Effects of Chronic Kidney Disease on Blood Cells Membrane Properties

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Chronic kidney disease (CKD) is a progressive loss of renal function over time associated with increased blood pressure, metabolic acidosis and fluid volume overload. It is divided into five stages of increasing severity. It was previously reported that CKD is also accompanied by increased intracellular concentration of calcium ions, whose origin has not been explained yet [1]. In this study, we attempted to identify the effects of the disease on some membrane properties such as human red blood cell Ca^{2+} ATPase activity, lymphocyte P2X7 receptors expression and function and red blood cell membrane fluidity, which could potentially help us in elucidating the origin of increased calcium concentration in blood cells. Using fluorescence spectroscopy we found out that Ca^{2+} ATPase activity is decreased in early-stage CKD patients compared to healthy volunteers thus resulting in altered calcium removal from cytoplasm. This is in compliance with the study by [2]. By means of flow cytometry we determined a twofold increase in expression of P2X7 receptor in lymphocyte membrane of CKD patients. Moreover, we determined an increased uptake of ethidium bromide through this receptor in CKD patients compared to healthy volunteers at basal conditions. It means that in CKD, lymphocyte membranes contain more receptors which are more permeable thus allowing an increased influx of calcium ions from extracellular milieu. As regards the last evaluated parameter, we didn't find any significant difference between fluorescence anisotropy from red blood cell membranes of early-stage CKD patients and healthy controls indicating that membrane fluidity is not affected by the disease. Finally, based on these results we can state that alterations in blood cells membranes are closely linked to CKD and are at least partially responsible for intracellular calcium accumulation in blood cells. We intend to further analyze whether the situation gets worse in end-stages of CKD.

References:

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[2] D.Polak-Jonkisz et al.; Nephrol Dial Transplant, 2010, 25, 438-444