2. Ghadri JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, et al. International expert consensus document on Takotsubo syndrome (part I): clinical characteristics, diagnostic criteria, and pathophysiology. Eur Heart J. 2018;39:2032—46.
3. Eliades M, El-Maouche D, Choudhary C, Zinsmeister B, Burman KD. Takotsubo cardiomyopathy associated with thyrotoxicosis: a case report and review of the literature. Thyroid. 2014;24:383—9.
4. Al-Salameh A, Allain J, Meimoun P, Benali T, Desailloud R. Takotsubo cardiomyopathy can occur in patients with apathetic hyperthyroidism. Thyroid. 2014;24:400—1.
5. Dahdouh Z, Roule V, Bignon M, Grollier G. Síndrome de takotsubo recurrente asociado a hipertiroidismo subclínico. Rev Esp Cardiol. 2011;64:1069—71.
6. Aggarwal S, Papani R, Gupta V. Can thyroid break your heart? Role of thyroid in Takotsubo cardiomyopathy: a single center retrospective study. Int J Cardiol. 2015;184:545—6.
7. Templin C, Ghadri JR, Diekmann J, Napp LC, Bataiosu DR, Jaguszewski M, et al. Clinical features and outcomes of Takotsubo (stress) cardiomyopathy. N Engl J Med. 2015;373:929—38.
8. Rueda D, Aguirre R, Contardo D, Finocchietto P, Hernandez S, di Fonzo H. Takotsubo myocardiopathy and hyperthyroidism: a case report and literature review. Am J Case Rep. 2017;18:865—70.
9. Stollberger C, Wegner C, Finsterer J. Seizure-associated Takotsubo cardiomyopathy. Epilepsia. 2011;52:e160—7.
10. Inamasu J, Ganaha T, Nakae S, Ohmi T, Wakako A, Tanaka R, et al. Therapeutic outcomes for patients with aneurysmal subarachnoid hemorrhage complicated by Takotsubo cardiomyopathy. Acta Neurochir (Wien). 2016;158:885—93.
11. Scheitz JF, Mochmann HC, Witzenbichler B, Fiebach JB, Audebert HJ, Nolte CH. Takotsubo cardiomyopathy following ischemic stroke: a cause of troponin elevation. J Neurol. 2012;259:188—90.
12. Yoshimura S, Toyoda K, Ohara T, Nagasawa H, Ohtani N, Kuwashiro T, et al. Takotsubo cardiomyopathy in acute ischemic stroke. Ann Neurol. 2008;64:547—54.
13. Abi-Saleh B, Iskandar SB, Schoondyke JW, Fahrig S. Takotsubo syndrome as a consequence of transient ischemic attack. Rev Cardiovasc Med. 2006;7:37—41.
14. Finsterer J, Aliyev R. Takotsubo syndrome: consequence or cause of ischemic stroke. Funct Neurol. 2014;29:282.
15. Bersano A, Melchiorre P, Moschwitis G, Tavarini F, Cereda C, Micieli G, et al. Takotsubo syndrome as a consequence and cause of stroke. Funct Neurol. 2014;29:135—7.

B. Febrero[a,*], J.M. Rodríguez[a], A. Morales[b], P. Parrilla[a]

[a] *Unidad de Cirugía Endocrina, Servicio de Cirugía General, Hospital Clínico Universitario Virgen de la Arrixaca, Instituto Murciano de Investigación Biomédica (IMIB), El Palmar, Murcia, Spain*
[b] *Servicio de Neurología, Hospital Clínico Universitario Virgen de la Arrixaca, Instituto Murciano de Investigación Biomédica (IMIB), El Palmar, Murcia, Spain*

* Corresponding author.
E-mail address: beatrizfebrero@hotmail.com (B. Febrero).

