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BRAIN TUMORS

TUMORS AND TUMOR-RELATED EPILEPSY

Epileptologists from the Case Medical Center, Cleveland, Ohio; and the Comprehensive Epilepsy Center, Northwestern University, Feinberg School of Medicine, Chicago, Illinois, are guest editors of the December 2013 Epilepsia Supplement on Tumors and Tumoral Epilepsy. Among 24 items discussed, one deals with the relation of developmental brain tumors to adjacent cortical dysplasia. The natural histories and presentations of "epileptomas" and "long-term epilepsy-associated tumors (LEATS)" differ according to histologic subtype. The associated pathologies of focal cortical dysplasia (FCD) and developmental tumor raise the question of epileptogenicity of FCD and the necessity to resect both tumor and adjacent dysplasia. Is the tumor and dysplasia the same lesion with distinct components along a developmental continuum, or is the FCD an entirely separate lesion, not requiring surgical resection? If the tumor and surrounding dyslaminated tissue are considered epileptogenic lesions on their own, or dual pathologies, surgical resection of both is indicated. If both are part of the same histopathologic continuum, or if the surrounding dyslaminated cortex is an incidental finding, the surgical relevance of the dysplasia as epileptogenic tissue is variable. Seizure control may be achieved by partial resection of only the epileptogenic zones of the lesion. Acute electrocorticography (ECoG) shows two patterns: 1) frequent, almost continuous spiking, found over the tumor and its immediate surroundings, areas requiring complete resection, and 2) occasional, intermittent spikes, over areas usually non-epileptogenic and not requiring resection. (Lhatoo SD, Moghimi N, Schuele S. Tumors and tumoral epilepsy. **Epilepsia** 2013 Dec;54 Suppl 9:1-4; Palmini A, Paglioli E, Silva VD. Developmental tumors and adjacent cortical dysplasia: Single or dual pathology? **Epilepsia** 2013 Dec;54 Suppl 9:18-24).

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COMMENTARY. **Association of FCD with dysembryoplastic neuroepithelial tumor (DNT).** DNT is a glioneuronal tumor, increasingly recognized as a cause of intractable partial epilepsy in children. Located in the supratentorial cortex, especially the temporal lobe, DNT occurs in three histologic subtypes (simple, complex, and nonspecific). Complex and nonspecific forms are frequently associated with FCD. Based on a series of 78 patients, aged 3-54 years, seen at Sainte-Anne Hospital and other centers in Paris, France, 73% of DNTs were located in the temporal lobe. Histologic subtypes differentiated by MRI were Type 1 (cystic/polycystic-like, complex or simple forms), and Type 2 (nodular-like) and Type 3 (dysplastic-like) nonspecific forms. The epileptogenic zone varied according to MRI subtype: surgical resection could be restricted to the tumor in type 1 and was more extensive in other MRI subtypes, especially in type 3 MRI. Early surgical excision is important in control of DNT-associated epilepsy [1].

References.
1. Chassoux F, Daumas-Duport C. Epilepsia. 2013 Dec;54 Suppl 9:129-34.

ATTENTION DEFICIT DISORDERS

METHYLPHENIDATE IN ADHD AND REFRACTORY EPILEPSY

Investigators from Great Ormond Street Hospital, and Institute of Child Health, London, UK, reviewed the case notes of all patients treated with methylphenidate (MPH) for ADHD at an epilepsy center between 1998 and 2005. All patients were taking AED treatment for at least 6 months at time of starting MPH. Of 18 patients identified with refractory epilepsies (14 generalized, 4 focal) and ADHD, 13 were male and 5 female, median age 11.5 years (range 6-18 years). ADHD symptoms improved in 61% of patients following behavioral management and daily MPH 0.3-1 mg/kg/day. Treatment was discontinued because of adverse effects in 3 (18%) patients (dysphoria in 2, anxiety in 1). Seizure control showed no statistical impairment caused by use of MPH; one patient showed an increase in seizure frequency after initiating MPH, and seizure frequency remained elevated after discontinuation of MPH. (Fosi T, Lax-Pericall MT, Scott RC, Neville BG, Aylett SE. Methylphenidate treatment of attention deficit hyperactivity disorder in young people with learning disability and difficult-to-treat epilepsy: Evidence of clinical benefit. **Epilepsia** 2013 Dec;54(12):2071-81).

COMMENTARY. MPH may be beneficial in treatment of ADHD and AED refractory epilepsy, without significant impairment of seizure control. Despite benefits of MPH in the majority of comorbid epilepsy/ADHD patients, an exacerbation of seizures occurring in the occasional patient requires caution and moderation in introduction of MPH and continuation of AED therapy in optimal dosage. Of 20 epilepsy/ADHD patients whose seizures were controlled by AEDs, none had attacks while taking MPH [1]. MPH is generally safe in children with comorbid epilepsy/ADHD whose seizures are controlled with AEDs. In contrast, children with ADHD and subclinical epileptiform EEG not treated with AED are at increased risk of seizures with introduction of MPH. The incidence of seizures following MPH was 16.7% in children with ADHD and centro-temporal (rolandic) spikes compared to 0.6% in ADHD patients with normal EEGs [2].