

Table 1
Classification of Spondyloarthritis With the Introduction of the Concept We Refer to as Radiographic axSpA That Is not AS.

Axial SpA	Peripheral SpA
AS (+ radiographies) nr axSpA (MRI+ radiography–) Radiographic axSpA not AS (radiography+ not AS, MRI–)	Reactive arthritis Psoriatic arthritis Arthritis associated with CIBD
	Undifferentiated SpA

AS, ankylosing spondylitis; CIBD, chronic inflammatory bowel disease; MRI, magnetic resonance imaging; nr axSpA, non-radiographic (or pre-radiographic) axial spondyloarthritis; radiographic axSpA no AS, radiographic axial spondyloarthritis no ankylosing spondylitis; SpA, spondyloarthritis;

those of Rome (1961),³ New York (1966)⁴ and their modification (1984).⁵ Their major drawback is the limited sensitivity in the initial stages, as they require the radiographic diagnosis of sacroiliitis. To make up for these limitations, in the nineties we had the publication of the criteria of Amor⁶ and those of the European Spondyloarthropathy Study Group (ESSG),⁷ which included radiographic sacroiliitis but not as a necessary requirement, extending the diagnostic spectrum to the group of undifferentiated spondyloarthritis. Recently, the criteria of the Assessment of Spondyloarthritis International Society (ASAS) have enabled us to improve its early diagnosis, classifying SpA into 2 groups. One group is comprised of predominantly axial SpA (2009),⁸ which includes classical AS (radiographic sacroiliitis, meeting the modified New York criteria) and pre-radiographic or non-radiographic axial SpA (with the support of sacroiliac magnetic resonance imaging). The other consists of predominantly peripheral SpA (2011),⁹ including psoriatic and reactive arthritis and arthritis associated with inflammatory bowel disease and undifferentiated SpA.

Nevertheless, there are patients who cannot be classified using the new ASAS criteria, although their clinical data suggest SpA, as occurs in the following clinical setting: a 40-year-old man with a 1-year history of inflammatory low back pain, HLA B27-negative, normal levels of acute-phase reactants, good response to nonsteroidal anti-inflammatory drugs (NSAID) and 2 earlier episodes of uveitis. First-degree family history of psoriasis. Radiographs showing bilateral sacroiliitis (left, grade 2, and right, grade 1), with no MRI evidence of edema/osteitis in sacroiliac joints.

Within the group of non-radiographic “or pre-radiographic” axial spondyloarthritides (nr axSpA)¹⁰ includes those patients with a normal radiography or initial sacroiliitis, that does not meet the modified New York criteria (no AS), have MRI-evidence of edema in sacroiliac joints; however, what would occur if we found patients without edema in MRI and without radiographic damage in the sacroiliac joints? How would they be classified?

Introducing certain small changes into the nomenclature, we could speak of non-radiographic (or pre-radiographic) axial SpA

in those cases in which MRI is positive for sacroiliac involvement, according to ASAS criteria, with normal radiographies or minimal changes in these joints, but do not meet the modified New York criteria. However, on the other hand, radiographic axSpA that is not AS, patients in whom there are initial radiographic changes in the sacroiliac joints (without meeting the modified New York criteria) and whose MRI is negative (Table 1).

In any case, studies in which the sensitivity and specificity of these modifications is evaluated are necessary. They could make it easier to achieve a better classification of those patients with incipient radiographic changes during the initial stages who have a negative MRI study.

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Parsonage–Turner Syndrome: A Case Report[☆]



Síndrome de Parsonage–Turner: a propósito de un caso

To the Editor,

Parsonage–Turner syndrome (PTS) is a neuritis of the idiopathic brachial plexus.¹ We report the case of a patient affected by this disorder, which had no triggering factor.

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The patient was 67-year-old man with type 2 diabetes mellitus, with good metabolic control and obstructive sleep apnea hypopnea syndrome. He had been diagnosed with a lung squamous cell carcinoma, stage pT₁apN₀, in March 2015. He was treated with left upper lobectomy and lymphadenectomy. He was admitted to our hospital in November after several days of severe pain in the right scapular region, followed by paresis involving extension of his 4th finger, with no history of traumatic injury or infection. Physical examination revealed weakness in dorsal interossei and in all the extensor carpi, with slight amyotrophy. He underwent cervicothoracic computed tomography, which ruled out lung apex disease, as well as cervical magnetic resonance imaging (MRI), which revealed

spondyloarthritis without myelopathy, and MRI of the brachial plexus, which showed no changes. An electromyogram performed 2 weeks after symptom onset indicated right brachial plexopathy with inferior predominance (C8-T1). Cerebrospinal fluid was normal. Suspecting PTS, we began treatment with analgesics, glucocorticoids and rehabilitation, which achieved resolution of the pain and nearly complete recovery of the movement of the 4th finger 2 months later.

Parsonage–Turner syndrome (amyotrophic neuralgia) is an acute neuritis of the brachial plexus characterized by shoulder pain, followed by a motor deficit and muscle atrophy, generally in the shoulder and the area of the elbow.¹ There are idiopathic and hereditary forms.² In the idiopathic form, 50% of the patients are exposed to a previous event (infection, surgery, systemic disease or vaccination), which would activate lymphocytes sensitized to the brachial plexus in individuals with a genetic predisposition.^{1,3,4} A number of cases of PTS have been reported after different types of surgery.^{1,5,6} Some authors suggest that surgical stress could activate an unidentified virus that remains latent in the peripheral nervous system, as occurs in some cases of postoperative reactivation of herpes zoster.⁵ Others propose a mechanical lesion affecting the plexus during general anesthesia. Our patient was subjected to pulmonary surgery (contralateral to the clinical findings) during the previous months. However, we do not consider that to be a triggering factor, since the clinical signs develop soon after surgery (1–13 days) in all the reported cases.⁶

It especially affects men, ranging from 20 to 60 years old, with an incidence of 1.6–3 cases/100 000 population/year.⁷ The typical clinical signs and symptoms consist of severe pain in the shoulder, that develops suddenly and is not traumatic, and frequently radiates toward the cervical region and the outside of the arm. After a variable period of time, muscle atrophy appears, followed by paresis; the latter is flaccid, patchy and progressive, and its distribution does not always coincide with the painful area. It can affect several peripheral nerves and nerve roots (especially C5 and C6) or a combination of both.⁴ A third of the patients develop bilateral and symmetrical symptoms.⁷ The diagnosis is based on the medical records, physical examination and the electromyogram, which typically shows acute denervation in a specific nerve or a patchy nerve loss throughout the entire plexus.⁸ Magnetic resonance imaging of the cervical spine and brachial plexus can, in the initial phases, look normal or have hyperintense signals in T2 due to muscle edema, and, once atrophy is established, there can be an increased intramuscular linear signal intensity in T1 due to fatty infiltration.⁹ The

attempt is made to treat the symptoms with analgesics, immobility of the limb and rehabilitation.⁴ The use of moderate doses of glucocorticoids at the initiation seems to relieve the pain and accelerate recovery, which is generally slow (from months to years).² After 3 years, a third of the patients have chronic pain and 2 thirds have a functional deficit, and the recurrence rate is 26%.¹⁰

Conflicts of Interest

The authors declare they have no conflicts of interest.

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