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Huntington's Disease: Pathophysiology, Clinical Features, and Emerging Therapeutic Approaches

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Abstract: *Huntington's disease (HD) is an inherited neurodegenerative disorder that gradually affects movement, thinking, and emotional well-being. It is caused by an abnormal expansion of CAG repeats in the huntingtin (Htt) gene, producing a mutant form of the protein (mHtt) that misfolds and interferes with normal cellular functions. These misfolded proteins, along with other abnormal sequences like polyalanine and polyserine, contribute to the progressive loss of neurons in key brain regions. Diagnosis of HD combines clinical evaluation, neurological examination, and imaging, but genetic testing, including analysis of CAG repeats, provides a definitive confirmation. Prenatal testing is also available for at-risk individuals. Clinically, patients show a mix of involuntary movements (chorea), slowed thinking, memory problems, and psychiatric symptoms such as anxiety, depression, and irritability. Recent advances in therapy include drug-based symptom management, non-drug interventions, and promising approaches like stem cell therapy aimed at replacing damaged neurons and supporting brain repair. Nanoparticle-based treatments are being explored, although challenges like toxicity and immune reactions remain. Additionally, natural herbs such as Bacopa monnieri (BM) and Curcuma longa (CL) offer antioxidant and neuroprotective benefits. Overall, a combined approach of medications, therapies, natural compounds, and multidisciplinary care is essential to improve quality of life and slow disease progression in HD.*

Keywords: Pathogenesis, Polyglutamine, Nanoparticles, mHtt protein, Neuroinflammation

I. INTRODUCTION

Huntington's disease (HD) is a hereditary neurodegenerative disorder marked by progressive motor, cognitive, and psychiatric impairments that significantly affect an individual's quality of life. The condition results from mutations in the huntingtin (HTT) gene, which lead to expanded CAG repeats encoding abnormal polyglutamine sequences. Misfolded mutant huntingtin protein (mHtt) disrupts normal cellular processes, forms toxic aggregates, and promotes neuronal degeneration, particularly in the striatum and other critical brain regions. (1) Additional abnormal polypeptides, such as polyalanine, polyserine, polyleucine, and polycysteine, further contribute to neuronal damage, emphasizing the complex pathogenic mechanisms underlying polyglutamine diseases.(2)

Accurate and timely diagnosis of HD involves a combination of clinical assessment, neuroimaging, and genetic testing. Clinical evaluation includes detailed neurological and cognitive examinations, while imaging can reveal structural brain changes, such as striatal atrophy and ventricular enlargement. Definitive confirmation is achieved through genetic testing that quantifies CAG repeat expansions in the HTT gene, providing insight into disease risk and guiding counseling.(3)

Therapeutic strategies for HD are evolving and encompass both pharmacological and non-pharmacological interventions. (4) Conventional treatments aim to manage motor and psychiatric symptoms, while non-drug-based therapies, including physiotherapy and occupational therapy, enhance daily functioning. Recent advances explore stem

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cell-based therapies to replace damaged neurons and support neuroprotection. Nanoparticle-mediated drug delivery offers targeted therapeutic potential but faces challenges such as toxicity and immunological responses. Complementary approaches using herbal compounds, such as Bacopa monnieri and Curcuma longa, demonstrate antioxidant and neuroprotective benefits, mitigating oxidative stress and supporting neuronal survival. (5) These multifaceted strategies reflect the comprehensive efforts to understand, manage, and potentially modify the course of HD.

Pathophysiology of Huntington's Disease: Huntington's disease (HD) is a progressive neurodegenerative disorder primarily marked by the loss of neurons in the striatum, particularly affecting medium spiny neurons (MSNs). This neuronal degeneration disrupts the normal functioning of critical neurotransmitter systems, including dopamine, glutamate, and gamma-aminobutyric acid (GABA), which play central roles in motor control, cognition, and behaviour. The imbalance of these neurotransmitters contributes significantly to the hallmark features of HD, such as involuntary movements (chorea), motor dysfunction, cerebellar abnormalities, and the toxic effects of mutated huntingtin protein. Pharmacological treatments for HD are largely aimed at modulating these altered neurotransmitter pathways to alleviate symptoms and improve quality of life. (6)

