

**Analysis of risk factors in diabetic neurophatic Osteoarthropathy (DNOA)**

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**Introduction:** The main cause of the development of DNOA is neuropathy, but other risk factors such as body mass index (BMI) and poor metabolic control have been investigated. Recently some theories have also provided some relation to medial arterial calcification (MAC). It is therefore important to determinate the role of these variables defined as potential risk factors and the association or not with the development of this complication. **Aims:** to determine whether these risk factors in a cohort of patients with diabetes and neuropathy, can influence the development of DNOA.

**Methods:** A cases-controls study involving 116 patients with Diabetes mellitus (DM) and neurophaty, 39 (33,6%) with DNOA, and 77 (66,4%) without DNOA. Mean age of patients with DNOA was  $60,77 \pm 9,07$  and  $63,71 \pm 11,96$  in patients without DNOA ( $p=0,036$ ). In patients with DNOA 59 (76,62%) were male and 18 (23,37%) female, and in patients without DNOA, 29 (74,35%) were male and 10 (25,64%) female ( $p=0,788$ ). 7 patients (17,94%) had type 1 DM, and 32 (82,05%) DM type 2 in group of DNOA, and 8 patients (10,38%) type 1 DM and 69 (89,61%) type 2 in patients without DNOA ( $p=0,252$ ). Diabetes suffered time was  $17,69 \pm 13,12$  years in patients with DNOA and  $16,13 \pm 10,55$  in without DNOA ( $p=0,113$ ). In all patients the following variables defined as risk factors were collected and compared between groups: elevated BMI defined as overweight or obese, specifically  $25.0 \text{ kg/m}^2$  or greater, MAC in XR, defined as presence of radiopaque image of the anatomical course of any of the arteries in the foot, and bad metabolic control defined as a HbA1c > 7%.

**Results:** MAC was present in 14 patients (35,90 %) with DNOA and in 26 patients (33,76%) without DNOA ( $p=0,820$ ). BMI median was  $27,25 \pm 4,27 \text{ kg/m}$  in patients with DNOA and  $28,30 \pm 4,11 \text{ kg/m}$  in patients without DNOA ( $p=0,775$ ). Hb1Ac median was  $7,57 \pm 1,86 \text{ mg/dl}$  patients with DNOA and  $7,37 \pm 1,88 \text{ mg/dl}$  in patients without this complication ( $p=0,930$ ).

**Conclusions:** Although it was expected that different clinical variables would be related to an increased risk of DNOA, no such relationship was observed. The presumably complex process by which the DNOA develops, coupled with the limitations of the study design could have contributed to the failure to observe the predicted relationship. Additional research is needed to fully explore the nature of the relationship between this clinical variables and DNOA.