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A glimpse into the pathology of Parkinson's Disease - An Ayurvedic Perspective

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ABSTRACT

Parkinson's disease (PD) is a progressive neuro degenerative disease characterised by a large number of motor and non-motor features that can impact on function to a variable degree. *Charaka* mentioned *Kampa* as one among 80 types of *Vataja Nanatmaja Vyadhi*. *Kampa* may be a symptom of many diseases. *Kampavata* is first described as a disease in *Basavarajeeyam* with cardinal symptoms as *Hastapadatala Kampa, Dehabharamana, Dukkha, Nidrabhanga, Matiksheena*. Here an effort is made to understand the *Nidana Panchakas* of *Kampavata* under the light of *Kaphaavarana* to all five types of *Vata*, especially *Prana, Udana* and *Vyana*. There is *Udanaavruta Vyana* and *Pranaavruta Samana* type of *Anyonyaavarana*, *Majjaavruta Vata, Snayuprapta Vata* and *Asthimajjagata Vata* as pathological processes depending upon the clinical presentation of the patient. The differential diagnosis of PD is also considered here to differentiate it from group of disorders which falls under Parkinsonism.

Key words: *Parkinson's disease (PD), Kampavata, Avarana, Parkinsonism.*

INTRODUCTION

Parkinson's disease (PD) is a progressive neurodegenerative disease characterised by a large number of motor and non-motor features that can impact on function to a variable degree. Parkinson's disease was first described in "An essay on shaking palsy" in 1817 by a London physician James Parkinson. It is estimated that, there are 5 million people all over the world suffering from this disease.[1] In India the crude age adjusted prevalence rate of Parkinson's disease per 1,00,000 population is 14 in North India, 27 in South and 16 in the East.[2] Mostly men are more affected than women.[3] Early in the disease process, it is hard to know whether a person has idiopathic PD or a syndrome that mimics it. Parkinsonism, also known as atypical PD, or Parkinson's plus, represent about 10-15% of all diagnosed cases of Parkinsonism.

Charaka mentioned *Kampa* as one among 80 types of *Vataja Nanatmaja Vyadhi*.[4] *"Na Kampovayuna Vina."*[5] There is no *Kampa* without *Vata*. *Kampa* may be a symptom of many diseases like *Vatajajwara, Unmada, Vatikakushta, Vatikapandu, Urusthambha.* Various synonyms used are *Kampana, Vepana, Vepathu, Spandana* all indicating tremors of varying nature and severity.

In Rigveda, it is mentioned that the Lord Indra suffered from *Vepathu*. Such references are also available in the Atharvaveda. Unlike *Charaka*, *Acharya Sushruta* has not mentioned *Vepathu* as a separate disease. But he mentioned it as a symptom of *Snayuprapta Vata*[6] and also complication of *Ardita*. As per *Ashtanga Hridaya*, *Kampa* is a symptom of *Vata Prakopa* and *Sarvangavata*. *Kampa* is noted in *Raktakshaya, Pittakshaya* and *Kaphakshaya*, *Sarvangagata Vata* according to *Ashtanga Sangraha.*

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Vepathu had been first mentioned as an independent clinical entity by *Madhavakara* in *Madhava Nidana* in the 7th century.[7] He states that the tremors in the whole body (*Sarvanga Kampa*) and head (*Shirokampa*) caused by *Vata* are called *Vepathu*. *Kampavata* is first described as a disease in *Basavarajeeyam*[8] with cardinal symptoms as *Hastapadatala Kampa, Deha Bhramana, Dukkha, Nidrabhanga, Matiksheena.* In *Sharangdhara Samitha, Kampavata* is numbered among *Nanatmaja Vata Vyadhis*. *Acharya Bhela* noted the symptom *Kampa* in condition of *Asthimajjagata Vata*. Treatment of *Kampavata* is described in *Chakradatta* and *Vangasena*. Many formulations useful in *Kampavata* are described in *Bhaishajya Ratnavali*. In most of the neurodegenerative disorder *Vata* dominant symptoms are appreciable due to vitiation of *Vata* either due to *Dhatukshaya* or *Avarana*.[9] Here *Nidana Panchakas* of *Kampavata* or PD are discussed in detail under the light of *Avaranajanya, Dhatukshayajanya Vata Prakopa.*

Etiological factors

When physicians diagnose PD, they often describe it as idiopathic. Researchers believe that aging, genetics and environmental factors interact to cause PD.

