ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 2| February2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

CHRONIC NEURODEGENERATIVE DISORDERS: A REVIEW OF APPLIED ANATOMY, STRUCTURAL CHANGES, AND AYURVEDIC CORRELATIONS

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Article DOI: https://doi.org/10.36713/epra26200
DOI No: 10.36713/epra26200

ABSTRACT

Chronic Neurodegenerative Disorders (CNDs) represent a progressive and irreversible group of neurological conditions characterized by sustained neuronal injury, synaptic dysfunction, and gradual structural degeneration of the central and peripheral nervous systems, ultimately leading to cognitive, motor, sensory, and autonomic impairment. The nervous system's unique microanatomy-comprising neurons, glial cells, synapses, myelinated and unmyelinated fibers, and specialized neurovascular units-undergoes sequential structural remodeling during the course of chronic neurodegeneration. Early alterations predominantly involve functional and ultrastructural changes at the neuronal and synaptic level, including mitochondrial dysfunction, oxidative stress, impaired axonal transport, and synaptic loss, while advanced stages are marked by neuronal death, gliosis, demyelination, and gross anatomical atrophy of specific brain and spinal cord regions.

Understanding the applied anatomy of neurons and their relationships with astrocytes, oligodendrocytes, microglia, and cerebral vasculature is essential for correlating structural damage with clinical manifestations, biochemical abnormalities, and neuroimaging findings in chronic neurodegenerative disorders. This review analyzes the applied neuroanatomy with emphasis on neuron-centered structural changes observed in chronic neurodegeneration, integrating evidence from neuropathology, imaging studies, and contemporary neurological literature. Structural alterations at the cellular, tract, and regional levels are correlated with functional consequences such as cognitive decline, movement disorders, sensory deficits, autonomic dysfunction, and behavioral changes. Radiological features including cortical and subcortical atrophy, ventricular enlargement, white matter changes, and regional signal alterations reflect underlying neuronal loss and progressive architectural disintegration. An integrated understanding of neuroanatomy, histology, and imaging provides a comprehensive framework for early diagnosis, disease classification, prognostication, and effective management of Chronic Neurodegenerative Disorders.

KEY WORDS: *Neurodegeneration, Neuronal Loss, Applied Neuroanatomy, Vata Vyadhi, Asthi–Majja Dhatu*

INTRODUCTION

Chronic Neurodegenerative Disorders (CNDs) encompass a heterogeneous group of progressive neurological diseases characterized by persistent neuronal injury, synaptic failure, and maladaptive repair responses that ultimately result in irreversible loss of neural tissue and functional decline. These disorders constitute a major global health burden, contributing significantly to long-term disability, reduced quality of life, and increased healthcare expenditure. Common examples include Alzheimer's disease, Parkinson's disease, motor neuron disease, Huntington's disease, and chronic demyelinating disorders. The disease process typically evolves over years or decades, during which subtle functional impairments precede overt structural degeneration of neural tissue.[1]

The nervous system is a highly specialized and complex organ system with precise anatomical and functional organization. Neurons serve as the primary functional units, supported by glial cells that regulate metabolism, insulation, immune surveillance, and synaptic homeostasis. Neural circuits are organized into cortical layers, nuclei, tracts, and networks that allow integration of sensory input, motor output, cognition, and autonomic regulation. Disruption of neuronal structure or connectivity inevitably compromises these finely coordinated processes, highlighting the intimate relationship between neuroanatomy and neurological function.[2]

In chronic neurodegenerative disorders, neuronal injury initially manifests at the molecular and subcellular level. Early adaptive and maladaptive changes include mitochondrial dysfunction, accumulation of misfolded proteins, oxidative stress, impaired axonal transport, and synaptic degeneration. These alterations precede overt neuronal loss and represent a critical window during which functional impairment may be partially reversible. Persistent injury, however, triggers neuroinflammation mediated by microglial activation and astrocytic response, leading to progressive neuronal apoptosis or necrosis.[3]

Progressive neuronal loss disrupts the normal anatomical organization of the nervous system. Region-specific

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vulnerability results in characteristic patterns of degeneration, such as cortical thinning in dementias, basal ganglia involvement in movement disorders, and corticospinal tract degeneration in motor neuron diseases. Reactive gliosis and loss of synaptic density further distort neural circuitry, impair neurotransmission, and reduce neural plasticity. This structural remodeling forms the anatomical basis for progressive cognitive decline, motor dysfunction, and behavioral abnormalities.[4]

