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Juvenile Myoclonic Epilepsy

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Editorial

Juvenile Myoclonic Epilepsy (JME) is an epilepsy syndrome characterized by myoclonic jerks, generalized tonic-clonic seizures (GTCSs), and sometimes, absence seizures. The seizures of juvenile myoclonic epilepsy often occur when people first awaken in the morning. Doctors diagnose JME with an electroencephalogram (EEG), a test that can find unusual patterns in brain waves.

To do the test, medical team places electrodes on scalp. They're connected with wires to a computer that shows the electrical activity of brain cells. The results look like a graph of spikes and wavy lines. Doctor will scan them to look for patterns that signal JME. Juvenile myoclonic epilepsy (JME) is the most common generalized epilepsy syndrome. It is also called Juvenile Myoclonic Epilepsy of Janz. It usually is first seen in adolescence. Less commonly, it can develop in a child who has had childhood absence epilepsy. Juvenile myoclonic epilepsy is a genetically determined syndrome. Most people with JME do not have abnormal results on testing for specific epilepsy genes. The inheritance pattern is a complex type, although there are certain subtypes with distinct genetic patterns. Juvenile myoclonic epilepsy is a condition characterized by recurrent seizures.

This condition begins in childhood or adolescence, usually between ages 12 and 18, and lasts into adulthood. The most common type of seizure in people with this condition is myoclonic seizures, which cause rapid, uncontrolled muscle jerks. People with this condition may also have generalized tonic-clonic seizures, which cause convulsions, muscle rigidity and loss of consciousness. Sometimes, affected individuals have absence seizures, which cause loss of consciousness for a short period that appears as a staring spell. Typically, people with juvenile myoclonic epilepsy develop the characteristic myoclonic seizures in adolescence, then develop generalized tonic-clonic seizures a few years later.

Seizures can happen at any time, they occur most commonly in the morning, shortly after awakening. Seizures can be triggered by a lack of sleep, stress, extreme tiredness or alcohol consumption. The genetics of juvenile myoclonic epilepsy are complex and not completely understood. Mutations in one of several genes can cause or increase susceptibility to this condition. The most studied of these genes are the *GABRA1* gene and the *EFHC1* gene, although mutations in at least three other genes have been identified in people with this condition. People with juvenile myoclonic epilepsy which do not have mutations in any of these genes. Changes in other, unidentified genes are likely involved in this condition.

A mutation in the *GABRA1* gene has been identified in several members of a large family with juvenile myoclonic epilepsy. The *GABRA1* gene provides instructions for making one piece, the alpha-1 subunit, of the *GABAA* receptor protein. The *GABAA* receptor acts as a channel that allows negatively charged chlorine atoms to cross the cell membrane. After infancy, the influx of chloride ions creates an environment in the cell that inhibits signaling between nerve cells (neurons) and prevents the brain from being overloaded with too many signals. Mutations in the *GABRA1* gene lead to an altered α1 subunit and a decrease in the number of *GABAA* receptors available. As a result, the signaling between neurons is not controlled, which can lead to overstimulation of neurons. The overstimulation of certain neurons in the brain triggers the abnormal brain activity associated with seizures.

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