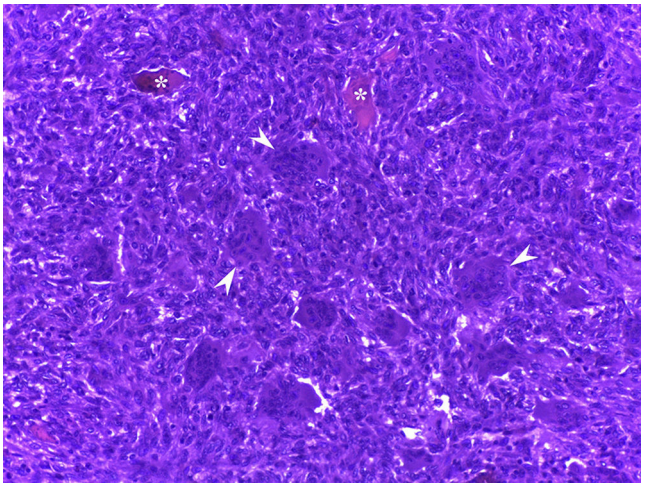

Fig. 2. Histopathology of the lesion in C2 vertebral body showing that the bone marrow has been replaced by a proliferation of round or fusiform mononuclear cells and osteoclastic-type multinucleated giant cells (arrow head). A few residual bone trabeculae can be observed among the tumor cells (asterisks) (hematoxylin and eosin, original magnification 20×).

fusion. Histopathology confirmed that the lesion was a GCT with extensions to neighboring soft tissue (Fig. 2). The patient recovered satisfactorily after the procedure, and has had no recurrences or complications after a follow-up period of 21 months.

The GCT is a benign neoplasm, characterized by the presence of osteoclastic-type multinucleated giant cells expressing CD68, that represents 4%–5% of all primary bone tumors.[2] Although it can be locally aggressive, it rarely undergoes malignant transformation (approximately 2%) and it generally metastasizes to lung.[3] It occurs most frequently between 20 and 40 years of age, slightly more among women and it has a predilection for the metaphyseal/epiphyseal region of the long bones.[4] It affects the spine in 2.7%–6.5% of cases, but it is rarely located in the cervical spine.[1,5] The most common symptoms are pain and the limited mobility, although neurological involvement is also common and occurs in up to 70% of the cases.[1,5] The differential diagnosis includes other giant cell bone tumors such as ABC, simple bone cyst, chondroblastoma, osteoid osteoma, osteoblastoma, osteosarcoma, giant cell reparative granuloma and brown tumor of hyperparathyroidism.[4,6] In this case, the presence of hyperparathyroidism had been ruled out, as the association between GCT and that disorder has been reported, including cases in which the cervical spine is involved.[7] Up to 14% of GCT exhibit cystic components consistent with secondary ABC, but this occurs with less frequency in the cervical spine.[8] The presence of osteolysis and trabeculations in imaging studies are suggestive of GCT, but the definitive diagnosis requires histopathology.[7] Magnetic resonance imaging has the best sensitivity for detecting cystic components and fluid-fluid levels.[7,9] The treatment is mainly surgical, in the attempt to resect the entire lesion whenever possible, especially if there may be neurological involvement.[1,5,10] However, the recurrence rates are high (26%–50%).[10] A number of adjuvant therapies have been utilized in the case of recurrence or inoperable lesions, including radiotherapy, selective arterial embolization, cryotherapy, argon plasma coagulation, bisphosphonates, interferon α-2b and denosumab.[5,10]

In conclusion, although the GCT in cervical spine is rare, it should be taken into account in the differential diagnosis of patients with neck pain and lytic lesions in vertebrae, especially when there are associated neurological signs.

References

1. Junming M, Cheng Y, Dong C, Jianru X, Xinghai Y, Quan H, et al. Giant cell tumor of the cervical spine: a series of 22 cases and outcomes. Spine (Phila Pa 1976). 2008;33:280–8.
2. Akerman M. Benign and malignant soft tissue tumours. In: Gray W, Mckee GT, editors. Diagnostic cytopathology. Edinburgh, UK: Churchill Livingstone; 2003. p. 17–29.
3. Chen CC, Liau CT, Chang CH, Hsu YH, Shih HN. Giant cell tumors of the bone with pulmonary metastasis. Orthopedics. 2016;39:e68–73.
4. Raskin KA, Schwab JH, Mankin HJ, Springfield DS, Hornicek FJ. Giant cell tumor of bone. J Am Acad Orthop Surg. 2013;21:118–26.
5. Martin C, McCarthy EF. Giant cell tumor of the sacrum and spine: series of 23 cases and a review of the literature. Iowa Orthop J. 2010;30:69–75.
6. Si MJ, Wang CG, Wang CS, Du LJ, Ding XY, Zhang WB, et al. Giant cell tumours of the mobile spine: characteristic imaging features and differential diagnosis. Radiol Med. 2014;119:681–93.
7. Ericsson M, Holm E, Ingemansson S, Lindholm T, Svendgaard NA. Secondary hyperparathyroidism combined with uremia and giant cell containing tumor of the cervical spine. A case report. Scand J Urol Nephrol. 1978;12:185–7.
8. Anchan C. Giant cell tumor of bone with secondary aneurysmal bone cyst. Int J Shoulder Surg. 2008;2:68.
9. Sifuentes Giraldo WA, Pachon Olmos V, Gallego Rivera JI, Vazquez Diaz M. Multiple vertebral metastases with fluid-fluid levels. Reumatol Clin. 2014;10:338–9.
10. Luksanapruksa P, Buchowski JM, Singhatanadgige W, Rose PC, Bumpass DB. Management of spinal giant cell tumors. Spine J. 2016;16:259–69.

