

Acquired Brown's Syndrome in a Patient With Psoriatic Arthritis[☆]

Síndrome de Brown adquirido en una paciente con artritis psoriásica

Dear Editor,

Brown's syndrome (SB) is an ocular motility disorder characterized by the inability to fully elevate the affected eye in complete adduction. It can be congenital or acquired, the latter being caused by a mechanical restriction of the movement of the superior oblique muscle tendon as it passes through the trochlea.¹ It has been described as a rare ocular complication in various rheumatic diseases, including rheumatoid arthritis,² JIA,³ adult Still's disease,⁴ lupus erythematosus,⁵ Sjögren's syndrome,⁶ poststreptococcal reactive arthritis⁷ and arthritis associated with Crohn's disease.⁸ Patients with psoriasis and psoriatic arthritis (PsA) frequently develop inflammatory processes, including conjunctivitis, uveitis and keratoconjunctivitis sicca,⁹ but so far there is only one described case of associated PsA and BS.¹⁰

A 24-year-old woman, with personal and family history of psoriasis, was diagnosed in November 2006 with PsA based on asymmetric oligoarthritis, dactylitis, and thoracic and lumbar inflammatory pain presenting a year later. She had elevated acute phase reactants (C reactive protein 21 mg/l, erythrocyte sedimentation rate 35 mm/h), but rheumatoid factor, anti-CCP antibodies and antinuclear antibodies were negative. The HLA-B27 I and Cw*06 were negative. Lumbosacral spine X-rays showed no signs of sacroiliitis and spinal involvement, and changes in the hands and feet were not significant. She was treated with indomethacin 150 mg/day, methotrexate 15 mg/week, sulfasalazine 2 g/day and prednisone 5 mg/day, yet maintained a significant inflammatory activity.

In November 2007 she came to the emergency room presenting diplopia and pain triggered by right eye movement. There was limitation in raising her right eye in adduction, with elevation preserved in abduction and diplopia on upper left version. The motility of the left eye was normal. The fundus examination and magnetic resonance imaging (MRI) showed no orbital abnormalities. She was diagnosed with acquired BS and treatment intensified (prednisone 10 mg/day, methotrexate 20 mg/week), with no improvement. In January 2008 there was an infiltration in the right superior oblique trochlea, with complete recovery within 2 weeks. Because of persistent activity of arthritis she later began treatment with adalimumab, achieving clinical remission within a few months. There has been no recurrence of BS during a follow up period of 55 months.

It has been suggested that the pathophysiology of rheumatic diseases associated to BS is a superior oblique impingement secondary to stenosing tenosynovitis, similar to trigger finger.^{6,7} Its appearance usually coincides with periods of high activity of the underlying disease,^{2,7,8} but has also been described in patients undergoing remission.^{5,10} The diagnosis is mainly clinical and imaging tests such as MRI may show inflammation of the superior oblique tendon or trochlea. However, this finding is not always present.⁶ The differential diagnosis includes isolated paralysis of the iv pair, traumatic injury, sinusitis and trochlear metastasis.^{6,8} The course of acquired BS is usually self-limiting, but in persistent cases the efficacy of corticosteroid treatment, both systemic and local, has been demonstrated.⁶ In conclusion, although BS is a rare disorder, it should be considered in patients with rheumatic diseases who develop diplopia or ocular dismotility.

References

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