least some indirect evidence that the use of diuretic therapy in the treatment of hypertension has not helped reduce coronary morbidity and mortality, perhaps due to the above complications, which offset or negate the benefits derived from lowering the blood pressure through the use of such agents. Similarly, the problem of beta blockers causing a suppression of insulin release, a masking of hypoglycemic reaction with a blunted response or recovery, vasoconstriction, hyperlipidemia, and a reduction of HDL[10–12] in a population with already compromised vascular and lipid status has not been mentioned or seriously emphasized in the report. The use of Clonidine in the diabetic hypertensive patient, with its possible consequences of suppressed insulin