Carpal Arthritis as the Initial Manifestation of Gitelman’s Syndrome

Arthritis de carpos como debut en un síndrome de Gitelman

To the Editor:

Gitelman’s syndrome is a renal tubular defect and an autosomal recessive disease with metabolic alkalosis, hypokalemia, increased aldosterone and plasma renin with blood pressure in normal range. It maintains a close similarity with Bartter syndrome but unlike the latter hypomagnesemia and hypocaliuria are also persistent. The fundamental change lies in the Na⁺/Cl⁻ cotransporter sensitive to thiazides in the distal tubule.1,2

We report the case of a 43-year-old male with no personal or family history, referred to the outpatient clinics from the hospital emergency room after presenting an acute episode of arthritis of both wrists treated for a few days with low dose corticosteroids. The patient had no previous injuries or infectious symptomatology.

The study of rheumatoid factor and autoantibodies (ANA, ENA, anti-CCP) and the serology for human immunodeficiency virus and hepatitis B and C were negative. The acute phase reactants were in the normal range. Radiological examination showed mild degenerative signs of the right third metacarphalangeal joint with no detectable calcification in the carpal triangular ligament, the symphysis pubis or knees. Incidental findings were a low serum potassium (2.1 mEq/l) and magnesium (1.4 mg/dl).

After study for an electrolyte disorder by the nephrology department, he was diagnosed with Gitelman’s syndrome. He was prescribed chronic oral supplements of potassium and magnesium and potassium-sparing diuretics, spironolactone initially, which had to be replaced by epleronone after developing gynecomastia.

It is well known that magnesium is a cofactor of many pyrophosphatases and plasma levels have been associated with calcium pyrophosphate crystal arthropathy. In cases of hypomagnesemia, it alters the solubility of calcium pyrophosphate leading to precipitation of crystals at the joint level and producing pseudogout.3

In Gitelman’s syndrome there is an increased elimination of magnesium via the kidneys which is not easy to correct. This is because high oral doses usually result in episodes of diarrhea that favor its loss via the gut. In addition, higher intake also correlate with greater urinary losses.3

Our patient had several episodes of arthritis of the wrists despite optimal medical therapy, usually coinciding with periods when plasma magnesium levels were lower. This, together with the absence of other causes explaining the presence of self-limited arthritis, suggests that we find ourselves with a rare form of calcium pyrophosphate crystal arthritis. Although we must emphasize that in this case the diagnosis of the disease is not definitive, we did not analyze the joint fluid for crystals and have not found characteristic4 radiological signs.

References


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