CORRESPONDENCE

PROGRESSION OF VASCULAR CALCIFICATION AND DYSLIPIDEMIA IN PATIENTS ON CHRONIC HEMODIALYSIS

To the Editor:

We read with interest the article by Tamashiro et al[1] pointing out a significant association between the progression of coronary calcification with high triglyceride and low high-density lipoprotein (HDL)-cholesterol concentration in hemodialysis patients.

Indeed, this observation is reminiscent of our own observation regarding the extension of calcification assessed prospectively on 3 years in 24 hemodialysis patients who never received vitamin D derivatives. The calcifications were measured on lateral and frontal X rays of lumbar spine and pelvis at the level of aorta, iliac, and femoral arteries.[2] This extension was exponential, and simple covariance analysis showed that the main significant risk factors for extension were male gender, age (only in male patients), blood pressure, triglyceride, serum calcium, and blood glucose, whereas serum phosphate was only a borderline factor and total cholesterol, alkaline phosphatase, uric acid, and doses of $CaCO_3$ and/or $Al(OH)_3$ were not significantly associated. Multiple covariance analysis of calcification extension to combination of blood pressure with one of the 3 most significant metabolic risk factors showed that triglyceride level was the strongest in increasing the risk of extension comparatively to serum calcium and blood glucose.

Although HDL cholesterol was not measured in this early study, it is very likely that the direct link between vascular calcification and triglyceride level was also associated with an inverse link between these calcifications and HDL cholesterol because of the usual inverse correlation between these two parameters as shown in the study by Tamashiro et al.[1]

These observations by Tamashiro et al[1] and Renaud et al[2] are particularly relevant for the assessment of the sevelamer hydrochloride (Renagel; Geltex Pharmaceuticals, Waltham, MA) in the prevention of coronary artery calcification and cardiovascular morbimortality in comparison to $CaCO_3$, especially since Chertow et al[3] have shown that Renagel effectively decreased not only $SPO_4 \times SCa$ product and parathyroid hormone, but also total and low-density lipoprotein (LDL) cholesterol and increased HDL cholesterol. Indeed, to prove that the decreased calcium load with the use of noncalcic phosphatebinder is responsible for the possible lower coronary artery calcium score and also for the lower cardiovascular morbimortality comparatively to the $CaCO_3$ group, dyslipidemia in the $CaCO_3$ group should be as adequately treated as in the Renagel group.

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COMBINED USE OF ENALAPRIL AND LOSARTAN TO REDUCE PROTEINURIA: A QUESTION OF SAFETY

To the Editor:

Russo et al[1] recently presented evidence showing that administration of a combination of the angiotensin-converting enzyme (ACE) inhibitor enalapril (E) and the angiotensin receptor antagonist (ARA) losartan (L) for 4 weeks in 10 young normotensive individuals with IgA nephritis with nonnephrotic proteinuria and normal renal function was associated with a greater antiproteinuric benefit than with use of either agent alone. There was no significant change in mean serum creatinine or systemic blood pressure. Although adverse effects were not detailed, there was no significant hyperkalemia despite administration of relatively large doses of E plus L (10 mg plus 50 mg/day or 20 mg plus 100 mg/day), presumably administered as a single dose. These observations on efficacy may encourage use of such combi-