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MUSCULOSKELETICAL ASSUMPTION OF HANDS AND HANDLES IN TYPE 2 DIABETES: CASE REPORT.

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BACKGROUND

Diabetes mellitus (DM) is a highly prevalent chronic metabolic disease responsible for public health expenditures due to systemic complications. Among them, musculoskeletal disorders should be highlighted. More than 30% of patients with DM have physical disability due to hand and shoulder disorders. An example of this is Quervain's tenosynovitis which corresponds to the inflammatory process of the first dorsal extensor compartment of the wrist that contains the tendons of the short extensor of the thumb and the long abductor of the thumb. In addition, musculoskeletal impairment of DM also involves compression of the median nerve resulting in the typical clinical picture of carpal tunnel syndrome.

CASE REPORT

A 55-year-old male patient, two years ago, reported constant pain in the radial region of both wrists. In addition, it refers to paresthesia in the thumb and index finger of the right hand associated with the loss of strength with prejudice of habitual activities. She has been following difficult-to-control diabetes mellitus for 20 years using glycerol and metformin. Physical examination: on inspection, right hand atrophy, pain on active wrist hyperflexion (positive Phallen maneuver), pain on right wrist percussion (positive Tinel sign), pain on passive ulnar deviation of right and left wrists (Finkenstein's test), impaired grip and grip forces in the right hand. Complementary examinations were requested: Doppler ultrasonography of the hands and wrists (right and left): thickening and hypoechogenicity of the tendons of the long abductor and short extensor of the thumb of both hands; right hand showing median nerve thickening with a cross-sectional area of 0.14 cm2 (normal is 0.10 cm2); mild synovitis. The therapeutic approach of choice for de Quervain tenosynovitis and carpal tunnel syndrome was conservative. Patient is currently in physiotherapy sessions, use of right hand orthosis while sleeping, being prescribed anticonvulsant for analgesia.

CONCLUSION

The pathophysiological mechanism of this type of DM complication is not fully understood. The products derived from non-enzymatic glycosylation are believed to bind to receptors in the chondrocytes, increasing the activity of metalloproteinases, thus degradation of connective tissue occurs. The recognition of the association of DM with these diseases is essential for the institution of appropriate therapy, preventing an increase in morbidity and mortality.