

Acute nephrocalcinosis following oral sodium phosphate bowel cleansing

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Acute kidney injury; nephrocalcinosis; phosphate.

Dear Sir,

Recently in the Portuguese Journal of Nephrology and Hypertension, Y. Aznar *et al.*¹ described a very interesting case of acute renal failure, intratubular and interstitial calcium phosphate deposits associated with the use of an osmotic purgative (Fosfo-soda®). There is currently great interest in linking acute deposit of calcium phosphate in renal parenchyma with concomitant tubular injury to the use of phosphate-containing cathartics.

This entity is differentiated from typical nephrocalcinosis by deposits of calcium phosphate² occurring in the absence of increased serum calcium levels; development of acute renal failure following the use of phosphate-containing cathartics³ often resulting in acute and persistent loss of kidney function; the specific histological pattern; no evidence of chronic injury³; experimental evidence of kidney damage associated with phosphorus overload⁴ and that the dominant mechanism is acute tubular injury³.

Pathology findings are important in diagnosing this entity. There is a particular pattern of abundant deposit of calcium phosphate, principally in distal tubules and collecting ducts^{2,3}; important focal peritubular lymphocytic inflammation and minimal tubular damage, including occasional necrotic and sloughed epithelial cells, coarse vacuolisation, loss of brush borders, nuclear enlargement, nucleolar prominence, and occasional binucleation. These tubular degenerative changes are accompanied by

interstitial oedema^{3,5}. The tubular injury closely resembles acute tubular necrosis³. This is further demonstrated by the immunohistochemical findings of markers of acute kidney injury³.

The term “nephrocalcinosis” denotes a clinical-pathological entity characterised histologically by abundant renal parenchymal deposits of calcium phosphate associated with chronic tubulointerstitial injury³, and this is a term coined to describe the deposit of calcium crystals in the renal parenchyma resulting from long-lasting hypercalcaemia.

Finally, unlike the usual form of nephrocalcinosis, there is strong evidence that the renal lesions can be induced by a high phosphate load as the sole causative factor. The temporal relationship with an overload of phosphate and kidney damage and the specific histopathological findings could more suitably be described as “acute renal damage by phosphate” or “acute phosphate nephropathy”. This would describe this entity more aptly than “acute nephrocalcinosis”⁶.

Conflict of interest statement. None declared.

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