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Spinocerebellar Ataxia: New Treatment Options

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Editorial

Spinocerebellar ataxias are a group of neurodegenerative illnesses that share the common feature of progressive ataxia caused by degeneration of the cerebellum and cerebellar connections, with onset in middle age and autosomal dominant inheritance. They are caused by genetic mutations that cause neurons to deteriorate and die. Forty genetically different subtypes have been discovered, defined, encoded, and designated in a stepwise manner (SCA1, SCA2, SCA3, etc.); however, only 12 of these subtypes have a causal gene. The autosomal dominant SCAs can be subcategorized into five groups: (1) CAG (polyglutamine) repeat expansion disorders within gene reading frames (SCAs 1, 2, 3, 7, 17); (2) disorders with noncoding repeats (SCAs 8, 10, 12); (3) disorders with known gene mutations which are not repeats (SCAs 5, 11, 13, 14, 15); (4) disorders with chromosomal linkage only (SCAs 4, 19, 21); and (5) autosomal dominant episodic ataxias (EAs) (E1, EA2/SCA6). Childhood is when autosomal recessive ataxias are more common. Friedreich ataxia (FA) and ataxia telangiectasia are the two most frequent autosomal recessive ataxias [1,2].

Disease progression

SCAs are all progressive conditions that frequently result in disability and death. However, both within and within SCAs, there is a great deal of variation in the rate of advancement. Several cohort studies, including the EUROSCA research, used the Scale for the Assessment and Rating of Ataxia to track the progression rates of the most common polyglutamine SCAs, SCA1, SCA2, SCA3/MJD, and SCA6, through time. SARA is a clinical scale based on a semi quantitative impairment level assessment of ataxia. SCA1 has the fastest progression rate of the most prevalent polyglutamine SCAs, SCA2 and SCA3/MJD have an intermediate progression rate, and SCA6 has the slowest progression rate, according to these research [3].

Conventional therapy

There are currently no approved pharmaceutical medications for routine usage in patients with SCAs, therefore clinical care is symptomatic and supportive for function maintenance. Physiotherapy, occupational therapy, and speech therapy are all examples of general supportive management methods. The use of physiotherapy to improve gait, balance, coordination, posture, and muscle strength is frequently suggested. Traditional physiotherapy exercises, computer-assisted training, treadmill training, and biofeedback therapy are all possible interventions. In children and young people with ataxia, research have shown that video games or computer-assisted training has a good effect. The importance of maintaining biomechanical alignment throughout the healing process cannot be overstated. Orthopedic disorders including foot abnormalities and scoliosis are commonly treated with orthotics or surgery, which can provide a temporary improvement in function. Early treatment for biomechanical abnormalities in the foot improves alignment and, as a result, weightbearing ability and mobility. Daily attention to range of motion, including muscle length and soft-tissue extensibility, should be part of the treatment strategy. A home fitness regimen and family instruction for follow-through would most likely be part of this plan of care [4,5].

Conclusion

In-depth examination of such aspects in distinct SCA subtypes can help manage patients' quality of life, and in the interim, these same qualities could be prospectively employed as a possible preclinical marker of the disease. Although the hunt for symptomatic and disease-modifying medicines for SCAs has generated no clear winners, recent developments in our understanding of the disease mechanisms point to promising options for both symptomatic and disease-modifying therapy.

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Conflict of Interest

The authors reported no potential conflict of interest.

References

1. Winchester, Sara, Piyush K. Singh, and Mohamad A. Mikati. "Ataxia." *Handb Clin Neurol* 112 (2013): 1213-1217.
2. Rüb, Udo, Kay Seidel, Inci Özerden, Kristin Gierga and Ewout R. Brunt, et al. "Consistent affection of the central somatosensory system in spinocerebellar ataxia type 2 and type 3 and its significance for clinical symptoms and rehabilitative therapy." *Brain Res Rev* 53 (2007): 235-249.
3. Gandini, J., Mario Manto, Tatiana Brémovà-Ertl and K. Feil, et al. "The neurological update: Therapies for cerebellar ataxias in 2020." *J Neurol* 267 (2020): 1211-1220.
4. Aranca, Tanya V., Tracy M. Jones, Jessica D. Shaw and Joseph S. Staffetti, et al. "Emerging therapies in Friedreich's ataxia." *Neurodegener Dis Manag* 6 (2016): 49-65.
5. Egorova, P.A and I.B. Bezprozvanny. "New Approaches in studies of the molecular pathogenesis of type 2 spinocerebellar ataxia." *Neurosci Behav Physiol* 50 (2020): 938-951.

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