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Diagnosis and Clinical Trials Management Of Huntington's Neurodegenerative Disease

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Perspective

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DESCRIPTION

A neurological condition that is primarily inherited is Huntington's Disease (HD), often known as Huntington's chorea. Insidious issues with mood or cognitive abilities are frequently the first indications. A general loss of coordination and a shaky walk frequently come next. It is a basal ganglia dysfunction that also causes chorea, a hyperkinetic movement disorder. The chorea's clumsy, uncontrollable motions become more noticeable as the illness worsens. The person's physical abilities steadily deteriorate until it is impossible for them to move with coordination and they are unable to speak. Dementia typically results in a deterioration in mental ability. Each individual has slightly different symptoms. Although they can appear at any age, symptoms typically appear between the ages of 30 and 50. With each new generation, the disease can progress more quickly. 8% of cases that begin before the age of 20, typically presents with chorea-like symptoms rather than the slow movement symptoms of Parkinson's disease.

The final stages of HD necessitate full-time care, and there is currently no recognized cure. Some treatments can reduce symptoms, while others can enhance quality of life. Tetrabenazine offers the finest support for treating mobility issues. Around 4 to 15 persons of European ancestry experience HD. It is uncommon among Japanese people, and its frequency in Africa is unreported. Men and women are both affected equally by the illness. Life expectancy is shortened by complications like pneumonia, heart disease, and bodily harm from falls. In around 9% of cases, suicide is the cause of death. Mortality usually happens 15 to 20 years after the disease was first discovered.

Diagnosis of the commencement of HD can be made following the manifestation of physical signs related to the condition. If there is no family history of HD, genetic testing can be used to confirm a physical diagnosis. Genetic testing can establish whether a person or an embryo possesses an enlarged copy of the Trinucleotide Repeat (CAG) in the HTT gene that causes the illness even before symptoms appear. There is genetic counselling available to offer suggestions and direction during the testing process and regarding the implications of a confirmed diagnosis. These repercussions include the effect on a person's psychology, career, and decisions about starting a family, relatives, and relationships. Despite the availability of pre-symptomatic testing, only 5% of persons at risk of inheriting HD opt to do so.

Families and the general public who have inherited or are at risk of inheriting HD have dealt with the condition for generations, but they might not be aware of current advances in medical knowledge or the availability of genetic testing. The benefits of genetic counselling for these people include updating their knowledge, attempting to debunk any erroneous views they may have, and assisting them in thinking through their alternatives and future goals. To assist in educating family members, careers, and those who have been diagnosed with Huntington's disease, the Patient Education Program for Huntington's Disease was established. Information on options for family planning, care management, and other factors is also provided.

Treatments are available to reduce the severity of some of HD symptoms. There is insufficient evidence to support many of these treatments' ability to specifically treat HD symptoms. As the illness worsens, the capacity for self-care decreases, necessitating more and more judicious multidisciplinary caregiving. There is some evidence that physical therapy, occupational therapy, and speech therapy are effective in treating cognitive symptoms of HD, despite the fact that relatively few research have examined these treatments.

There were 197 clinical studies for various treatments and biomarkers for Huntington's disease that were either in progress, recruiting participants, or had just been finished as of the year 2020. Remacemide, coenzyme Q10, riluzole, creatine, minocycline, ethyl-EPA, phenylbutyrate, and dimebon are some of the compounds that have been tried but failed to stop or halt the progression of Huntington's disease.