27 October 2018

https://doi.org/10.1016/j.nrleng.2019.01.012
2173-5808/

Acute lower limb paresis in a patient with rheumatoid arthritis: emergency neuroimaging findings*

Paresia aguda de miembro inferior en paciente con artritis reumatoide: hallazgos en la neuroimagen de urgencia

Dear Editor:

Rheumatoid arthritis is a chronic, inflammatory, systemic disease that mainly affects ligaments, joints, and ultimately bone. Its pathophysiology involves genetic, autoimmune, and environmental factors. The condition is caused by synovial inflammation, leading to destruction of the joints.[1] However, the condition has also been associated with cardiovascular, pulmonary, digestive, haematological, and more rarely neurological manifestations.[2] Rheumatoid arthritis is one of the most frequent rheumatic disorders worldwide, with an estimated prevalence of 0.5%-1% in the adult population.[3,4]

We present the case of a 76-year-old woman with no known vascular risk factors, under treatment with methotrexate and folic acid due to chronic rheumatoid arthritis. She visited our hospital's emergency department due to inability to walk as a result of loss of muscle strength in the right leg of less than 24 hours' progression. She presented no neck pain and reported no history of trauma. The examination revealed paresis in the proximal part of the right leg (3/5), mild hyperreflexia of the right limbs, and extensor plantar reflex in the right foot, associated with impaired proprioceptive sensitivity (arthrokinetic and positional) in the right leg. Examination of cranial nerves and tactile and pain sensitivity revealed no alterations. An emergency blood analysis including a biochemistry study and a complete blood count yielded normal results, and a head CT scan revealed no alterations in the brain parenchyma. However, it did show incipient compression of the medulla oblongata by the dens of the axis, with a posterior atlantodental interval < 13 mm (radiological measurement between the posterior surface of the odontoid process and the posterior arch of the atlas,[5] with normal values being > 14 mm) (Fig. 1).

Clinical findings were anatomically correlated with radiological findings, and compatible with incomplete right

* Please cite this article as: Macías-García D, Jurado Serrano J, Parada Blazquez MJ, Moniche F. Paresia aguda de miembro inferior en paciente con artritis reumatoide: hallazgos en la neuroimagen de urgencia. Neurología. 2020;35:593—595.

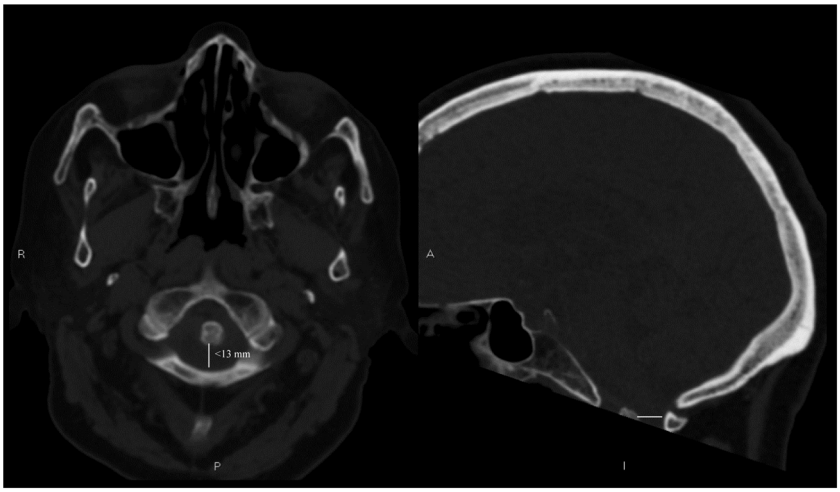

Figure 1 Head CT scan (bone window): axial and sagittal planes. Both images show a posterior atlantodental interval < 13 mm (white line).

