Focal cortical dysplasia type IIIa and isolated hippocampal sclerosis

TO THE EDITOR: We read with great interest the paper by Dührsen et al.7 (Dührsen L, Sauvigny T, House PM, et al: Impact of focal cortical dysplasia Type IIIa on seizure outcome following anterior mesial temporal lobe resection for the treatment of epilepsy. J Neurosurg [epub ahead of print July 28, 2017. DOI: 10.3171/2017.2.JNS161295]). The authors’ study is an effort to gain a deeper insight into the correlation between histopathological features and seizure outcome in temporal lobe epilepsy (TLE) treated using anteromesial temporal lobe resection (AMTLR).

Recent neuropathological classifications of epileptogenic lesions such as hippocampal sclerosis (HS),2 granule cell pathology (GCP),2 and focal cortical dysplasia (FCD)4 achieved a more precise definition of histopathological features and subtypes, which in turn may allow more accurate clinicopathological correlations and prognostic assessment. The 2011 International League Against Epilepsy (ILAE) classification4 introduced a new class, FCD type III, evidencing that those cases in which cortical lamination abnormalities (typical of FCD type I) are adjacent to a principal lesion present clinical features and seizure outcome similar to those of the latter.

We absolutely agree with the hypothesis that FCD IIIa is a single epileptic unit and therefore an individual disease pattern, and that reelin, a glycoprotein responsible for neuronal migration and architecture in the hippocampus and cortical layers, may play an important role in a subset of FCD IIIa.13 However, Dührsen et al. report that patients with FCD IIIa (the combination of HS and FCD type I in the temporal pole) had a significantly better seizure outcome after AMTLR than patients with HS alone. These results challenge the original description of FCD IIIa, in which the principal lesion-determining seizure outcome is HS.4

In our experience, patients with FCD IIIa who were submitted to AMTLR showed a seizure outcome similar to patients with isolated HS (84% vs 82% in Engel class I) and a better outcome than patients with isolated FCD type I (63% in Engel class I).10,12 A worse postsurgical outcome for isolated FCD type I was reported by many other authors.18,19 In 60%–70% of cases HS is associated with FCD in the temporopolar cortex.4,6,8,10,11,19 Dührsen et al. observed FCD IIIa in only 25.5% of patients, but the challenges in this field of neuropathology are well known.5,14 Furthermore, it has been hypothesized that atypical HS subtypes, i.e., HS ILAE types 2 and 3, are associated with less favorable outcome,3,10,11,16,20 which may also be related to the status of the dentate gyrus (namely the absence or presence of GCP)2,10,15 but in the series by Dührsen et al. these pathological findings have not been provided.

We agree with the authors that the presence of FCD type I is currently difficult to detect by MRI.10,18 In the face of clinical and imaging features of TLE related to HS, the only manner to decide the extension of temporal resection and consequently the surgical strategy (i.e., selective amygdalohippocampectomy [SAH] or AMTLR) is the use of noninvasive preoperative neurophysiological study (long-term video-electroencephalography monitoring), which only rarely highlights a strictly mesial epileptogenic zone. Temporal pole neocortex is frequently involved in the epileptogenic network either functionally or with a postsurgically documented pathological substrate (such as FCD type I).6,10,19 These data should imply a clear advantage of AMTLR in terms of seizure outcome. Instead, although we agree with the authors about considering AMTLR the optimal surgical strategy for TLE associated with HS, the superiority of this approach over SAH has not yet been determined.16

The authors report that 33.3% of the patients included in the study were undergoing a second procedure because of persistent seizures, which may suggest the presence of a more complex epileptogenic network, as in temporal plus epilepsy. The authors’ results of a worse seizure prognosis for isolated HS may therefore have some explanations, such as a possible role of histopathological subtypes of HS and granule cell status on seizure outcome,5,14 the extension of resection of mesial temporal structures,17 and the presence of temporal plus epilepsy.1,19

Marco Giuliani, MD
Gianfranco Vornetti, MD
IRCCS Institute of Neurological Sciences, Bellaria Hospital, Bologna, Italy
Gianluca Marucci, MD, PhD
IRCCS Foundation Neurological Institute “C. Besta,” Milan, Italy

References
Is there any relationship between estrogen receptor/progesterone receptor status and recurrence of meningioma?

TO THE EDITOR: We read with great interest the article by Hua and colleagues, who have reported the prognostic value of estrogen receptor (ER) expression in a total of 87 patients whose tumors were pathologically diagnosed as WHO grade III meningioma (Hua L., Zhu H., Li J., et al: Prognostic value of estrogen receptor in WHO Grade III meningioma: a long-term follow-up study from a single institution. J Neurosurg [epub ahead of print August 18, 2017. DOI: 10.3171/2017.2.JNS162566]). The authors concluded that patients treated for recurrent meningioma had worse progression-free survival than those treated for primary disease (p = 0.001) and that ER expression (p = 0.008) was an independent prognostic factor for progression-free survival of patients with WHO grade III meningioma. We commend the authors for performing such an interesting study. We noticed, however, that they did not analyze cases in which tumors demonstrated both ER and progesterone receptor (PR) expression. Pravdenkova et al. reported that in their series of 18 patients with ER-positive meningiomas, ER expression alone was found in only 2 tumors and that in 16 cases the tumors showed ER/PR status and recurrence of meningioma. If such a relationship exists, we wonder whether there is a relationship between ER/PR status and meningioma recurrence. If such a relationship exists, which ER/PR status can predict recurrence?

A few studies have explored the relationship between ER/PR status and recurrence of meningioma and have demonstrated a relationship between PR status and meningioma recurrence. If such a relationship exists, which PR status can predict recurrence?