

## OP5

### The prevalence of calcification of the pedal arteries in patients with disease of the foot in diabetes

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The mechanisms underlying the increased prevalence of medial calcification in diabetes are not understood. An association with distal symmetrical neuropathy has been reported in a number of small studies, and a particularly high prevalence was reported in two series of patients with Charcot's disease. **The aim** of this study was to attempt to confirm the high prevalence of calcification in Charcot's disease and to determine whether it is specific to that disorder by comparing the results with patients other types of foot disease. The prevalence of calcification was determined retrospectively in three groups of patients managed by a specialist service for the diabetic foot. Group A comprised those with an acute Charcot foot who were managed between 2002 and 2005, Group B comprised all those managed in the foot service who had had been diagnosed as having osteomyelitis between 2002 and 2004 and Group C those who had had X-rays taken between 2002 and 2004 who had neither osteomyelitis nor Charcot's disease. All X rays were scored independently examined by three observers. All three observers were blinded as far as possible to the underlying diagnosis, with films from the three groups being mixed. There were 34 patients in Group A (Charcot), 53 in Group B (osteomyelitis) and 35 in Group C. There were no differences ( $p>0.05$ ) in the mean age of the patients in the three groups (60, 72 and 68 years, respectively), the proportion of men (68%, 64% and 51%) and the prevalence of nephropathy (41%, 30% and 14%). 100% patients in Group A, 94% in Group B and 80% of Group C had clinical evidence of neuropathy. The overall prevalence of arterial calcification in the three groups was 53%, 66% and 54% ( $p>0.05$ ). When all three groups were combined, the only factor associated with calcification on logistic regression analysis was disease duration ( $p=0.004$ ). The prevalence of arterial calcification in patients with foot disease was higher than the 40% previously reported in patients with neuropathy, but lower than that reported in other series of Charcot. As there was no difference in the prevalence of calcification between the three groups, it is concluded that the increase is not specific to Charcot's disease. It is possible that the increase in calcification in each of the three groups reflects the effect of local inflammation, possibly by activation of the RANKL/OPG signalling system.