

may allow for the full remission of symptoms and prevent irreparable cerebellar damage.

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Comments on the review article



«Cerebral radiation necrosis: Diagnostic challenge and clinical management»[☆]

Comentario al artículo de revisión «Necrosis cerebral por radiación: desafío diagnóstico y tratamiento clínico»

Dear Editor:

I read with great interest the review article by Eisele and Dietrich¹ on the exciting, little-understood subject of

cerebral radiation necrosis secondary to surgical treatment for brain tumours.

The mechanisms involved in the pathogenesis of the condition are largely unknown; they may even vary with respect to the factors cited by authors as determinants: type of radiation, dose, treatment volume, and fractionation schedule. I would also like to add the type of supplementary treatment administered, which is normally chemotherapy, as mentioned in the review article.

The adjuvant chemotherapy used was different in each of the various studies establishing neuro-radiological criteria over the past 3 decades.^{2–4} It is therefore admirable that other authors⁵ have attempted to compare the degree of correlation or concordance according to these criteria, in order to assess the type of progression, and suggest adding hyperintensity to FLAIR sequences to make contrast-enhanced sequences even more useful for assessing subsequent clinical worsening. We should also highlight that they compare treatments with the same adjuvant chemotherapy, in this case bevacizumab + irinotecan, which confers validity to the study and its results. Their main

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contribution was the inclusion of FLAIR sequence to the RECIST criteria (RECIST-F).

Based on the recruitment of a large number of patients from the AVAglio trial and after failing to demonstrate the usefulness of antiangiogenic therapy as a first line treatment after surgery in patients with glioblastoma, other authors also establish criteria to assess the response to this therapy and for this to be done uniformly.⁶

Our understanding of neuroradiology is expanding in parallel to the different surgical treatments and chemotherapies for patients with brain tumours. Although our gold standard continues to be anatomical pathology complemented by genetic/molecular techniques, advanced magnetic resonance imaging makes categorisation of tumours quicker and more reliable, which provides clarity in decision-making regarding these patients.

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Clinical and magnetic resonance imaging abnormalities of the tongue in patients with amyotrophic lateral sclerosis[☆]



Anormalidades clínicas y por resonancia magnética en lengua de pacientes con esclerosis lateral amiotrófica

Dear Editor:

Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disease characterised by rapid clinical deterioration. Survival times in patients with the condition range between 15.7 and 47 months from disease onset (mean, 29.1 months), depending on the series.^{1,2} The clinical features of the disease are heterogeneous and have been attributed to the combination of neurological signs and symptoms of upper and lower motor neuron dysfunction and death.³

Dysarthria and tongue atrophy with fasciculations are salient clinical features of bulbar- and bulbospinal-onset ALS, and also appear at later stages of spinal-onset ALS. Abnormalities of the tongue are caused by damage to the hypoglossal nucleus, which leads to flaccid dysarthria, a typical feature of lower motor neuron dysfunction. Upper motor neuron death results in corticobulbar tract dysfunction, which causes spastic dysarthria. Nasal voice may be observed in some patients with ALS who have no structural alterations in the tongue.⁴ Previous studies of patients with ALS report an incidental finding called the “bright tongue sign” in sagittal MR images of the brain. This sign has been associated with degeneration of the tongue and proposed as a useful radiological feature for diagnosing ALS.^{5–8} However, the significance of this magnetic resonance imaging (MRI) finding is still unknown. All patients with ALS and tongue abnormalities undergo brain MRI scans. However, clinicians frequently pay little attention to these abnormalities if patients show clinical signs of tongue atrophy and fasciculations.

We present a series of patients with ALS and tongue abnormalities, displaying a correlation between clinical and radiological (MRI) findings (bivariate analyses, *t* test, chi-square test, Mann–Whitney *U* test). We analysed the following clinical data: phenotype at baseline, disease severity, and progression time at the time of the MRI scan. We evaluated 43 patients with ALS according to the revised El Escorial clinical and neurophysiological diagnostic criteria.⁹ All patients completed the revised ALS Functional Rating

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