Liudmila Valentina Maldonado-Romero,[a]
Walter Alberto Sifuentes-Giraldo,[a,*]
María Aurora Martínez-Rodrigo,[b] Carlos de la Puente-Bujidos[a]

[a] *Servicio de Reumatología, Hospital Universitario Ramón y Cajal, Madrid, Spain*
[b] *Servicio de Neurocirugía, Hospital Universitario Ramón y Cajal, Madrid, Spain*

* Corresponding author.
E-mail address: albertosifuentesg@gmail.com
(W.A. Sifuentes-Giraldo).

http://dx.doi.org/10.1016/j.reumae.2016.03.005
2173-5743/

Axial Spondyloarthritis: Can All Be Classified?☆

Espondiloartritis axiales: ¿se pueden clasificar todas?

To the Editor,

The history of ankylosing spondylitis (AS) has evolved since the first descriptions (2000 years BC), passing through the Middle Ages and, especially, the second half of the 20th century, with the discovery of HLA B27 (1973). In the fifties of the last century, there were 2 opposing schools of thought: the North American ("lumpers") and the European ("splitters"), which considered AS to be a variant of rheumatoid arthritis (RA), or as a different entity, respectively. Then evidence arose that was favorable to their separation, grouping AS and other similar diseases as "seronegative arthritides" or "spondyloarthritides" (Moss and Wright, 1976). At the present time, we prefer calling them "spondyloarthritis" (SpA), a name that indicates its inflammatory nature.[1]

With respect to the diagnosis, classification tools have been appearing, from the criteria of Boland and Present (1945),[2] then

☆ Please cite this article as: Moreno Ramos MJ, Moreno Martinez MJ, Linares Ferrando LF. Espondiloartritis axiales: ¿se pueden clasificar todas? Reumatol Clin. 2017;13:59–60.

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)	Reactive arthritis
nr axSpA (MRI+ radiography−)	Psoriatic arthritis
Radiographic axSpA not AS (radiography+ not AS, MRI−)	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 Amor[6] 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.

References

1. Zeidler H, Calin A, Amor B. A historical perspective of the spondyloarthritis. Curr Opin Rheumatol. 2011;23:327–33.
2. Boland EW, Present AJ. Rheumatoid spondylitis. A study of one hundred cases with special reference to diagnostic criteria. JAMA. 1945;129:843–9.
3. Kellgren JH, Jeffrey MR, Ball J, editors. The epidemiology of chronic rheumatism, vol. 1. Oxford: Blackwell; 1963. p. 326.
4. Population studies on the rheumatic diseases.Bennett PH, Wood PHN, editors. Proceedings of the 3rd International Symposium. Amsterdam: Excerpta Medica Foundation; 1968. p. 456–7.
5. Van der linden S, Valkenbur HA, Cats A. Evaluation of diagnostic criteria for ankylosing spondylitis. A proposal for modification of the spondylitis. New York criteria. Arthritis Rheum. 1984;27:361–8.
6. Amor B, Dougados M, Mijiyawa M. Criteria of the classification of spondylarthropathies. Rev Rhum Mal Osteoartic. 1990;57:85–9.
7. Dougados M, van der Linden S, Juhlin R, Huitfeldt B, Amor B, Calin A, et al. The European Spondyloarthropathy Study Group preliminary criteria for the classification of spondyloarthropathy. Arthritis Rheum. 1991;34:1218–27.
8. Rudwaleit M. New approaches to diagnosis and classification of axial and peripheral spondyloarthritis. Curr Opin Rheumatol. 2010;22:375–80.
9. Rudwaleit M, van der Heijde D, Landewé R, Akkoc N, Brandt J, Chou CT, et al. The Assessment of SpondyloArthritis International Society (ASAS) classification criteria for peripheral spondyloarthritis and for spondyloarthritis in general. Ann Rheum Dis. 2011;70:25–31.
10. Baraliakos X, Braun J. Non-radiographic axial spondyloarthritis and ankylosing spondylitis: what are the similarities and differences? RMD Open. 2015;1:e000053.

Manuel José Moreno Ramos,[a,*] Maria José Moreno Martinez,[b] Luis Francisco Linares Ferrando[a]

[a] *Servicio de Reumatología, Hospital Clínico Universitario Virgen de la Arrixaca, El Palmar, Murcia, Spain*
[b] *Unidad de Reumatología, Hospital Rafael Méndez, Lorca, Murcia, Spain*

* Corresponding author.
E-mail address: mjmoreno1@yahoo.es (M.J. Moreno Ramos).

http://dx.doi.org/10.1016/j.reumae.2016.03.003
2173-5743/

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.

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 pT_1apN_0, 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

☆ Please cite this article as: Cabezón I, Barreiro G, Egurbide MV. Síndrome de Parsonage-Turner: a propósito de un caso. Reumatol Clin. 2017;13:60–61.