Structural alterations at the neuronal and network level directly correlate with the clinical and biochemical manifestations of chronic neurodegenerative disorders. Loss of functional neurons results in neurotransmitter deficiencies, impaired signal conduction, and network disintegration, leading to memory loss, bradykinesia, tremors, weakness, spasticity, and autonomic instability. These functional deficits reflect the cumulative impact of neuronal degeneration, synaptic failure, and glial dysfunction.[5]

Neuroimaging plays a pivotal role in visualizing the macroscopic consequences of chronic neurodegeneration. Magnetic resonance imaging, computed tomography, and advanced functional imaging techniques commonly demonstrate cortical and subcortical atrophy, ventricular dilatation, white matter degeneration, and region-specific signal changes. These imaging features correlate closely with underlying neuropathological changes such as neuronal loss, demyelination, and gliosis, emphasizing the applied anatomical basis of radiological diagnosis in chronic neurodegenerative disorders.[6]

Thus, Chronic Neurodegenerative Disorders represent a continuum in which progressive neuronal injury and maladaptive responses lead to irreversible structural remodeling of the nervous system. A comprehensive understanding of neuronal anatomy and its pathological alterations is essential for correlating clinical features, laboratory findings, and imaging characteristics. The present article aims to review neuron-centered structural changes in Chronic Neurodegenerative Disorders and to establish clear anatomical–functional correlations relevant to diagnosis, classification, and management.

APPLIED ANATOMY OF THE NERVOUS SYSTEM

The nervous system is a highly specialized and complex organ system responsible for coordination, integration, and regulation of all bodily functions. It is anatomically divided into the central nervous system (CNS), comprising the brain and spinal cord, and the peripheral nervous system (PNS), consisting of cranial and spinal nerves. The CNS is enclosed within the rigid confines of the skull and vertebral canal, providing mechanical protection but limiting compensatory expansion during pathological processes. This unique anatomical organization renders neural tissue particularly vulnerable to chronic metabolic, toxic, ischemic, inflammatory, and degenerative insults. An understanding of the applied anatomy of the nervous system is essential for appreciating the structural vulnerability of neurons in Chronic Neurodegenerative Disorders, wherein sustained injury leads to irreversible architectural degeneration.[7]

Gross Anatomy

In adults, the human brain weighs approximately 1.3–1.4 kg and is divided into the cerebrum, cerebellum, and brainstem, each with distinct structural and functional roles. The cerebrum is further organized into lobes, gyri, sulci, and deep nuclei, while the spinal cord is arranged into segmental levels giving rise to spinal nerves. Functional organization is further refined into cortical areas, subcortical nuclei, ascending and descending tracts, and neural networks. Preservation of this macroscopic anatomical organization is essential for normal cognition, motor control, sensory processing, and autonomic regulation.[8]

In Chronic Neurodegenerative Disorders, gross anatomical changes include regional brain atrophy, cortical thinning, ventricular dilatation, and loss of normal sulcal–gyral patterns. These alterations reflect progressive neuronal loss, synaptic degeneration, and gliosis and are commonly demonstrated on neuroimaging in advanced disease stages.[9]

Neuron: Structural and Functional Unit

The neuron represents the fundamental structural and functional unit of the nervous system. Each neuron consists of a cell body, dendrites, and an axon, enabling reception, integration, and transmission of electrical signals. Neurons are supported by glial cells, which provide metabolic support, insulation, immune surveillance, and maintenance of the extracellular environment. The precise anatomical organization of neurons into circuits and networks allows efficient neural communication and plasticity.[10]

In chronic neurodegenerative conditions, sustained neuronal stress disrupts axonal transport, synaptic integrity, and intracellular signaling. Progressive degeneration of neurons and their connections leads to disintegration of neural networks, resulting in functional decline and irreversible neurological deficits.[7,8]

Cerebral Cortex and Subcortical Structures

The cerebral cortex is composed of six histologically distinct layers, each containing specific neuronal populations involved in sensory perception, motor planning, cognition, and behavior. Subcortical structures such as the basal ganglia, thalamus, hippocampus, and brainstem nuclei play critical roles in movement regulation, memory, emotion, and autonomic control. The integrity of these interconnected regions is vital for coordinated neurological function.[10]