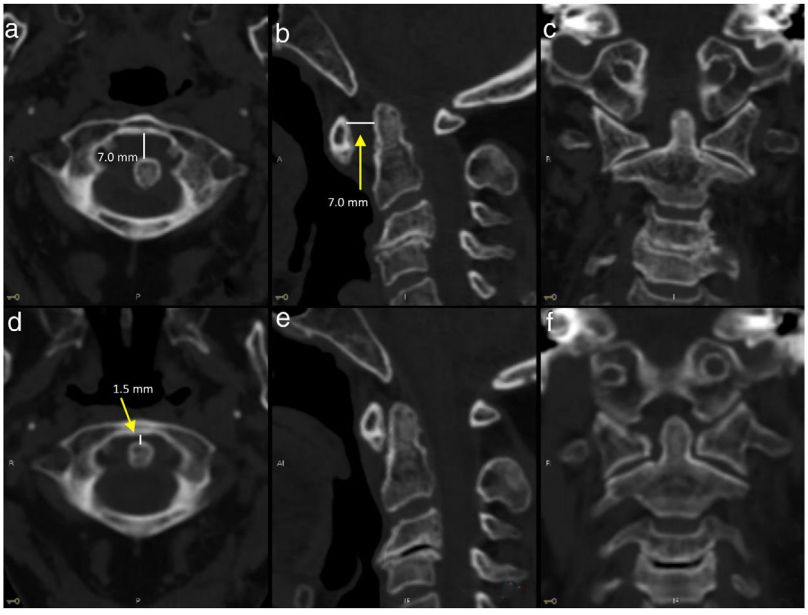

Figure 2 Non-contrast neck CT images taken before (a—c) and after (d—f) cervical spine immobilisation with a Halo vest. The neuroimaging study performed before the intervention revealed an abnormal anterior atlantodental interval (> 3 mm, a and b), which was subsequently corrected (d and e).

hemimedullary syndrome. In view of the patient's history and symptoms, we decided to perform a neck CT scan (Fig. 2), which revealed atlantoaxial subluxation and an anterior atlantodental interval of 7 mm (distance between the anterior surface of the odontoid process and the anterior arch of the atlas[5]; normal value, < 3 mm). The patient was instructed to wear a Halo vest to immobilise the cervical spine, and subsequently underwent surgery for screw fixation. At discharge, motor deficits in the right leg persisted.

Cervical myelopathy secondary to atlantoaxial subluxation is a known complication in patients with rheumatoid arthritis, and is associated with poorer prognosis and greater mortality rates.[6] Involvement of this large joint, which is occasionally asymptomatic, is described in up to 40% of patients with rheumatoid arthritis, and is one of the most frequent alterations after small joint erosion.[7] The risk of developing cervical instability has been associated with a number of factors, including long disease progression, long-term corticosteroid treatment, involvement of other large joints, and high degree of inflammatory activity of the disease (elevated C-reactive protein levels or erythrocyte sedimentation rate).[7,8] The development of myelopathy in the context of rheumatoid arthritis is frequently associated with neck pain, occipital headache, Lhermite sign, tinnitus, otalgia, and crepitation with neck movement.[7]

We present the case of a patient with rheumatoid arthritis and atlantoaxial subluxation manifesting as acute hemimedullary syndrome, without neck pain or history of

trauma. Although several cases have been reported of cervical myelopathy associated with Brown-Sequard syndrome,[9] the interest of our case lies in the incomplete, atypical presentation of the syndrome (acute presentation without the prodromal symptoms listed above), which may have delayed diagnosis. Emergency neuroimaging (atlantodental interval in axial CT images) may be extremely useful for early diagnosis in the event of acute presentation in patients with no history of trauma, or not presenting other symptoms suggestive of spinal cord compression.

Atlantoaxial subluxation is a known, alarming complication of rheumatoid arthritis. However, cervical instability may be silent in some patients and can remain undiagnosed for years. Neuroimaging studies may help in the early diagnosis of atypical cases, enabling early surgical treatment.

Funding

Daniel Macías-García has received funding as part of the ''Río Hortega'' training programme (CM18/00142; Institute of Health Carlos III). The remaining authors have received no funding for this study.

Conflicts of interest

The authors have no conflicts of interest to declare.

