High dickkopf-1 levels in sera and leukocytes from children with 21-hydroxylase deficiency on chronic glucocorticoid treatment

Children with 21-hydroxylase deficiency (21-OHD) need chronic glucocorticoid (cGC) therapy to replace congenital deficit of cortisol synthesis, and this therapy is the most frequent and severe form of drug-induced osteoporosis. Therefore, 21-OHD patients are at risk of a great incidence of low bone mass. In this study, we enrolled 18 patients (9 females) and 18 sex- and age-matched controls and we evaluated the serum and leukocyte levels of dickkopf-1 (DKK1), a secreted antagonist of the Wnt/β-catenin signaling pathway known to be a key regulator of bone mass. We also studied the effects of dexamethasone on the expression of DKK1 in human leukocytes from controls in vitro. Finally, we examined the effects of the conditioned media by the serum of the patients on osteoblast (OB) differentiation and RANKL expression.

**INTRODUCTION**

**CONCLUSIONS**

Our data indicated that DKK1, produced by leukocytes, may contribute to the alteration of bone remodeling in 21-OHD patients on cGC treatment.