In Chronic Neurodegenerative Disorders, selective vulnerability of cortical and subcortical regions leads to disease-specific patterns of degeneration. Cortical neuron loss and synaptic depletion contribute to cognitive decline and behavioral disturbances, while degeneration of basal ganglia and brainstem nuclei results in movement disorders and autonomic dysfunction.[11]

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Neurons and Synapses
Neurons are highly specialized, metabolically active cells with abundant mitochondria and complex cytoskeletal architecture. Synapses serve as specialized junctions enabling chemical and electrical communication between neurons. Structural integrity of synapses is essential for learning, memory, and neural plasticity.[12]

In chronic neurodegeneration, neurons exhibit structural alterations including dendritic spine loss, cytoskeletal disruption, mitochondrial dysfunction, accumulation of misfolded proteins, and eventual apoptosis. Synaptic loss often precedes neuronal death and correlates strongly with early clinical manifestations such as cognitive impairment and motor incoordination. These structural changes form the anatomical basis for progressive neurological disability.[13]

Neuroglia and Neurovascular Unit
Neuroglial cells, including astrocytes, oligodendrocytes, and microglia, play a crucial role in maintaining neuronal homeostasis. Astrocytes regulate neurotransmitter metabolism and blood–brain barrier integrity, oligodendrocytes provide myelination, and microglia function as resident immune cells. The neurovascular unit integrates neurons, glia, and cerebral vasculature to maintain metabolic and vascular homeostasis.[14]

In Chronic Neurodegenerative Disorders, glial activation leads to chronic neuroinflammation, demyelination, and disruption of the blood–brain barrier. These changes impair neuronal survival, exacerbate oxidative stress, and contribute to progressive neural tissue loss. Microvascular alterations further compromise oxygen and nutrient delivery, accelerating neurodegeneration.[15]

White Matter Tracts
White matter consists of myelinated axonal tracts that facilitate rapid communication between different regions of the nervous system. Integrity of myelin and axonal architecture is essential for efficient signal conduction and neural synchronization.[16]

In chronic neurodegenerative disorders, white matter degeneration manifests as demyelination, axonal loss, and gliosis. These changes disrupt neural connectivity and contribute to motor weakness, sensory deficits, and cognitive dysfunction. Progressive white matter involvement reflects widespread structural disintegration of the nervous system.[17]

CHRONIC NEURODEGENERATIVE DISORDERS: DEFINITION, MAJOR CAUSES, AND TYPES

Chronic Neurodegenerative Disorders (CNDs) refer to a spectrum of progressive neurological conditions characterized by persistent neuronal dysfunction, gradual neuronal loss, and maladaptive repair responses extending over months to years, ultimately resulting in irreversible structural degeneration of the nervous system. These disorders represent a dynamic and continuous pathological process in which repeated or sustained injury to neurons and supporting glial cells leads to progressive disruption of neural circuits and functional decline. Chronic neurodegenerative disorders are a major cause of global morbidity and mortality, contributing significantly to long-term disability and dependence worldwide.[18]

From an anatomical and pathological perspective, CNDs are defined not merely by duration but by the presence of irreversible structural changes involving neurons, synapses, myelin, neuroglial cells, and the neurovascular unit. Early stages are dominated by functional synaptic alterations and molecular abnormalities, whereas advanced stages are characterized by neuronal loss, gliosis, demyelination, tract degeneration, and gross regional atrophy of the brain and spinal cord.[19]

Major Causes of Chronic Neurodegenerative Disorders
Chronic neurodegenerative disorders arise from a wide range of etiological factors that induce persistent neuronal injury through genetic, metabolic, toxic, inflammatory, vascular, or protein-misfolding mechanisms. Despite differing initiating insults, these disorders converge on common pathogenic pathways involving mitochondrial dysfunction, oxidative stress, impaired proteostasis, neuroinflammation, and progressive neuronal loss.[20]

Protein Misfolding and Aggregation Disorders
Abnormal accumulation of misfolded proteins is a central mechanism in many chronic neurodegenerative disorders. Aggregates such as amyloid-β, tau, α-synuclein, huntingtin, and TDP-43 disrupt neuronal structure and synaptic function, impair axonal transport, and trigger neuronal apoptosis. These proteinopathies form the pathological hallmark of disorders such as Alzheimer's disease, Parkinson's disease, Huntington's disease, and amyotrophic lateral sclerosis.[21]