Beyond neural degeneration, metabolic dysregulation is increasingly recognized as a key factor in HD pathogenesis. Patients often experience unexplained weight loss despite adequate or even increased caloric intake, indicating a systemic metabolic imbalance. Studies have reported abnormal levels of aliphatic amino acids and fatty acids in the blood of HD patients, suggesting disruptions in protein and lipid metabolism. Additionally, post-mortem analyses of HD brains have revealed elevated concentrations of urea, highlighting potential alterations in nitrogen metabolism. These metabolic changes contribute not only to the physical decline seen in HD but also to the progressive deterioration of neural function. (7)

Physiologically, HD manifests through a variety of symptoms that affect both the nervous system and overall well-being. Patients may exhibit heightened nervous activity, restlessness, fatigue, anxiety, and general exhaustion. Motor abnormalities, such as involuntary movements, impaired coordination, and balance issues, further complicate daily functioning and increase the risk of falls. These physical impairments, combined with cognitive and psychiatric challenges, often lead to sleep disturbances, weight loss, and difficulties with routine activities, underscoring the multifaceted impact of the disease. (8)

Genetic factors play a pivotal role in the onset and progression of HD. Offspring of an HD-affected parent have a 50% risk of inheriting the mutated gene. Predictive genetic testing serves as an essential diagnostic tool for at-risk individuals, even before clinical symptoms appear. By analyzing the DNA for abnormal expansions of the CAG trinucleotide repeat in the HTT gene, clinicians can identify carriers and provide guidance on disease management and monitoring. This approach, combining clinical observation with genetic analysis, allows for early detection and informed decision-making regarding lifestyle, family planning, and therapeutic interventions. (9) Overall, HD represents a complex interplay of neuronal degeneration, neurotransmitter dysfunction, metabolic abnormalities, and genetic predisposition. Understanding these mechanisms is crucial for developing effective therapeutic strategies and providing comprehensive care for affected individuals.

Pathogenic Mechanism of Polyglutamine Diseases:

Huntington's disease (HD) is caused by a mutation in the huntingtin (Htt) protein, where repeated CAG sequences lead to an abnormally long stretch of glutamine near the protein's amino-terminal end. Huntingtin is essential for normal cellular functions, including metabolism and neuronal maintenance. When the protein misfolds due to this mutation, it loses its normal function and disrupts important cellular processes. The small fragments of the protein often seen in HD are not the original cause of damage—they are the byproducts of faulty protein processing within cells.(10)

Removing the huntingtin gene in adult organisms does not cause obvious physical changes, suggesting that its critical functions are most important during development. However, if the gene is completely knocked out in embryos, it leads to severe defects or death, highlighting its essential role in early growth. Overall, HD reflects a delicate balance between the normal functions of huntingtin and the toxic effects of its misfolded forms, explaining why neurons gradually degenerate in affected individuals.(11)

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Diagnostic test types:

Huntington's disease (HD) is an inherited genetic disorder that can be identified using three main types of diagnostic approaches. Type I focuses on clinical evaluation, which includes a thorough collection of the patient's medical and family history, as well as a detailed neurological examination. (12) Physicians may look for subtle motor, cognitive, or psychiatric signs during this assessment. If these findings are inconclusive, Type II involves diagnostic imaging techniques, such as MRI or CT scans, which can reveal characteristic brain changes, including atrophy of the striatum and enlargement of ventricles.(13)

Type III is genetic testing, which provides the most definitive diagnosis. Direct analysis of the HD gene determines the number of CAG trinucleotide repeats, confirming whether an individual carries the mutation and their risk of developing the disorder. Prenatal exclusion testing can also assess the presence of the mutation in unborn children. Individuals at risk of HD often undergo testing before major life decisions, such as marriage or career planning, due to the significant emotional impact of the results. (14)

Clinical Aspects:

