Continuing Medical Education Article

The Role of Radionuclide Imaging in Epilepsy, Part 2: Epilepsy Syndromes

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Disclosure
In accordance with ACCME Revised Standards for Commercial Support and SNM Conflict-of-Interest Policy, the authors have indicated no relevant relationships that could be perceived as a real or apparent conflict of interest. Disclosure of a relationship is not intended to suggest or to condone bias but is made to provide participants with information that might be of potential importance to their evaluation of the activity.

Target Audience
This article contains information of value to physicians in nuclear medicine, neurologists, and nuclear medicine technologists.

Objectives
On successful completion of this activity, participants should be able to:
1. Identify syndromes in infancy and childhood associated with medically refractory seizures.
2. Define the roles of molecular imaging in the management of epilepsy syndromes.
3. Distinguish favorable and unfavorable molecular imaging characteristics of patients with epilepsy syndromes.

Questions
1. What is the most common $^{18}$F-FDG PET finding in patients with isolated epileptic spasms?
   
   A. Diffuse cortical hypometabolism.
   
   B. Solitary focal cortical hypometabolism.
   
   C. Bilateral temporal lobe hypometabolism.
   
   D. Multifocal cortical hypometabolism.
2. What is the main role of $^{18}$F-FDG PET in children with hemimegalencephaly, a condition with a unilateral enlarged and defectively developed hemisphere and intractable seizures?
   A. To identify the possible epileptogenic region and guide the subsequent subdural electrode placement for surgical resection.
   B. To evaluate the contralateral normal hemisphere for prognostic purposes.
   C. To perform quantitative estimation of glucose metabolic rate in the affected hemisphere to characterize the nature of cortical dysplasia, usually underlying this condition.
   D. To correlate with MR imaging abnormalities to tailor the margins of surgical resection to minimize the loss of brain function.

3. What is the most common pathology underlying unilateral focal $^{18}$F-FDG hypometabolism in epileptic spasms of unknown cause?
   A. Low-grade astrocytoma.
   B. Tuberous sclerosis.
   C. Cortical dysplasia.
   D. Mesial temporal sclerosis.

4. In patients with tuberous sclerosis complex and refractory seizures, epileptogenic tubers are most reliably imaged by which interictal imaging modality?
   A. Contrast enhancement and edema on x-ray CT.
   B. $^{18}$F-FDG hypometabolism relative to adjacent normal cerebral cortex.
   C. Increased activity after administration of $^{11}$C-alphamethyl-L-tryptophan ($^{11}$C-AMT).
   D. Increased signal intensity in fluid-attenuated inversion recovery (FLAIR) sequence proton MR imaging.

5. What feature best differentiates Lennox-Gastaut syndrome from West syndrome?
   A. $^{18}$F-FDG PET hypometabolism patterns.
   B. Presence of neurodevelopmental delay only in West syndrome.
   C. Presence of multifocal enhancing MR imaging lesions in Lennox-Gastaut syndrome.
   D. Characteristic seizure semiology and EEG findings.

6. What feature is most often associated with Sturge-Weber syndrome?
   A. An affected parent or sibling.
   B. A trigeminal cutaneous lesion.
   C. Developmentally limited (transient) seizures.
D. Normal T1-weighted MR imaging results.

7. What feature predicts the development of medically refractory seizures in Sturge-Weber syndrome?
A. The progression of EEG changes during childhood development.
B. Anatomic imaging demonstrating increased mass effect and edema.
C. Increased interictal cortical $^{18}$F-FDG uptake early in the course of the disease.
D. Progressive developmental delay and regression.

8. What is the most common indication of molecular imaging in infants and children with epilepsy syndromes?
A. Syndrome diagnosis.
B. Presurgical assessment.
C. Therapeutic response evaluation.
D. Confirmation of the ictal state.

9. Which phacomatosis has the highest incidence of refractory seizures?
A. Neurofibromatosis.
B. Von Hippel-Lindau syndrome.
C. Ataxia telangiectasia.
D. Tuberous sclerosis.

10. What is the most likely explanation for a focal cerebral origin of bilateral, clinically generalized movement manifestations of epileptic spasms of unknown etiology?
A. A thalamic epileptogenic focus.
B. Bilateral, synchronous, cerebral cortical epileptogenic foci.
C. An epileptogenic brainstem lesion.
D. Cerebral developmental immaturity.