Bibliografía

1. Scott DL, Wolfe F, Huizinga T. Rheumatoid arthritis. Lancet. 2010;376:1094—108.
2. Gullicl NJ, Scott DL. Co-morbidities in established rheumatoid arthritis. Best Pract Res Clin Rheumatol. 2011;25:469—83.
3. Balsa A, Lojo-Oliveira L, Alperi-López M, García-Manrique M, Ordónez-Cañizares C, Pérez L, et al. Prevalence of comorbidities in rheumatoid arthritis and evaluation of their monitoring in clinical practice: the Spanish cohort of the COMORA study. Reumatol Clin. 2019;15:102—8, http://dx.doi.org/10.1016/j.reuma.2017.06.002.
4. Myasoedova E, Crowson CS, Kremers HM, Therneau TM, Gabriel SE. Is the incidence of rheumatoid arthritis rising? Results from Olmsted County, Minnesota, 1955—2007. Arthritis Rheumatol. 2010;62:1576—82.
5. Krauss WE, Bledsoe JM, Clarke MJ, Nottmeier EW, Pichelmann MA. Rheumatoid arthritis of the craniovertebral junction. Neurosurgery. 2010;66:A83—95.
6. Riise T, Jacobsen BK, Gran JT. High mortality in patients with rheumatoid arthritis and atlantoaxial subluxation. J Rheumatol. 2001;28:2425—9.
7. Gillick JL, Wainwirght J, Das K. Rheumatoid arthritis and the cervical spine: a review on the role of surgery. Int J Rheumatol. 2015, http://dx.doi.org/10.1155/2015/252456. ID 252456.
8. Terashima Y, YurubeT, Hirata H, Sugiyama D, Sumi M. Predictive risk factors of cervical spine instabilities in rheumatoid arthritis: a prospective multicenter over 10-year cohort study. Spine. 2017;42:556—64.
9. Ellkayam O, Yaron M. Brown-Sequard syndrome as a presenting symptom of cervical myelopathy in a rheumatoid arthritis patient. Clin Exp Rheumatol. 1995;13:673—4.

D. Macías-García[a,*], J. Jurado Serrano[b], M.J. Parada Blazquez[b], F. Moniche[a]

[a] *Servicio de Neurología y Neurofisiología, Hospital Universitario Virgen del Rocío, Sevilla, Spain*
[b] *Servicio de Radiodiagnóstico, Hospital Universitario Virgen del Rocío, Sevilla, Spain*

* Corresponding author.
E-mail address: danielmacgar@gmail.com
(D. Macías-García).

https://doi.org/10.1016/j.nrleng.2019.05.001
2173-5808/

Amyotrophic lateral sclerosis and myasthenia gravis overlap syndrome: 3 new cases☆

Esclerosis lateral amiotrófica y miastenia gravis (síndrome *overlap*): presentación de 3 nuevos casos

Dear Editor:

The association of myasthenia gravis (MG) and amyotrophic lateral sclerosis (ALS) (overlap syndrome) is infrequent in clinical practice. The available evidence suggests that immunomodulatory therapy has a protective effect in the early stages of motor neuron disease (MND).[1,2] We present 3 cases of this overlap syndrome; Table 1 summarises the characteristics of these patients.

Patient 1

The patient was a 52-year-old man with initial symptoms of bilateral ptsosis, diplopia, and dysphagia; 6-8 months later, he presented left brachial paresis with thenar atrophy, global hyperreflexia, and increased jaw jerk reflex. 3-Hz repetitive stimulation obtained a decrement of > 10% in the fifth potential in the abductor digiti minimi. Anti-acetylcholine receptor (anti-AChR) antibody titre was 0.74 (positive results, > 0.7), and remained stable in subsequent determinations. Fasciculations and denervation potentials were observed in an electromyography study (EMG) per-

☆ Please cite this article as: Santos-Lasaosa S, López-Bravo A, Garcés-Redondo M, Atienza-Ayala S, Larrodé-Pellicer P. Esclerosis lateral amiotrófica y miastenia gravis (síndrome *overlap*): presentación de 3 nuevos casos. Neurología. 2020;35:595—597.