Genetic and Hereditary Factors
Several chronic neurodegenerative disorders have a strong genetic basis, either as monogenic conditions or through polygenic susceptibility. Mutations affecting protein folding, mitochondrial function, synaptic integrity, or axonal transport predispose neurons to progressive degeneration. Even in sporadic cases, genetic vulnerability influences disease onset, progression, and regional neuronal susceptibility.[22]

Mitochondrial Dysfunction and Oxidative Stress
Neurons are highly energy-dependent cells, and chronic mitochondrial dysfunction leads to impaired ATP production, increased reactive oxygen species generation, and failure of cellular repair mechanisms. Oxidative stress damages neuronal membranes, cytoskeletal proteins, and DNA, accelerating neuronal degeneration and synaptic loss across multiple neurodegenerative conditions.[23]

Neuroinflammation and Immune-Mediated Injury
Chronic activation of microglia and astrocytes results in sustained neuroinflammation, release of pro-inflammatory cytokines, and excitotoxic damage to neurons. While initially protective, prolonged neuroinflammatory responses contribute to progressive neuronal loss and structural disintegration of neural tissue.[24]

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Vascular and Metabolic Factors
Chronic cerebral hypoperfusion, microvascular disease, and metabolic disorders such as diabetes and dyslipidemia contribute to neuronal vulnerability and accelerate neurodegeneration. Vascular insufficiency impairs oxygen and nutrient delivery, disrupts the blood–brain barrier, and exacerbates neuroinflammatory processes.[25]

Types of Chronic Neurodegenerative Disorders
Based on predominant clinical features, anatomical involvement, and pathological mechanisms, chronic neurodegenerative disorders can be broadly classified into distinct categories. This classification aids in understanding disease mechanisms, predicting progression, and guiding diagnostic and therapeutic strategies.[26]

Neurodegenerative Dementias
These disorders are characterized by progressive cognitive decline, memory impairment, and behavioral disturbances due to cortical and hippocampal neuronal loss. Alzheimer's disease is the most common example, marked by widespread cortical atrophy, synaptic loss, and accumulation of amyloid and tau pathology.[27]

Movement Disorders
Movement-predominant neurodegenerative disorders primarily involve basal ganglia, cerebellar, or brainstem structures. Parkinson's disease is characterized by degeneration of dopaminergic neurons in the substantia nigra, leading to tremor, rigidity, bradykinesia, and postural instability.[28]

Motor Neuron Disorders
Motor neuron diseases involve progressive degeneration of upper and lower motor neurons, resulting in muscle weakness, atrophy, spasticity, and eventual respiratory failure. Amyotrophic lateral sclerosis represents the most common and severe form in this group.[29]

Demyelinating and White Matter Degenerative Disorders
These disorders are characterized by progressive loss of myelin and axonal integrity within central or peripheral nervous system white matter. Structural disintegration of myelinated tracts leads to impaired signal conduction, sensory deficits, motor dysfunction, and cognitive impairment.[30]

Compensated and Advanced Neurodegenerative States
Clinically, chronic neurodegenerative disorders may be described as early or compensated stages-where functional reserve and neural plasticity partially preserve neurological function-and advanced or decompensated stages, marked by severe disability, loss of independence, and widespread structural degeneration of neural tissue.[31]

STRUCTURAL CHANGES IN CHRONIC NEURODEGENERATIVE DISORDERS

Chronic Neurodegenerative Disorders (CNDs) are characterized by progressive and largely irreversible structural alterations involving neurons, synapses, neuroglial cells, white matter tracts, and the neurovascular unit. Although the initiating etiological factors may vary, the nervous system responds to persistent injury through relatively uniform pathological mechanisms, including neuronal degeneration, synaptic loss, neuroinflammation, demyelination, and architectural disintegration of neural networks. These structural changes form the anatomical basis for neurological dysfunction and the clinical manifestations observed in chronic neurodegenerative disorders.[32,33]