The of Huntington's disease (HD) is typically divided into three stages: early, middle, and late, with each stage marked by a combination of motor, cognitive, and emotional disturbances. Motor symptoms are among the most noticeable features and include involuntary movements, abnormal voluntary movements, and the hallmark chorea—rapid, jerky, and progression unpredictable motions that affect the limbs, face, and trunk. Cognitive impairments also develop as the disease progresses. Patients may experience bradyphrenia (slowed thinking), difficulties with memory recall, impaired planning and problem-solving, and noticeable changes in personality, including reduced initiative or social withdrawal. (15)

Emotional and psychiatric disturbances are common, significantly impacting quality of life. Affected individuals often experience depression, anxiety, irritability, aggression, and impulsive behaviours. In some cases, the emotional burden may lead to suicidal thoughts or actions. These combined motor, cognitive, and psychiatric symptoms underscore the multifaceted and progressive nature of HD, requiring comprehensive management strategies throughout the disease course.(16)

Recent Developments Therapy in Huntington's disease:

Several natural compounds and plant-derived extracts have demonstrated potential in managing Huntington's disease (HD). Bioactive molecules such as resveratrol and melatonin have been investigated for their neuroprotective properties, helping to mitigate oxidative stress and neuronal damage associated with HD.(17) Combinations of cannabidiol, quinidine, and thiamine have also shown promise in modulating disease progression by supporting cellular health and neurotransmitter balance. In addition, certain plant extracts, including Calendula officinalis and Celastrus paniculatus, exhibit significant antioxidant activity, which may protect neurons from oxidative damage, a key factor in HD pathology. Another therapeutic strategy involves promoting the autophagic degradation of the mutated huntingtin (mHTT) protein, which accumulates and contributes to neuronal dysfunction in HD. Compounds like gossypol have been studied for their ability to enhance this autophagic clearance, potentially reducing the toxic effects of mHTT. Together, these natural and plant-based interventions offer complementary approaches for supporting neuronal health and slowing HD progression. (18)

Therapy Based on Stem Cells:

Stem cell-based therapies are emerging as a promising approach for the treatment of Huntington's disease (HD), focusing on alleviating symptoms and potentially modifying disease progression. These therapies aim to replace neurons that have been lost due to the neurodegenerative processes characteristic of HD. By introducing healthy stem cells into affected regions of the brain, it is possible to restore neuronal populations, enhance neurogenesis, and support the survival of existing neurons, thereby slowing further neural deterioration. (19)

Various types of stem cells have been investigated for their therapeutic potential in HD. Mesenchymal stem cells (MSCs) are valued for their regenerative properties and ability to secrete neurotrophic factors that support neuronal

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health. Embryonic stem cells (ESCs) offer the advantage of pluripotency, allowing them to differentiate into multiple neural cell types necessary for repair. (20) Neural stem cells (NSCs), inherently committed to the neural lineage, can integrate into brain tissue and contribute to neuronal replacement. Additionally, induced pluripotent stem cells (iPSCs) derived from patient cells provide a personalized approach, enabling the generation of patient-specific neurons for transplantation while minimizing immune rejection risks.(21)

Challenges in Nanoparticle-Based Treatments:

Nanoparticles are tiny particles that hold great promise for delivering drugs precisely where they are needed in the body. Their small size even allows them to cross the blood-brain barrier, making them attractive for treating neurological conditions. (22) However, this same ability can become a problem. If nanoparticles accumulate in the brain while the intended target is another organ, they can potentially cause neurotoxicity. Similarly, nanoparticles can trigger inflammation in organs like the liver, lungs, and brain, mainly through oxidative stress, which can damage tissues and reduce the effectiveness of the treatment.

Another concern is how nanoparticles interact with the immune system. They can sometimes alter immune responses in unpredictable ways, which may complicate treatment or trigger side effects. To address these issues, researchers have developed stimuli-responsive nanoparticles that release their drug payload only in response to specific triggers, such as changes in pH, temperature, or light. While this approach is promising, it still faces challenges in terms of precision and consistency, meaning more research is needed before it can be widely used. (23)

Management guidelines:

Huntington's disease (HD) is a complex condition that requires a combination of pharmacological and non-pharmacological approaches to manage symptoms effectively and enhance quality of life. (24) Medications are commonly used to address behavioural and motor symptoms. Antipsychotic drugs such as olanzapine, risperidone, quetiapine, clozapine, and aripiprazole are frequently prescribed to help control agitation, psychosis, and chorea. Mood disturbances are often managed with anticonvulsants, including sodium valproate, lamotrigine, and carbamazepine, which act as mood stabilizers.

