Correspondence

Type 1 autoimmune hepatitis revealed by a dysphonia related to cricoarytenoid arthritis

Sir,

Autoimmune hepatitis disease may be accompanied, or revealed by, extra-hepatic (particularly rheumatological) manifestations, in approximately 5% of patients. These manifestations include arthralgia, symmetrical non-erosive, non-deforming polyarthritis, commonly localized on large articulations (knees, elbows, ankles and wrists), and myalgia. We report a case of type 1 autoimmune hepatitis revealed by a dysphonia related to cricoarytenoid arthritis, which has not previously been described to our knowledge.

A 45-year-old woman presented with a sudden dysphonia accompanied by dysesthesiae of the upper right limb. Fifteen days earlier, she had been seen for a 38 °C fever, oligoarthralgia, inflammatory syndrome (CRP > 100 mg/l, VS > 100 mm) and an acute cytolysis (ASAT and ALAT 4–5× normal), which regressed spontaneously.

General clinical examination was normal, except that examination of the upper respiratory tract revealed a reduced mobility of the vocal cords related to probable cricoarytenoid arthritis. Standard laboratory tests showed evidence of inflammation (CRP 50 mg/l, VS 80 mm), and only mild acute cytolysis (ASAT and ALAT 1.5–2× normal), which regressed spontaneously.

Serological tests for hepatitis A, B and C viruses, Epstein-Barr virus, cytomegalovirus, human immunodeficiency virus (HIV), B19 Parvovirus, Coxiella burnetti, Bartonella sp., Rickettsia sp., Borrelia burgdorferi and Francisella tularensis were negative, as were tests for tuberculous infection. High titres of anti-smooth muscle (anti-actin) antibodies (1:200 [normal 1:20], by enzyme-linked immunoassay) were detected, suggesting type 1 autoimmune hepatitis. Anti-DNA, anti-LKM1, anti-hepatic cytosol, anti-mitochondrial antibodies, rheumatoid factor and angiotensin-converting enzyme were absent. Only an oligoclonal profile with a monoclonal IgG lambda was detected.

Hepatic biopsy confirmed the diagnosis of early type 1 autoimmune hepatitis. Electromyograms of the limb and of the vocal cords were normal, as was the cerebral RMN and analysis of the cerebral fluid.

Thus, a diagnosis of dysphonia arising from cricoarytenoid arthritis, related to in type 1 autoimmune hepatitis, was made. A short period of corticosteroid therapy (prednisolone 40 mg/day for 7 days, then 20 mg/day for 7 days) with orthophonic rehabilitation produced regression of the dysphonia and normalization of the biological parameters. No relapse was seen one year later, when serum anti-smooth muscles antibodies remained at 1:100.

We believe this to be the first reported case of type 1 autoimmune hepatitis revealed by cricoarytenoid arthritis. To our knowledge, only few systemic diseases have been described with cricoarytenoid arthritis: rheumatoid polyarthritis, acute systemic lupus erythematosus and ankylosing spondylitis. In our case, the diagnosis of type 1 autoimmune hepatitis was well established by high titres of anti-smooth muscle and histological data (hepatic biopsy). As the usual aetiologies of cricoarytenoid arthritis were not found, this manifestation was probably related to the type 1 autoimmune hepatitis, by temporal arguments. We therefore propose that dysphonia related to cricoarytenoid arthritis be considered as a possible cause of autoimmune hepatitis.

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References