Neuronal Structural Changes
Neuronal injury represents the primary event in the pathogenesis of chronic neurodegenerative disorders. In the early stages, neurons exhibit adaptive and potentially reversible changes such as mitochondrial dysfunction, impaired axonal transport, synaptic dysfunction, and accumulation of misfolded proteins. These alterations compromise neuronal metabolism and intracellular signaling without immediate cell death.[33,34]
With disease progression, sustained cellular stress leads to dendritic spine loss, cytoskeletal disruption, and activation of apoptotic pathways. Repeated cycles of neuronal apoptosis and necrosis result in a progressive reduction of functional neuronal populations. Unlike many other cell types, neurons have limited regenerative capacity; therefore, neuronal loss leads to permanent disruption of neural circuits and irreversible functional decline.[34,35]

Clinically, these structural neuronal changes manifest as cognitive impairment, memory loss, motor dysfunction, sensory deficits, and behavioral disturbances, reflecting loss of region-specific neuronal populations.[35]

Synaptic and Network-Level Changes
Synaptic degeneration is an early and critical structural feature of chronic neurodegenerative disorders. Synapses serve as the fundamental sites of neuronal communication, and their integrity is essential for learning, memory, and neural plasticity. Structural changes include reduction in synaptic density, loss of dendritic spines, and altered neurotransmitter release mechanisms.[36]

Disruption of synaptic networks precedes overt neuronal loss in many neurodegenerative diseases and correlates strongly with early clinical symptoms. Progressive breakdown of neural connectivity leads to network disintegration, impaired information processing, and loss of functional reserve.[36,37]

Neuroglial and Inflammatory Changes
Neuroglial cells play a central role in maintaining neuronal homeostasis. In chronic neurodegeneration, astrocytes and microglia undergo reactive changes leading to sustained neuroinflammation. Activated microglia release pro-inflammatory cytokines, reactive oxygen species, and excitotoxic mediators, which further damage neurons and synapses.[37,38]

Oligodendrocyte injury and dysfunction result in demyelination and impaired axonal conduction. Glial scarring and gliosis contribute to structural rigidity of neural tissue, limiting plasticity and repair. These glial-mediated changes significantly accelerate neuronal degeneration and disease progression.[38]

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White Matter and Axonal Changes
White matter degeneration is a prominent structural feature in several chronic neurodegenerative disorders. Axonal injury, demyelination, and gliosis disrupt long-range neural connectivity essential for coordinated motor, sensory, and cognitive functions. Structural disintegration of white matter tracts contributes to slowed neural transmission, weakness, sensory impairment, and cognitive decline.[39]

Vascular and Neurovascular Unit Changes
Structural remodeling of the neurovascular unit is increasingly recognized as a contributor to chronic neurodegeneration. Microvascular alterations, blood–brain barrier disruption, and reduced cerebral perfusion impair oxygen and nutrient delivery to neural tissue. These vascular changes exacerbate oxidative stress, neuroinflammation, and neuronal vulnerability.[39]

Cumulative vascular dysfunction contributes to disease progression and facilitates the transition from compensated to advanced neurodegenerative states marked by widespread neural tissue loss.[39]

RADIOLOGICAL FEATURES AND STRUCTURAL CORRELATION IN CHRONIC NEURODEGENERATIVE DISORDERS

Radiological imaging provides essential non-invasive assessment of structural alterations in chronic neurodegenerative disorders and closely reflects underlying neuropathological changes. Imaging findings correlate with neuronal loss, synaptic degeneration, demyelination, and network disruption, thereby assisting in diagnosis, disease staging, and prognostication.[40]

Magnetic Resonance Imaging (MRI)
Magnetic resonance imaging is the primary imaging modality for evaluating chronic neurodegenerative disorders. Early disease stages may demonstrate subtle cortical thinning or regional signal changes, while advanced stages show pronounced cortical and subcortical atrophy, ventricular enlargement, and white matter degeneration. Region-specific patterns of atrophy often correspond to clinical phenotypes and underlying pathology.[40]

Computed Tomography and Advanced Imaging Techniques
Computed tomography may demonstrate global or regional brain atrophy in advanced disease, while advanced MRI techniques such as diffusion tensor imaging and functional MRI allow assessment of white matter integrity and network connectivity. Radiological findings correlate closely with histopathological features such as neuronal loss, demyelination, and gliosis, reinforcing the applied anatomical basis of neuroimaging in chronic neurodegeneration.[41]

