ABSTRACT

Corticosteroids are commonly used drugs due to antiinflammatory, antiallergic and immunesupressive effects. They are the main drugs in treatment of nephrotic syndrome and bronchial hyperreactivity and can be used long periods in high doses. From clinical and experimental experiments, systemic corticosteroids are known to cause hypercalciruria by inhibition of tubular calcium reabsorption and increasing bone resorption. As inhaled corticosteroids absorbed via the lung and gastrointestinal system, like systemic corticosteroids can cause systemic side effects such as hypercalciuria. Recently, many researches are reported about the hypercalciuric effect of inhaled corticosteroids.

In childhood, hypercalciuria is important because it can cause hematüria, recurrent urinary tract infections, bone system influences and urinary stone disease. Therefore, in this study we aimed to show the excretion of calcium in the urine, mechanism of corticosteroids and the differences between children using high dose oral prednisolon (for nephrotic syndrome) and long term inhaled budesonide (for bronchial hyperreactivity).

The cases included to the study divided into three groups as group 1 nephrotic syndrome; group 2 bronchial hyperreactivity; group 3 healthy controls. All the cases are searched for urinary calcium excretion (spot urinary Ca/Cr ratio), bone formation markers (ALP, osteocalcin), bone resorption markers (urinary deoxypyridinoline cross link/Cr), bone metabolism markers (Ca, P, PTH), urinary prostaglandine/Cr ratio and urinary NAG/Cr ratio as a tubulopathy marker. In group 1, we detected significant increase in urinary Ca/Cr after oral prednisolone treatment. While, in group 2 there wasn't any significant change in urinary Ca/Cr after inhaled budesonide treatment. This showed us the hypercalciuric effect of oral corticosteroids is not observed in inhaled corticosteroids. In nephrotic syndrome, after corticosteroid treatment urinary NAG/Cr ratio increased, while ALP and urinary deoxypyridinoline cross link/Cr ratio decreased after oral prednisolone treatment. In bronchial hyperreactivity cases deoxypyridinoline cross link/Cr ratio significantly decreased after inhaled budesonide treatment while other parameters didn't change. In conclusion, in this study with these results, we found that oral corticosteroids increase urinary calcium excretion and we couldn't show the mechanism of urinary calcium excretion. In the future, different more studies are needed to determine the effect and mechanism of corticosteroids on urinary calcium excretion.

Key words: Corticosteroid, hypercalciuria, nephrotic syndrome, bronchial hyperreactivity