Acute phosphate nephropathy

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Introduction

Oral sodium phosphate (OSP) is a well known and generally well accepted bowel purgative (1). Recently it has, however, been identified as a course of acute and subsequently chronic renal failure (2, 3)

Case presentation (3)
77-year-old male was in January 2010 operated for colon adenocarcinoma. For many years he has been treated for hypertension with Beta-blockers, diuretics and alfa-blockers. In April 2010 he underwent colonoscopy which did not show any signs of recurrent disease. Prior to the colonoscopy, he was orally given sodium phosphate (OSP) bowel purgative. Two weeks later he was again admitted to hospital due to intestinal bleeding. At the time of admission his s-creatinine was 700 µmol/l and after rehydration 450 µmol/l. Proteinuria 1,1 g/d. No hyperphosphatemia og hypercalcaemia. A renal biopsy was done in May 2010.

Pathological diagnosis
Acute phosphate nephropathy (APhN) with calcium-phosphate containing deposits in tubules.

Discussion

APhN was first described in 2003 by Desmeules et al. (2). A 71-year-old woman developed acute renal failure after OSP. The renal biopsy showed nephrocalcinosis with multiple intratubular deposits containing calcium and phosphate. Systemic review and meta-analysis by Brunelli, however, could not definitively prove an association between receipt of oral sodium phosphate and kidney injury (4).

Markowitz et al. have proposed the following diagnostic criteria for APhN (5):

The pathological findings in the renal biopsy is acute and chronic tubulointerstitial nephropathy with calcium-phosphate deposits in tubules and interstitium. The deposits are mainly confined to the distal tubules and collecting ducts. They do react with von Kossa but do not polarize.

The normal dose of OSP contains an amount of phosphate that is 12 times the normal daily intake and leads to a doubling of s-phosphate. The diarrhea following the intake of OSP is associated with volume depletion and these two mechanisms leads to a decreased proximal tubular reabsorption and an increased concentration of calcium/phosphate in the distal tubules with subsequently deposition in the tubular lumen (5).

The clinical course of the disease may be mild with only slight renal failure or may be progressive renal failure with severe loss of function (5).

Referencer: