EFFECT OF ASPIRIN AND INDOMETHACIN ON THE SERUM AND URINARY CALCIUM, MAGNESIUM AND PHOSPHATE A COMPARATIVE STUDY

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Recent data have shown that administration of prostaglandins inhibitors to patients with hypercalciuria nephrolithiasis decreased urinary calcium excretion, implying a possible role for postaglandins in calcium excretion. To explore this hypothesis, we investigated the effect of single dose or 7 days administrations of aspirin (100 mg/kg orally) or indomethacin (20 mg/kg, orally) on the urinary and serum concentrations of calcium, magnesium and inorganic phosphate. Experiments were performed in normocalcemic and hypercalcemic rats. Hypercalcemia and hypercalciuria were induced in male wistar albino by administration of vitamin D_3 (20,000 IU/daily) for 7 days.

Aspirin and indomethacin each, lowered significantly the urinary calcium excretion in normocalciuric and hypercalciuric rats. The acute administration of indomethacin caused greater reduction of calcium excretion than that produced by the acute administration of aspirin, wheares aspirin showed greater activity than indomethacin after the long-term use of each. Aspirin-induced hypocalcemia in normocalcemic rats and abolished the hypercalcaemia in hypercalcemic rats. By contrast, indomethacin, a specific prostaglandin biosynthesis inhibitor, increased levels of calcium.

Hypophosphatemia was observed only after the administration of single dose of aspirin in normocalcemic rats, while the reduction of urinary phosphate excretion was investigated in hypercalciuric rats after the acute and chronic administration of indomethacin. Single dose of indomethacin significantly reduced urinary excretion of magnesium in both groups of rats. However, the acute and chronic administration of aspirin resulted in non significant changes in serum and urinary concentrations of magnesium.

These data suggest that aspirin has hypocalcemic and hypocalciuric actions while indomethacin has only hypocalciuric effect. Aspirin may produce these actions by two mechanisms, one of them simulate indomethacin which is dependent on the inhibition of biosynthesis of prostaglandins, and another possible mechanism that is not related to the inhibition of prostaglandin biosynthesis, perhaps, through inhibition of bone resorption. This suggestion may be supported by the discrepancy between the effects of aspirin and indomethacin on the renal handling and serum concentrations of magnesium and inorganic phosphate.

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