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Short Communication

Neuropathology of the Rett Syndrome in Austic Brain Development Disorders

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DESCRIPTION

Rett syndrome is a rare genetic neurological developmental syndrome which affects the brain development. This condition causes a gradual decrease in cognitive and impulse control. Rett syndrome can also cause seizures and cognitive impairments and it primarily affects females. The first six months of life remain symptomatic for the majority of infants with rett syndrome and it shows impact on neonates' capacities for crawling, walking, interacting. Studies are being conducted even though there is no curative therapy for the Rett syndrome [1].

The main objectives of contemporary treatment are to enhance movement and communication, manage seizures, and provide assistance and care to individuals with rett syndrome and their communities.

Stages of rett syndrome

Stage 1 (Early onset): Between the ages of 6 and 18 months, the initial stage is characterized by mild symptoms that are easy to avoid. Stage 1 can last for several months or perhaps a year.

Stage 2 (Rapid deterioration): Children begin to lose their previous skills and abilities between the age of 1 and 4 years old. This loss can occur immediately or more gradually over a period of weeks or months. Rett syndrome symptoms include an absence of social contact and communication, slowed reason.

Stage 3 (Late motor deterioration): After the age of 10, this stage might last a lifetime or for many years. Symptoms include decreased mobility, muscle weakness, joint contractures, and instability. Spasms may happen less frequently, but cognition, manual dexterity, and communication typically remain the same or even significantly improve [2].

Rett Syndrome is distinct from all other paediatric neurologic disorders, and its clinical-pathologic interaction cannot be characterized by conventional histology methods. Based on hypotheses provided by detailed clinical observations, the neurological system of the Rett child has been analyzed utilizing morphometry, golgi preparations, computerised tomography, magnetic resonance imaging, chemistry, immunocytochemistry, autoradiography, and molecular biologic techniques. The patient's rett brain is small and some of the lobes have even less dendritic structures in pyramidal neurons of layers III and V. (frontal, motor, and temporal) [3]. It develops microtubular protein-2 and cyclooxygenase. Microscopic neurons with expanded neuronal packing density, and has an expanding pattern of neurotransmitter receptors. While Z-filaments in skeletal muscle revealed alterations in the sarcoplasmic reticulum with circular profiles, small fibres without demyelination and a rise in the number of neurofilaments in axons and peripheral nerves suggested distant axonopathy. These indistinct changes can be seen as the first symptoms of nerve impingement. A typical female patient with a constant phenotype is produced by the clinical and biochemical significance of the numerous rett syndrome abnormalities in the central, neuroendocrine, and peripheral neuromuscular systems [4].

CONCLUSION

The treatment is based on clinical experience and is multidisciplinary. Mutations in the X-linked MECP2 gene, which encode for the MeCP2 protein, leads to rett syndrome which is a severe neurological developmental disorder. Patients with Rett syndrome initially develop normally, but after a few months, their growth drops significantly. The clinical and biochemical significance of the distinct rett syndrome abnormalities in the central, neuroendocrine, and peripheral neuromuscular systems results in patients with a variety of phenotypes.

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