**Editorial**
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# Editorial on Metabolic Epilepsies

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## Editorial

When evaluating children who are having seizures, metabolic problems should always be considered. This is because many metabolic problems are theoretically curable, and when these diseases are properly treated, seizure control can be attained. In cases of unexplained neonatal seizures, refractory seizures, seizures related to fasting or food intake, seizures associated with other systemic or neurologic features, parental consanguinity, and a family history of epilepsy, seizures caused by underlying metabolic diseases (metabolic seizures) should be considered. Various amino acid metabolic illnesses, energy metabolism disorders, cofactor-related metabolic diseases, purine and pyrimidine metabolic diseases, congenital disorders of glycosylation, and lysosomal and peroxisomal disorders can all produce metabolic seizures. It's critical to diagnose metabolic seizures as soon as possible since, for many metabolic illnesses, the commencement of proper therapy can prevent or reduce problems. Although metabolic problems (inborn errors of metabolism) are a rare cause of juvenile seizures, they should always be explored when screening children who arrive with seizures. This is because many metabolic problems are theoretically curable, and when these diseases are properly treated, seizure control can be attained. For many metabolic diseases, prompt commencement of effective medication can avoid or reduce consequences; thus, early detection of these disorders is critical. Even when dealing with metabolic problems for which there is no viable treatment currently, making the diagnosis is critical for directing care regimens and providing appropriate counselling.

Seizures can be caused by more than 200 different metabolic illnesses. Epilepsy can be the primary presenting symptom of certain metabolic illnesses, or it can be part of a complex phenotype that includes other neurological and metabolic symptoms. Seizures can be caused by a variety of metabolic illnesses. The accumulation of hazardous metabolites such as ammonia can cause seizures. Hyperammonemia occurs in a variety of metabolic illnesses, including urea cycle abnormalities and organic acidemias. Ammonia buildup is harmful because it causes astrocyte swelling and brain edema by increasing glutamine production. Neurotransmission can be disrupted by some metabolic diseases. Glycine is an agonist for the glutamate receptors NMDA (N-methyl D-aspartate). Glycine encephalopathy is caused by an overstimulation of the excitatory NMDA receptors, resulting in seizures. Seizures in metabolic illnesses can potentially be caused by a lack of energy. Because glucose is the brain's main energy source, illnesses that cause hypoglycemia (e.g., fatty acid oxidation abnormalities) or hinder brain glucose transport (e.g., glucose transporter type 1 (GLUT-1) deficiency) can result in brain energy shortage, which can lead to seizures [1-3]. Mitochondrial abnormalities can also cause seizures and reduced adenosine triphosphate (ATP) synthesis. Seizures can be seen in serine deficiency, glycine encephalopathy, maple syrup urine illness, urea cycle abnormalities, organic acidemia, phenylketonuria, and sulfite oxidase deficiency, among other amino acid metabolic disorders. Seizures can be caused by energy deprivation in a variety of metabolic conditions, including mitochondrial diseases, glucose transporter type 1 (GLUT-1) deficiency, guanidinoacetate methyltransferase (GAMT) deficiency, and hypoglycemia-causing disorders including fatty acid oxidation abnormalities.

Seizures are common in pyridoxine-dependent epilepsy, pyridoxal phosphate-responsive epilepsy, early-onset vitamin B6-dependent epilepsy, cerebral folate deficiency, methylenetetrahydrofolate reductase (MTHFR) deficiency, molybdenum cofactor deficiency, biotinidase deficiency, and holocarboxylase synthetas Seizures can be caused by metabolic abnormalities in one of three ways: a lack of critical substrates for cellular metabolism or membrane function, intracellular buildup of toxic chemicals, or a change in intracellular osmolality. Biotinidase and holocarboxylase synthase deficiency, cerebral folate deficiency, creatine disorders, folinic acid responsive seizures, glucose transporter type 1 (GLUT-1) deficiency, mitochondrial disorders, peroxisomal disorders, and pyridoxine-dependent epilepsy are the eight types of metabolic epilepsies recognised by the International League Against Epilepsy (ILAE) (PDE). More forms of metabolic epilepsies have been discovered, including urea cycle abnormalities, glutaric aciduria, molybdenum cofactor insufficiency, and non-ketotic hyperglycemia [4-5].

## Conflict of Interest

None.

## References

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