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**Evaluation of the OPG gene polymorphisms and serum levels of osteoprotegerin in Charcot artropathy: A pilot study**

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Genes of the OPG/RANKL/RANK axis have been identified as candidate mediators in bone metabolism. They regulate osteoclastogenesis and may play a role in vascular calcification. Osteoprotegerin (OPG) is a member of the tumor necrosis factor (TNF) receptor family, it is a potent inhibitor of osteoclast activation and differentiation, and plays an important role in bone remodeling. Recently, polymorphisms in the *OPG* gene have been associated with various bone phenotypes, cardiovascular diseases and osteoporosis. Increased OPG levels were also observed in patients with diabetes and in subjects with diabetes and microangiopathy. The Charcot syndrome is a rare complication of neuropathy in diabetes and is characterized by an acute inflammatory episode of the foot that is associated with variable degrees of dislocation, fracture, and deformity. Charcot arthropathy is characterized by the presence of osteopenia and vascular calcification. The association of the active phase of the disease with inflammation, increasing osteopenia, and increasing calcification of the arterial walls strongly suggests the involvement of the receptor of activator of nuclear factor-kappaB ligand (RANKL)/osteoprotegerin (OPG) cytokine pathway, which is closely involved in all three processes. One hypothesis is that an imbalance between RANKL and OPG leads to increased osteoclastic activity in acute Charcot osteoarthropathy and that the RANKL/OPG ratio can act as a novel marker to monitor disease activity in the Charcot foot. The aim of our work was to analyze polymorphisms in the OPG gene and to investigate their possible contribution to the genetic susceptibility to Charcot arthropathy, as well as to investigate the association between *OPG* polymorphisms and neuroartropathy changes. In the present study, we evaluated the impact of sequence variations in the *OPG* gene on bone mass and bone-related biochemistry, including serum OPG. A total of 131 patients (95 controls, 30 with artropathy, 6 diabetic patients without Charcot foot) were genotyped for 5 different single nucleotide polymorphisms (SNP) within the *OPG* gene (G1181C, T950C, T245G, C1217T and A6890C). Patients were genotyped for OPG SNPs by PCR/RFLP method. Serum OPG levels were detected by enzyme-linked immunosorbent assay. The effects of these SNPs and serum OPG on Charcot artropathy were examined. We found no significant relationship between sequence variations in the *OPG* gene or serum OPG levels and diabetes with or without Charcot artropathy. Further studies, based on more samples, are needed to delineate any relationship between artropathy, polymorphisms of bone metabolism genes and serum OPG levels.