CLINICAL AND FUNCTIONAL CORRELATION OF STRUCTURAL CHANGES IN CHRONIC NEURODEGENERATIVE DISORDERS

The clinical manifestations of chronic neurodegenerative disorders arise directly from progressive structural alterations of neurons, synapses, glial cells, and white matter tracts. Loss of functional neuronal populations leads to impaired neurotransmission, network failure, and reduced neural plasticity, while vascular and glial changes exacerbate disease severity.[32,33]

Region-specific neuronal loss produces characteristic clinical syndromes, including dementia, movement disorders, motor neuron weakness, and sensory deficits. Progressive structural degeneration ultimately results in loss of independence, severe disability, and systemic complications.[38,39]

Thus, chronic neurodegenerative disorders represent an integrated anatomical–functional continuum in which progressive structural remodeling of the nervous system culminates in global neurological failure. Understanding these correlations is essential for accurate clinical assessment, disease classification, and therapeutic decision-making.[32]

AYURVEDIC REVIEW OF CHRONIC NEURODEGENERATIVE DISORDERS

Ayurvedic physiology describes the structural and functional integrity of the human body through the concept of *Sapta Dhatus*, namely *Rasa*, *Rakta*, *Mamsa*, *Meda*, *Asthi*, *Majja*, and *Shukra*. These tissues are formed sequentially through the process of *Dhatu Poshana*, wherein each Dhatu is nourished and refined by its respective *Dhatvagni*, ultimately supporting the stability and vitality of the organism. This hierarchical organization ensures continuity of structure and function across tissues, and derangement at any level predisposes to degenerative pathology.[42]

Among the *Sapta Dhatus*, *Asthi Dhatu* and *Majja Dhatu* hold particular significance in the context of neurological health. *Asthi Dhatu*, formed from *Meda Dhatu* by *Asthidhatvagni*, provides rigidity, protection, and structural support, while *Majja Dhatu*, derived from *Asthi Dhatu* through *Majjadhatvagni*, fills the cavities of bones and supports neurological and cognitive functions. Classical texts describe *Majja* not merely as bone marrow but as a functional substrate intimately associated with the nervous system via *Majjavaha Srotas*.[43]

Ayurveda emphasizes a close structural and functional interdependence between *Asthi* and *Majja*, explained through the principle of *Ashraya–Ashrayi Bhava*, wherein *Vata Dosha* resides in *Asthi Dhatu*. Consequently, degeneration or depletion (*Kshaya*) of *Asthi Dhatu* leads to *Majja Kshaya*, resulting in heightened *Vata Prakopa* and progressive neurological dysfunction. This concept aligns with the modern understanding that skeletal integrity and marrow health influence neural stability and repair mechanisms.[44]

From a contemporary neurological standpoint, chronic neurodegenerative disorders encompass a wide spectrum of conditions such as Alzheimer's disease, Parkinson's disease, Huntington's disease, amyotrophic lateral sclerosis, multiple sclerosis, peripheral neuropathies, vascular dementias, metabolic neuropathies, and secondary neurological manifestations arising from skeletal degeneration and marrow

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compromise. These disorders are characterized by structural degeneration, inflammation, demyelination, and neuronal loss, with aging being a major contributing factor.[45]

In Ayurveda, most neurodegenerative conditions are classified under *Vata Vyadhi*, as *Vata Dosha* governs sensory perception, motor activity, cognition, and neural coordination. Depletion (*Kshaya*) or vitiation (*Dushti*) of *Asthi–Majja Dhatus* leads to aggravated *Vata*, manifesting as disorders analogous to modern neurodegenerative diseases. Classical descriptions such as *Kampavata*, *Smritibhramsha*, *Pakshaghata*, *Gridhrasi*, *Avabahuka*, *Ardita*, and *Vepathu* demonstrate striking clinical parallels with Parkinsonism, dementia, stroke, radiculopathies, neuropathies, and tremor disorders.[46]

Etiological factors contributing to *Asthi–Majja Kshaya* include *Jara* (aging), diminished *Agni*, inadequate *Brimhana Ahara*, excessive intake of *Ruksha*, *Laghu*, and *Sheeta Ahara*, chronic stress, suppression of natural urges, excessive physical exertion, trauma, and long-standing systemic diseases. These factors collectively weaken tissue regeneration, enhance *Vata* dominance, and initiate progressive structural degeneration.[47]

