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Nephrology: Vascular calcification in patients with peritoneal dialysis- Augusto Octavio Salinas Meneses- Clinical ISSSTE Chilpancingo

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Cardiovascular mortality is the leading cause of death in patients with chronic kidney disease and is markedly higher than in the general population when stratified by age, race, and gender. However, a portion of cardiovascular disease (CVD) in the ESRD population cannot be explained by these and other traditional risk factors alone. Defects in mineral metabolism, particularly hyperphosphatemia and secondary hyperparathyroidism, increase the risk of CVD in patients on dialysis. The burden of vascular calcification in the dialysis population is reflected by the higher prevalence and severity of coronary artery calcification when compared to age-matched healthy controls. In addition to abnormalities in serum calcium and phosphorus, risk factors for the development for vascular calcifications also include increased age, longer duration of dialysis, inflammation, hypertension, dyslipidemia, and calcium-based phosphate binders. The process of vascular calcification is a complex process, and attributing the pathogenesis to the precipitation of calcium and phosphate in the walls of arteries oversimplifies the problem. Inflammation, uremia, hyperphosphatemia, hypertension, hyperlipidemia, and hypercalcemia, all could be expected to play a role in this process. Interestingly, some dialysis patients, despite the uremic environment and hyperphosphatemia, do not develop vascular calcifications. Reducing the risk of vascular calcifications has been aimed at controlling serum phosphorus, calcium, and PTH. As with calciphylaxis, in severe cases of coronary and peripheral vascular disease, parathyroidectomy may be indicated and can lower the rate of long-term mortality associated with secondary hyperparathyroidism. Before that, traditional control of serum phosphorus, however, may have the biggest impact in reducing the risk of progression of extraosseous calcification. Use of calcium-based phosphate binders and the nonabsorbable polimer sevelamer both have roles in controlling serum phosphorus; however, the calcium load imposed by high doses of calcium salts may increase the risk of vascular calcification.

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