

likelihood of adverse events.⁵ Levetiracetam is excreted largely by the kidneys and a positive correlation has been reported between creatinine clearance and drug clearance. In fact, the elderly have been shown to require a 40% dose reduction in order to achieve the same serum concentration and also to have a greater risk of side effects even at the same serum levels in comparison with young individuals.⁶

Consequently, with this case we have wanted to illustrate that before prescribing levetiracetam in elderly patients, kidney clearance must be calculated and the dose must be titrated up gradually in order to prevent adverse events;^{6,10} in addition, electrocardiographic monitoring is also needed after initiating de novo treatment in patients at risk for developing QT prolongation.

References

1. Rautaharju PM, Surawicz B, Gettes LS, Bailey JJ, Childers R, Deal BJ, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. *J Am Coll Cardiol*. 2009;53:982-91.
2. Muñoz Castellano J. Síndrome de QT largo y Torsade de Pointes. *Emergencias*. 2004;16:85-92.
3. Perales Fraile I, Moya Mir M.S. Actuación en urgencias ante una crisis convulsiva en adultos. *Emergencias*. 2005;17:S83-9.
4. Yusta A. Crisis convulsiva. Concepto, Clasificación y Etiología. *Emergencias*. 2005;17:S68-73.
5. French J. Use of levetiracetam in special populations. *Epilepsia*. 2001;42(Suppl 4):40-3.
6. Hirsch LJ, Arif H, Buchsbaum R, Weintraub D, Lee J, Chang JT, et al. Effect of age and comedication on levetiracetam pharmacokinetics and tolerability. *Epilepsia*. 2007;48:1351-9.
7. Hulhoven R, Fosillon D, Bridson WE, Meeus MA, Salas E, Stockis A. Effect of levetiracetam on cardiac repolarization in healthy subjects: a single-dose, randomized, placebo- and active-controlled, four-way crossover study. *Clin Ther*. 2008;30:260-70.
8. Letsas KP, Efremidis M, Kounas SP, Pappas LK, Gavrielatos G, Alexanian IP, et al. Clinical characteristics of patients with drug-induced QT interval prolongation and torsade de pointes: identification of risk factors. *Clin Res Cardiol*. 2009;98:208-12.
9. Roden D.M. Taking the "idio" out of "idiosyncratic": predicting torsade de pointes. *Pacing Clin Electrophysiol*. 1998;21:1029-34.
10. Abajo del Alamo C, García Rodicio S, Casado Pérez J, Ausín Pérez ML, Catalá Fndado M.A. Prevention of adverse events through renal dosage adjustment in institutionalized elders. *Rev Esp Geriatr Gerontol*. 2009;44:34-7.

B. Rojano Martín, M. Maroto Rubio, N. Bilbao Ormazabal, F.J. Martín-Sánchez*

Servicio de Urgencias, Hospital Clínico San Carlos, Madrid, Spain

*Corresponding author.

E-mail: fjjms@hotmail.com (F.J. Martín-Sánchez).

Balo's concentric sclerosis

Esclerosis concéntrica de Baló

Dear Editor,

Balo's concentric sclerosis is a demyelinating disease described for the first time by Jozsef Baló in 1928 as "periaxial concentric sclerosis".¹

The classical clinical presentation is that of a sub-acute, fulminating, fatal encephalopathy, albeit in recent years cases have appeared in the literature with a fairly benign course, including full recovery from the disease.²

Historically speaking, the diagnosis has been made post mortem, by the characteristic pathological anatomy with lesions consisting of concentric rings of demyelination alternating with normal white matter. However, the most recent revisions support the role of magnetic resonance of the brain for early and definitive diagnosis, which has had a dramatic impact on the prognosis of the illness, improving the morbi-mortality it entails.

We present the case of a female patient with a sub-acute syndrome of neurological focality and a magnetic resonance image of the brain compatible with Balo's concentric sclerosis.

A twenty-seven-year-old female without any history of interest was admitted to our hospital due to a progressive

course of weakness on the right side of the body, difficulty in co-ordinating the upper right limb and gait alteration for the last three months.