Classical features of *Asthi Kshaya* include dental looseness, hair and nail fragility, bone pain, and deformity, while *Majja Kshaya* manifests as weakness, dizziness, cognitive decline, tremors, impaired coordination, sensory disturbances, and memory loss. These descriptions correlate with modern entities such as osteoporosis, myelopathy, demyelinating disorders, neurodegeneration, and dementia. Pathologically, *Asthi–Majja Kshaya* initiates a cascade of tissue depletion, *Vata* aggravation, impaired nerve conduction, and progressive neurological failure.[48]

Assessment of *Asthi–Majja* involvement in Ayurveda is performed using *Darshana*, *Sparshana*, and *Prashna*, along with *Ashtavidha* and *Dashavidha Pariksha*, focusing on *Dhatu Bala*, *Srotas Dushti*, and *Vata Lakshana*. Modern diagnostic tools such as bone mineral density assessment, MRI, CT, neurophysiological studies, and biochemical markers complement Ayurvedic evaluation and enhance early detection of degeneration.[49]
Therapeutically, Ayurveda emphasizes *Nidana Parivarjana*, *Brimhana Chikitsa*, *Snehana–Swedana*, *Basti Chikitsa*, *Nasya*, and *Rasayana* therapy for *Asthi–Majja* disorders. *Ksheera Basti* and *Anuvasana Basti* act systemically to nourish *Majja Dhatu* and pacify *Vata*, while *Medhya Rasayana* drugs promote cognitive preservation and neuroprotection. These principles parallel modern neuroprotective and rehabilitative strategies.[50]

An integrative understanding of the *Asthi–Majja Axis* bridges Ayurvedic tissue-based pathology with modern neurobiological mechanisms such as oxidative stress, mitochondrial dysfunction, neuroinflammation, and impaired neuroplasticity. The recognition of bone-derived endocrine and immune influences on neural function further supports the Ayurvedic assertion that structural tissue health underpins neurological integrity.[51]

Thus, Ayurveda provides a foundational framework for understanding chronic neurodegenerative disorders as diseases of progressive tissue depletion and *Vata* dominance. Integrating this perspective with modern neurological science offers a comprehensive, preventive, and regenerative approach to neurodegenerative disease management.[52]

DISCUSSION

Chronic neurodegenerative disorders represent a heterogeneous yet conceptually unified group of diseases characterized by progressive and irreversible structural degeneration of the nervous system. The present review synthesizes applied neuroanatomy, neuron-centered structural pathology, radiological correlates, and Ayurvedic interpretations to provide a comprehensive, integrative understanding of neurodegeneration. Despite differences in explanatory frameworks, both modern neuroscience and Ayurveda converge on the principle that sustained tissue degeneration underlies functional neurological decline.[1,6]

From a modern anatomical perspective, neurons and their synaptic networks form the fundamental structural and functional units of the nervous system. Chronic neurodegenerative disorders are initiated at the molecular and subcellular level, with early changes involving mitochondrial dysfunction, impaired axonal transport, protein misfolding, and synaptic failure.[3,21,23] These early alterations precede overt neuronal loss and account for the subtle cognitive, motor, and sensory disturbances seen in prodromal stages. As degeneration progresses, neuronal apoptosis, dendritic spine loss, demyelination, and gliosis lead to irreversible disruption of neural circuits, culminating in region-specific atrophy and global neurological failure.[32,35]

Radiological imaging substantiates these structural changes at the macroscopic level. Magnetic resonance imaging demonstrates cortical thinning, subcortical atrophy, ventricular dilatation, and white matter degeneration, which closely correlate with histopathological neuronal loss and demyelination.[6,9,40] Advanced imaging modalities such as diffusion tensor imaging further reveal microstructural white matter damage, reflecting axonal degeneration and network disintegration.[41] These findings reinforce the concept that neurodegenerative disorders are fundamentally diseases of progressive structural remodeling rather than isolated neurochemical abnormalities.

