Hemimegalencephaly

- Electrical Status Epilepticus in Sleep in a Patient with Hemimegalencephaly and Tuberous Sclerosis Complex
- Transarterial embolization for infants under 3 months of age with refractory seizures due to hemimegalencephaly: complication analysis and evolution of treatment strategy
- mTORopathies in Epilepsy and Neurodevelopmental Disorders: The Future of Therapeutics and the Role of Gene Editing
- Uncrossed Cerebellar Diaschisis in Hemimegalencephaly: Evaluated by FDG-PET and Diffusion Tensor Tractography
- Corpus Callosotomy for Atonic Drop Seizures in Bilateral Malformations of Cortical Development: A Systematic Review of Literature
- Effectiveness and Safety of High-Dose Oral Phenobarbital in Children With Recurrent and Treatment-Refractory Seizures
- The fetal neurologist: Strategies to improve training, practice, and clinical care
- A Novel RHEB Germline Variant Associated With Intellectual Disability and Epilepsy: Expanding the Spectrum of mTORopathies

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Hemimegalencephaly (HME), or unilateral megalencephaly, is a rare congenital disorder affecting all or a part of a cerebral hemisphere.

It causes severe seizures, which are often frequent and hard to control. A minority might have seizure control with medicines, but most will need removal or disconnection of the affected hemisphere as the best chance. Uncontrolled, they often cause progressive intellectual disability and brain damage and stop development.

Treatment

Hemimegalencephaly treatment.

Case reports

A case report of hemimegalencephaly with super-refractory status epilepticus and brain atrophy associated with NPRL3 gene mutation [1)]

Koboldt et al. from the Nationwide Children's Hospital, reported two somatic variants of PTEN affecting a single patient presenting with intractable epilepsy and hemimegalencephaly that varied in clinical severity throughout the left cerebral hemisphere. High-throughput sequencing analysis of affected brain tissue identified two somatic variants in PTEN. The first variant was present in multiple cell lineages throughout the entire hemisphere and associated with mild cerebral overgrowth. The second variant was restricted to posterior brain regions and affected the opposite PTEN allele, resulting in a segmental region of more severe malformation, and the only neurons in which it was

found by single-nuclei RNA-seq had a unique disease-related expression profile. This study reveals brain mosaicism of PTEN as a disease mechanism of hemimegalencephaly and furthermore demonstrates the varying effects of single- or bi-allelic disruption of PTEN on cortical phenotypes [2)].

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