Letters to the Editor

Hyperinsulinemia, Intracellular Calcium, and Membrane Function in Essential Hypertension

We read with interest comments by Dr. Sela et al dealing with a possible link between intracellular calcium content of polymorphonuclear leukocytes (PMN) and plasma insulin level in patients with essential hypertension. The results of their presented study demonstrated that PMN in essential hypertension showed increased levels of intracellular calcium correlating positively with the individual’s blood pressure and plasma insulin. Sela et al proposed that because PMN priming may lead to oxidative stress and inflammation, intracellular calcium and insulin are involved in the pathogenesis of hypertension-induced vascular injury.

Numerous studies have reported abnormalities in the intracellular calcium metabolism in blood cells (lymphocytes, erythrocytes, and platelets) in essential hypertension. However, their contribution to the pathophysiology of hypertension is not fully understood.

In a study we presented earlier, a relationship between membrane fluidity of erythrocytes and plasma insulin was investigated in patients with essential hypertension by means of electron paramagnetic resonance. The membrane fluidity of erythrocytes was significantly lower in patients with essential hypertension than in normotensive subjects. The plasma content of insulin while fasting was significantly greater in hypertensive patients than in normotensive subjects. In addition, it was demonstrated that the higher the plasma insulin level, the lower the membrane fluidity of erythrocytes, which might indicate that hyperinsulinemia is a determinant of membrane fluidity of erythrocytes in essential hypertension. Barbagallo et al reported that insulin significantly elevated the intracellular calcium level of human erythrocytes in a dose- and time-dependent manner. With regard to the interaction between calcium and membrane fluidity, it was observed that calcium strongly decreased membrane fluidity of erythrocytes and other cells. In an in vitro study we showed that insulin alone and in combination with calcium decreased membrane fluidity of erythrocytes in essential hypertension. It is likely that the insulin-evoked decrease in membrane fluidity of erythrocytes may be partially mediated by the increased intracellular calcium content. Because membrane fluidity is a reciprocal value of membrane microviscosity, the decreased membrane fluidity of erythrocytes might cause a disturbance in blood rheologic behavior and microcirculation, which could, at least in part, contribute to the pathophysiology of hypertension.

In this context, it can be speculated that insulin might accelerate abnormalities in intracellular calcium metabolism and membrane function in blood cells such as PMN and erythrocytes, which might partially explain the vascular complications in hypertensive subjects with hyperinsulinemia.

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References


Role of the Na+/Mg2+ Exchanger in Hypertensions

We read with interest the comments by Standley et al dealing with the identification of a functional sodium (Na+)/magnesium (Mg2+) exchanger in human trophoblast cells.

They concluded that the JEG-3 cells appear to possess...