Ayurvedic literature provides a parallel yet deeply tissue-oriented explanation for neurodegenerative pathology. The concept of *Sapta Dhatus* emphasizes sequential tissue nourishment and structural continuity, with *Asthi Dhatu* and *Majja Dhatu* playing a pivotal role in neurological health. Depletion (*Kshaya*) or vitiation (*Dushti*) of these Dhatus leads to aggravated *Vata Dosha*, which governs neural conduction, motor activity, cognition, and sensory perception.[42,44] The *Ashraya–Ashrayi Bhava* between *Vata* and *Asthi* explains why degeneration of skeletal tissue precipitates *Majja Kshaya* and neurological dysfunction, offering a structural rationale for age-related neurodegeneration.[44,48]

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The Ayurvedic classification of neurological disorders under *Vata Vyadhi* demonstrates remarkable clinical overlap with modern neurodegenerative conditions. Descriptions of *Kampavata*, *Smritibhramsha*, *Pakshaghata*, *Gridhrasi*, and *Ardita* closely resemble Parkinsonism, dementia, stroke, radiculopathies, and facial palsy respectively.[46] These correlations suggest that classical Ayurvedic clinicians recognized neurodegeneration as a progressive tissue-depleting process long before the advent of modern neuropathology.
Aging (*Jara*) emerges as a central etiological factor in both systems. Ayurveda recognizes age-related dominance of *Vata*, weakening of *Agni*, and natural decline of *Asthi–Majja Dhatus*, predisposing individuals to degenerative disorders.[48,51] Modern medicine parallels this with observations of reduced bone density, marrow cellularity, myelin synthesis, neurotrophic support, and increased oxidative stress with advancing age.[23,25,31] The convergence of these views strengthens the validity of integrative models targeting early degeneration rather than late-stage symptom control.

Diagnostic approaches in Ayurveda, including *Darshana*, *Sparshana*, *Prashna*, *Ashtavidha*, and *Dashavidha Pariksha*, emphasize early detection of *Dhatu Kshaya* through subtle clinical markers such as brittle nails, hair loss, gait disturbances, and cognitive changes.[49] When complemented with modern diagnostic tools such as MRI, BMD assessment, neurophysiological studies, and biochemical markers, an integrated diagnostic framework emerges that may enhance early identification of preclinical neurodegeneration.[16,49]

Therapeutically, Ayurveda adopts a tissue-restorative and preventive approach through *Brimhana*, *Rasayana*, *Basti*, and *Nasya* therapies aimed at nourishing *Asthi–Majja Dhatus* and pacifying *Vata*.[50] These strategies conceptually align with modern goals of neuroprotection, neuroplasticity enhancement, and functional rehabilitation.[20] The emerging recognition of bone-derived endocrine factors such as osteocalcin influencing cognition further bridges Ayurvedic insights with contemporary neuroscience, validating the *Asthi–Majja–Manas* axis described in classical texts.[51]

Thus, the discussion highlights that neurodegenerative disorders are best understood as integrated structural-functional diseases involving neuronal, glial, skeletal, and vascular systems. Ayurveda and modern neuroscience, when viewed together, provide complementary perspectives that enrich understanding of disease origin, progression, and therapeutic potential.[45,52]

CONCLUSION

Chronic neurodegenerative disorders are progressive conditions marked by irreversible structural degeneration of neurons, synapses, white matter tracts, and supporting tissues, ultimately resulting in functional neurological decline. Applied neuroanatomy reveals that degeneration begins at the cellular and network level long before overt clinical manifestations, emphasizing the importance of early structural assessment.

Radiological imaging serves as a crucial bridge between microscopic pathology and clinical expression, allowing visualization of regional atrophy, demyelination, and network disruption. From an Ayurvedic standpoint, neurodegeneration is fundamentally rooted in *Asthi–Majja Dhatu Kshaya* and *Vata Prakopa*, providing a tissue-centric explanation that aligns closely with modern observations of skeletal, marrow, and neural interdependence.

The integrative framework presented in this review underscores that neurodegenerative disorders are not isolated diseases of the brain alone but systemic disorders of structural integrity and metabolic balance. Ayurveda contributes preventive, regenerative, and holistic strategies aimed at preserving foundational tissues, while modern neuroscience offers precision diagnostics and targeted interventions.

A combined approach that integrates early structural diagnosis, modern neuroimaging, and Ayurvedic tissue-nourishing therapies may offer a more comprehensive strategy for slowing disease progression, improving quality of life, and addressing the growing global burden of neurodegenerative disorders. Future research should focus on longitudinal, integrative studies that validate these correlations and translate them into evidence-based clinical practice.

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ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 12| Issue: 2| February2026|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2026: 8.753 || ISI Value: 1.188

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