



Neonatal Seizures

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Abstract

The most frequent occurrence of neurological dysfunction in the neonatal period is convulsions. Ethology determines the outcome and therapeutic approach.

Keywords: Neonatal Seizures; Etiology; Prognosis

Introduction

Neonatal attacks occur in the first seven days, and less than half of diseased neonates develop later in life.

Neonatal epilepsy is not used to describe neonatal seizures [1]. Attacks occur during excessive, synchronized neural depolarization. Depolarization occurs as a result of the release of excitatory amino acids (e.g., glutamate) or defective neurotransmitter inhibitors (e.g., Gama amino tartaric acid [GABA]) [2]. The etiology of neonatal convulsions is: hypoxic-ischemic encephalopathy, intracranial haemorrhage, hypoglycaemia, hypocalcaemia and hypomagnesemia, metabolic errors, intracranial infections (meningitis, encephalitis, toxoplasmosis and cytomegalo virus, and bacterial causes of *Escherichia coli* and *Streptococcus pneumoniae* [3].

Benign family neonatal attacks usually occur in the first 48-72 hours of life; attacks disappear at the age of 2-6 months [4,5].

The prognosis is determined by the etiology of neonatal attacks. If the EEG medium is normal, it is likely a normal development [6-8].

Severe abnormalities in the background of EEG indicate a poor prognosis; Such patients often have cerebral palsy and epilepsy [6,9].

The prognosis after neonatal seizures resulting from isolated subarachnoid bleeding is excellent, with 90% of children who have no residual neurological deficits [10].

Conclusion

Diagnosis and therapy of neonatal seizures are very important for the later development and reduction of the frequency of neurological consequences.

Competing Interests

None stated.

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