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LETTER TO THE EDITOR

SPASTICITY WITH HYPOCALCAEMIA: DOES SPASTICITY HAVE A METABOLIC DETERMINANT?

Spasticity is a common chronic complication of spinal cord injury (SCI). It is characterized by hypertonicity, increased deep tendon reflexes, clonus and painful spasms (1). More than 80% of patients with SCI develop spasticity (2). Stimuli such as heat, cold, bladder distension and other miscellaneous factors can trigger spasticity (3), but increased spasticity due to hypocalcaemia has not been described previously.

We report here the case of a 44-year-old woman who sustained a SCI due to failure of back surgery for an extruded disc 3 years previously. She developed painful spasms in her legs, that had increased in severity recently, and which did not respond to treatment with oral baclofen, 80 mg, or tizanidine, 12 mg. Detailed questioning about factors that exacerbate spasticity found that they were non-contributory. Her medical history included a total thyroidectomy 12 years previously. Musculoskeletal examination revealed severe spasticity of the lower extremities, with a Modified Ashworth Scale score of 3, especially in the hip adductors and hamstrings. Neurological examination was compatible with American Spinal Injury Association (ASIA) impairment scale L1 ASIA B. Her deep tendon reflexes were overactive and her toes were upgoing. Her upper limbs were normal on neuromuscular examination. Laboratory tests revealed: serum total calcium (Ca) 5.9 mg/dl (normal range 8.2–10.6 mg/dl); ionized calcium (iCa) 3 mg/dl (normal range 4.7–5.3 mg/dl); parathormone (PTH) 34 pg/ml (normal range 15–68.3 pg/ml); 25-dihydroxyvitamin D, 11.3 ng/ml (normal >30 ng/ml); inorganic phosphate (P) 4.7 mg/dl (normal range 2.5–4.5 mg/dl). Oral 3,000 mg calcium and 0.5 µg calcitriol daily and 50,000 IU cholecalciferol weekly was commenced, with a likely diagnosis of hypocalcaemic muscular spasms due to hypoparathyroidism. Over the following 2 weeks her Ca increased to 8.5 mg/dl and her complaints resolved, with a significant decrease in spasticity.

Up to 60–80% of individuals with SCI have spasticity, and the ratio of problematic spasticity, defined as spasticity that restricts activities of daily living, causes pain, or both, is defined as 40% (2–4). The ASIA impairment scale and the anatomical level of injury together predict the possibility of development of spasticity, with maximum spasticity in the cervical ASIA impairment scale A group (4). Common factors that induce spasticity include: urinary tract infections, constipation, pressure ulcers, ingrown toe nails, skin infections and ill-fitting orthotics. Other less common factors, such as urinary tract calculi or post-traumatic syringomyelia, should also be considered (5). Hypocalcaemia has not previously been reported as a factor that exacerbates spasticity in patients with SCI. To the best of our knowledge this is the first published case in which hypocalcaemic muscle spasms have contributed to spasticity in a patient with SCI.

Serum calcium concentrations are mainly regulated by parathormone (PTH) and vitamin D levels (6). Therefore, as a metabolic response, PTH should be elevated in hypocalcaemia. A low or low-normal level of PTH suggests hypocalcaemic hypoparathyroidism. The most common cause of hypoparathyroidism is iatrogenic, during thyroid, parathyroid or radical neck surgery. It is usually transient, but in rare cases it may continue for longer than months and years, becoming permanent (7–8).

Hypocalcaemic symptoms, such as muscle cramps, tetany, twitching and paresthesias (8), can easily be masked in a patient with SCI, complicating the clinical manifestation. Clinicians should be alert to this rare scenario, in which care and vigilance are required. In the rehabilitation of patients with SCI, especially those with persistent spasticity despite proper treatment, hypocalcaemia should always be ruled out.

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