The general examination is normal and the neurological examination yields the following findings of interest: hemiparesis on the right side with signs of pyramidalism and spasticity of the lower limbs, accompanied by postural and kinetic tremor of the right arm. Three diffuse, rounded hypodense areas in the sub-cortical white matter with ring-shaped uptake of contrast are seen on the computerized tomography taken in the Emergency Room.

A complete magnetic resonance (MR) scan was performed of the brain and spine revealing several lesions in the supratentorial sub-cortical white matter (the largest measuring 4 cm) with an onion-like structure, displaying hypointense concentric rings in T1-weighted sequences (fig. 1) and hyperintense in T2 (fig. 2), alternating with isointense layers. After injecting paramagnetic contrast, they display ring enhancement (fig. 3). These images show no associated oedema surrounding the lesion or increased relative blood volume in the perfusion sequences. On spectroscopy, the choline spike is increased to 3.2 ppm and lactate is evidenced at 1.3 ppm (fig. 4). No infratentorial lesions are evident.

The cerebrospinal fluid (CSF) study rules out the presence of oligoclonal bands of IgG and malignant cells.

After the results of the complementary testing and with the diagnostic suspicion of Balo's concentric sclerosis-type



Figure 1 T1-weighted axial magnetic resonance image. Three hypointense areas are seen, one of which is rounded, with hypointense concentric rings.

demyelinating disease, treatment was initiated with 1-gramme intravenous boli of methylprednisolone for 5 days, with gradual improvement of the clinical syndrome. The patient remains asymptomatic with good evolution at the subsequent check-ups.

Balo's concentric sclerosis (BCS) is an unusual demyelinating disease, although its true incidence and prevalence is not known, given the fact that the vast majority of cases have been diagnosed post mortem. To date, 70 cases have been reported in the scientific literature.^{3,4}

The aetiopathogenesis of the disease is unknown: it shares clinical and pathological characteristics with multiple



Figure 2 T2-weighted axial magnetic resonance image. Three hyperintense areas are seen in the sub-cortical white matter, without any mass effect. Two of them, right fronto-parietal, rounded lesions with hyperintense concentric rings alternating with isointense bands.



Figure 3 T1-weighted axial magnetic resonance image, after administration of intravenous contrast. Ring-shaped enhancement of the lesions is seen revealing the typical "onion-like" image.

sclerosis and acute disseminated encephalitis, which points to a possible autoimmune origin.

Clinically, it debuts as a sub-acute encephalopathy with signs of focal neurological dysfunction. The clinical course is monophasic in most patients, rapidly evolving towards mortality weeks to months after onset; nevertheless, in recent years cases have been reported with a relatively benign course of disease and even full remission.⁵

Classically, the diagnosis has been made by specific pathology study, where we find lesions comprising concentric rings with alternating demyelinated and normal white matter.⁶

The appearance of MR has made it possible to describe findings deemed to be pathognomonic, which makes early diagnosis and treatment possible. The MR scan exhibits the typical "onion-like" image, of hypointense concentric rings on T1 sequences and hyperintense on T2-weighted sequences, alternating isointense normal white substance; after injecting gadolinium, the demyelinated layers reveal ring-shaped enhancement. The spectroscopy reveals an increase in the choline spike with a decrease in N-acetylaspartate, a finding not specific to BCS, and representing an increase in membrane turnover and neuronal damage.⁷⁻⁹

The analysis of the CSF did not yield the specific characteristics of BCS and an increase in IgG and oligoclonal bands was found.

Given its low prevalence, there are no placebo-controlled, randomized studies demonstrating the efficacy of any specific treatment agent for BCS. Due to the similarities with multiple sclerosis, some authors support treating the acute episodes of neurological dysfunction with high doses of corticosteroids and plasmapheresis in the case of a poor response.¹⁰ There is no evidence regarding long-term immunosuppressant treatment.