Aging: The possible role of aging in the pathogenesis is suggested by its usual occurrence in late middle age, or older ages, but it remains unclear what precise role aging plays in pathogenesis.

Genetic factors: There are several genes that, when mutated, can increase the risk of PD. They are LRRK2, alpha-syncline gene, GBA gene, the parkin gene and the DJ-1gene.

Environmental factors: Certain environmental factors, such as significant exposure to pesticides or heavy metals, repeated head injuries, drugs etc.

No specific cause for *Kampavata*. But common causative factors of *Vata* and *Kapha* (which causes *Avarana* to *Vata*) can be considered here.

Pathology

There is a loss of neurons in certain areas of the brain, including a region called 'Substantia-nigra', Latin for 'black substance'. The neurons in this region (which appear black under the microscope) produce a neurotransmitter called dopamine. Dopamine helps to regulate movement. As the number of cells in the Substantia-nigra decreases, there is less dopamine available in the brain. Loss of neurons in other parts of the brain also occurs in PD, and accounts for some of the non-motor symptoms of the disease.

Another pathology which contributes to the development of PD is the presence of 'Lewy bodies' in the residual neuronsin the brain regions showing the most neuron loss in PD. These are the microscopic aggregates of the protein called 'alpha-synuclein'.[10]

The concept that free radical mediated injury for the neuronal degeneration has been the leading hypothesis for its pathogenesis. A genetically regulated cell death process may underlie the neuron-specific degeneration of later life has gathered greater attention in recent years. The possibility that protein aggregation may play a role in PD had long been suggested by the presence of Lewy bodies in disease brains.[11]

Decreased copper along with increased iron has been found in Substantia-nigra and caudate nucleus of Parkinson's disease patients. The decreased protein bound copper in brain may enhance iron accumulation and the associated oxidative stress. The increased copper in CSF could imply that the copper is leaking from proteins or cells or that it is not adequately transported in or out of cells.

Experts even have suspected that there may be a 'gut-brain axes where the intestinal environment influence the functioning of the central nervous system and intestinal imbalance especially IBD may precede and cause Parkinson's disease. Of interest, a specific pattern of p-alpha- sync aggregates can be identified in the enteric nervous system (ENS) in patients with PD. Braak and colleague's state that the disease begins in the enteric nervous system and gains entry to the CNS through the vegus nerve.

In this disease *Vata* aggravates by *Vatakara Ahara Vihara, Kapha Avarana* and *Dhatu Kshaya* as a result of *Avarana*. *Kaphakaraahara* and *Vihara* causes aggravation of *Kapha* by impairing the *Agni*. This

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aggravated *Kapha* causes *Avarana* to the varieties of *Vata* especially *Prana, Udana, Vyana* (*Doshavarana*), there is *Anyonyavarana* between *Udana* & *Vyana*[12] & *Majjavrita Vata*[13] (*Dhatwavarana*). The *Vata* aggravated due to *Vatakara Aharavihara* directly and by *Avarana* takes *Ashraya* in *Asthi Majja* & *Snayu* to present with the set of clinical features of *Vataprakopa*, specific *Avarana* and *Gata Vata* which explains the same pathological process taking place at different parts of the brain to present with PD or Parkinsonism. Clinical features of aggravated *Vata* at one site and decreased at another, which explains *Prakupita Vata Lakshanas*, is the hallmark of *Avarana Vata*[14].Part of *Samprapti* (pathological process) involved is explained in detail under each clinical feature.

Samprapti Ghataka

Dosha :

Vata - all five typesespecially *Prana, Udana* and *Vyana*.

Pitta - Sadhaka.

Kapha : *Tarpaka*

Dushya : *Rasa, Mamsa, Majja, Snayu.*

Agni and Ama : *Jataragni* and *Dhatwagnimandya Janya Ama.*

Srotas : *Rasa, Mamsa, Majjavaha.*

Srotodushtiprakara : *Sanga.*

Udbhava Sthana : *Pakvashaya.*

Adhishtana : *Shirastha Majja.*

Rogamarga : *Madhyama.*

Vyakta Sthana: *Sarvanga*

Prodromal features

Early prodromal symptoms include profound fatigue, unilateral aches and pains, tension, restlessness, tingling and burning, shoulder sourness, anosmia[15] drenching sweats, and the feeling of inner tremor.[16] Milder form of clinical features of *Kampavata* or *Prakupita Vata Lakshanas* are the *Purvarupas*.

