Increased osteoclastic activity in acute Charcot’s osteoarthopathy: the role of receptor activator of nuclear factor-kappaB ligand

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Mots-clés: Charcot's osteoarthropathy [5], Human Physiology [6], Internal Medicine [7], Metabolic diseases [8], OPG [9], Osteoclasts [10], osteolysis [11], RANKL [12], Resorption [13]
Aims/hypothesis Our aims were to compare osteoclastic activity between patients with acute Charcot’s osteoarthropathy and diabetic and healthy controls, and to determine the effect of the receptor activator of nuclear factor-kappaB ligand (RANKL) and its decoy receptor osteoprotegerin (OPG). Methods Peripheral blood monocytes isolated from nine diabetic Charcot patients, eight diabetic control and eight healthy control participants were cultured in the presence of macrophage-colony stimulating factor (M-CSF) alone, M-CSF and RANKL, and also M-CSF and RANKL with excess concentrations of OPG. Osteoclast formation was assessed by expression of tartrate-resistant acid phosphatase on glass coverslips and resorption on dentine slices. Results In cultures with M-CSF, there was a significant increase in osteoclast formation in Charcot patients compared with healthy and diabetic control participants (p = 0.008). A significant increase in bone resorption was also seen in the former, compared with healthy and diabetic control participants (p < 0.0001). The addition of RANKL to the cultures with M-CSF led to marked increase in osteoclastic resorption in Charcot (from 0.264 ± 0.06% to 41.6 ± 8.1%, p < 0.0001) and diabetic control (0.000 ± 0.00% to 14.2 ± 16.5%, p < 0.0001) patients, and also in healthy control participants (0.004 ± 0.01% to 10.5 ± 1.9%, p < 0.0001). Although the addition of OPG to cultures with M-CSF and RANKL led to a marked reduction of resorption in Charcot patients (41.6 ± 8.1% to 5.9 ± 2.4%, p = 0.001), this suppression was not as complete as in diabetic control patients (14.2 ± 16.5% to 0.45 ± 0.31%, p = 0.001) and in healthy control participants (from 10.5 ± 1.9% to 0.00 ± 0.00%, p < 0.0001). Conclusions/interpretation These results indicate that RANKL-mediated osteoclastic resorption occurs in acute Charcot’s osteoarthropathy. However, the incomplete inhibition of RANKL after addition of OPG also suggests the existence of a RANKL-independent pathway.

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