PP-135

Hypercalciuria in idiopathic calcium nephrolithiasis: An acid-base metabolic perspective

P.J.S. Osther, K.V. Pedersen, K.H. Andreassen, S.S. Osther

Urological Research Center, Department of Urology, Fredericia Hospital, Fredericia, Denmark

Hypercalciuria in renal stone formers is usually idiopathic in the sense that most patients are normocalcaemic without any obvious cause of the increased calcium excretion, and although addressed in several studies the exact aetiology of hypercalciuria in calcium nephrolithiasis remains debatable. The aim of this study was to evaluate the role of acid-base metabolism.

Material and Methods: 118 consecutive male recurrent idiopathic calcium stone formers and 122 healthy males matched for age and BMI were included in the study. 24-h urines were sampled without dietary restrictions. A subgroup of 12 stone formers with hypercalciuria defined as $Ca(U)_{24h}$<0.1 mmol/kg body mass and 10 healthy males were further evaluated by NH_4Cl-loading studies in a double blind randomized cross-over design. Serum ionized calcium and rates of renal calcium and non-metabolizable acid (NA) were measured hourly.

Results: The cumulative frequency distributions curve for 24-h urinary excretion of calcium clearly showed that the stone formers and the healthy controls presented as distinctly different populations with significantly higher excretion levels in the stone formers ($p<0.002$). Ionized calcium in serum increased significantly in both groups in response to acid loading compared to placebo ($p<0.001$), however, with significantly higher levels in the stone formers ($p<0.01$). During acid loading urinary calcium excretion increased markedly in both groups with significantly higher excretion rates in the stone formers ($p<0.01$). Also fractional excretion of calcium in urine was higher in the stone formers ($p<0.01$). Furthermore, the stone formers excreted significantly more calcium in the urine at a given rate of renal NA excretion.

Conclusion: As a group recurrent idiopathic calcium stone formers had higher urinary calcium excretion rates than matched healthy controls. With regard to calcium metabolism recurrent idiopathic calcium stone formers seem to be more sensitive to an acid load than healthy subjects. The most obvious explanation is that the stone formers experienced a higher degree of bone buffering during acid loading. This mechanism may explain why calcium stone formers frequently have moderate bone mineralization defects, and it may be a significant pathophysiological factor explaining why these individuals form calcium containing renal stones.

As published in the *Supplement of AFJU, Volume 18 (2012), 1st ESD "Experts in Stone Disease" Conference* (page 88)