Our case report supports the latest reviews indicating that Balo's concentric sclerosis may be a self-limiting

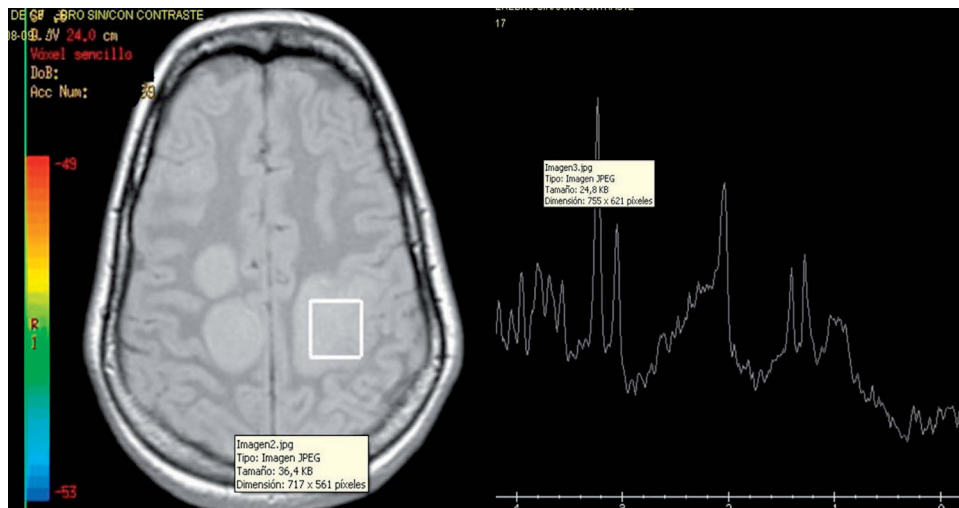


Figure 4 Spectroscopic study. An increase in the choline spike and decrease in the Nacetylaspartate is seen in the lesions, which represents increased membrane turnover and neuronal damage.

disease, following a fairly benign course of disease, and the importance that brain MR scans have taken on as a non-invasive method enabling early diagnosis of the disease with the aim of beginning early treatment with corticosteroids.

References

1. Balo J. Encephalitis periaxialis concentrica. *Arch Neurol Psychiatry*. 1928;19:242-64.
2. Karaarslan and, Altintas A, Senol U, et al. Balo's concentric sclerosis: clinical and radiologic features of five cases. *AJNR Am J Neuroradiol*. 2001;22:1362-7.
3. Pearce J.M.S. Baló's encephalitis periaxialis concentrica. *Eur Neurol*. 2007;57:59-61.
4. Pelzers and, Van Goethem JW, Van den Hauwe L, et al. Baló's concentric sclerosis. *JBR-BTR*. 2008;91:108-9.
5. Moore GRW, Berry K, Oger JF, et al. Balo's concentric sclerosis: Surviving normal myelin in a patient with a relapsing-remitting clinical course. *Mult Scler*. 2001;7:375-82.
6. Moore GR, Neumann PE, Suzuki K, et al. Balo's concentric sclerosis: new observations on lesion development. *Ann Neurol*. 1985;17:604-11.
7. Caracciolo JT, Murtagh RD, Fojiani AM, et al. Pathognomonic MR imaging findings in Balo concentric sclerosis. *AJNR Am J Neuroradiol*. 2001;22:292-3.
8. Chen CJ, Fo LS, Wang LJ, Wong Y.C. Balo's concentric sclerosis: MRI. *Neuroradiology*. 1996;38:322-4.
9. Kastrup O, Stude P, Limmroth V, et al. Balo's concentric sclerosis demonstrated by contrast MRI. *Neurology*. 2001;57:1610.
10. Weinshenker BG, O'Brien PC, Petterson TM, et al. A randomized trial of plasma exchange in acute central nervous system inflammatory demyelinating disease. *Ann Neurol*. 1999;46:878-86.

R. Hernández-Clares,* D. Di Capua, M. Bartolomé,
V. de las Heras, R. Arroyo

Unidad de Enfermedades Desmielinizantes, Servicio de Neurología, Hospital Clínico San Carlos, Madrid, Spain

*Corresponding author.

E-mail: rociohclares@hotmail.com (R. Hernández-Clares).