In addition to drug therapy, non-drug-based interventions play a critical role in improving functional abilities and overall well-being. Physiotherapy helps maintain mobility and balance, while occupational therapy supports the performance of daily activities. (25) Speech and language therapy can address communication difficulties and swallowing problems, and structured exercise programs can enhance physical and mental health. These non-pharmacological measures are often more effective than medications alone in preserving independence and slowing functional decline.

Optimal management of HD requires care from a multidisciplinary team of healthcare professionals. Patients benefit from referral to specialist HD clinics where experienced practitioners, including neurologists, geneticists, psychiatrists, physiotherapists, occupational therapists, speech and language therapists, dietitians, social workers, and community mental health teams, work collaboratively to provide comprehensive care. (26)

As the disease progresses, complications such as dysphagia become common. Professional guidance on diet and nutrition is essential for patients and caregivers to ensure safety and adequate nourishment. By combining medications, therapeutic interventions, and expert multidisciplinary support, patients with HD can achieve better symptom control, maintain functionality, and improve their quality of life throughout the disease course. (27)

Herbs and phytoconstituents' protective benefits in HD:

Bacopa monnieri (BM): Bacopa monnieri, widely known as Brahmi, is a traditional medicinal herb used for enhancing memory and managing conditions such as epilepsy, insomnia, and anxiety. Its key bioactive compounds include dammarane-type triterpenoid saponins, Bacosides A and B, along with other saponins. (28) Research has shown that BM exerts significant neuroprotective effects, particularly in experimental models of neuronal damage like 3-nitropropionic acid (3-NP)-induced impairment, which simulates aspects of Huntington's disease. BM works through multiple mechanisms, including chelation of metal ions, scavenging of free radicals, and boosting endogenous

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antioxidant enzyme activity. Studies using BM leaf powder have demonstrated a reduction in oxidative stress markers and an increase in thiol-based antioxidant molecules, confirming its potent antioxidant capacity and ability to preserve neuronal health.(29)

Curcuma longa (CL):

Curcuma longa, also called turmeric or Haldi, is a perennial herb known for its diverse therapeutic properties. It exhibits antioxidant, anti-inflammatory, antibacterial, antifungal, antiparasitic, hepatoprotective, analgesic, antiviral, and anti-mutagenic activities.(30) Its neuroprotective effects are primarily attributed to its antioxidant action, including the inhibition of cyclooxygenase-2 (COX-2) and scavenging of reactive oxygen species, which help reduce neural inflammation and oxidative stress. Studies have demonstrated that CL can safeguard neurons in various neurological conditions, making it a promising natural agent for maintaining brain health. (31) Its combination of safety, multifunctional activity, and neuroprotective potential makes CL a valuable complementary therapy for mitigating oxidative damage and supporting neuronal function.

II. CONCLUSION

Huntington's disease (HD) is a complex brain disorder that gradually affects movement, thinking, and emotions. It is caused by the toxic effects of mutant huntingtin proteins and polyglutamine expansions, which damage neurons over time. Diagnosis combines clinical assessments, brain imaging, and genetic testing for accurate detection. Advances in therapy include conventional medications, stem cell-based approaches, and nanoparticle-assisted drug delivery, though challenges like safety and targeting remain. Effective management relies on a multidisciplinary approach, involving doctors, therapists, dietitians, and caregivers to support physical, cognitive, and emotional health. Additionally, natural remedies such as Bacopa monnieri and Curcuma longa provide antioxidant and neuroprotective benefits, helping to protect brain cells. By combining modern medical treatments, supportive care, and herbal interventions, patients can experience better symptom control, slower disease progression, and improved overall quality of life.

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