Clinical features

'Braak' *et al.* developed a staging system which is divided into six different stages, with each stage being attributed to abnormal pathology in particular neurological structures.

Early stages are characterised by non-motor symptoms, such as a lessened sense of smell or constipation. Motor symptoms are often displayed around the mid-stage state, and cognitive symptoms arise as later Braak stages are reached.

Primary motor symptoms

Tremor, Rigidity, Akinesia (bradykinesia), and Postural instability are generally considered as cardinal features of PD that can be grouped under the acronym TRAP. These begin in an insidious asymmetric fashion. In addition, flexed posture and freezing(motor blocks) have been included among classical features of PD.

Secondary motor symptoms

Hypomimia, dysarthria, dysphagia, sialorrhoea, micrographia, shuffling gait, festination, freezing, dystonia, positive glabellar reflexes, mirror movements.

Non-motor symptoms

Autonomic dysfunction, cognitive / neurobehavioral abnormalities sleep disorders, sensory abnormalities such as anosmia, paraesthesia and pain.

Non-motor symptoms

Resting tremor

Basavarajeeyam describes '*Hastapadatalakampa*' tremors of hands and legs are the cardinal features of *Kampavata*. *Kampa* is *Vata Vriddhi Lakshana* and is described as *Vepathu*. Under *Prakupita Vata Lakshanas* it is commented as *Ativepana* or *Chalana*. It is one of the *Snayuprapta* Vata Lakshana. *Kampa* results when *Asthira Guna* of *Kapha* decreases and *Chala Guna* of *Vata* increases.

Rest tremor is the most common and easily recognized symptom of PD. Clinical-pathological studies have demonstrated that the patients with PD

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and prominent tremor have degeneration of subgroup of midbrain (A8) neurons, whereas this area is spared in PD patients without tremor. Tremors are unilateral, occur at a frequency between 4 and 6 Hz, and almost always are prominent in the distal part of an extremity. Hand tremors are described as supination-pronation (pill-rolling) tremors. Rest tremor in patients with PD can also involve the lips, chin, jaw, legs but unlike essential tremor, rarely involves the neck/head or voice.

Some patients also report an "internal" shaking that is not associated with a visible tremor. Parkinson's related postural tremor ("re-emergent tremor")[17] is differentiated from essential tremor in that the appearance of tremor after assuming an outstretched horizontal position.

Rigidity

Mobility is the function of *Vata*. Loss of upward, downward, flexion, extension etc. movements of different parts of the body is called as *Stambha* or *Sankocha* as described as *Prakupita Vata Lakshana* by *Vagbhata*. *Stambha* is considered as a clinical feature of *Kaphavritavyana Vata* by *Vagbhata*, *Snayuprapta Vata* by *Sushrutha* and *Udanavritavyana* by *Charaka*. This will explain the rigidity of PD. Rigidity is a prominent feature of many extrapyramidal diseases. Rigidity is characterized by increased resistance, usually accompanied by the "lead pipe rigidity", "cogwheel" phenomenon, present through out the range of passive movement of a limb (flexion, extension or rotation about a joint). It may occur proximally (e.g., neck, shoulders, hips) and distally (e.g., wrists, ankles). Reinforcing manoeuvres (e.g. voluntary movements of the contralateral limb), known as the Formant's manoeuvre,[18] usually increase rigidity. Rigidity may be associated with pain and painful shoulder is one of the most frequent initial manifestations of PD.

Bradykinesia

Chestapravartana (motor activities) is the function of *Vata* specifically *Vyana Vata* is responsible for '*Mahajava'* i.e, *Sheeghragati* or fast movement of the body parts. *Apakshepana* (downward), *Utkshepana* (upward), *Unmesha*, *Nimesha* (blinking of eyes) etc. movements are done by *Vyana Vata*.

Cheshtahani is mentioned as a feature of *Udanavritavyana*. *Cheshtastambha*, *Skalitagati*, *Adhikagatisanga* described as *Kaphavritavyana Vata Lakshana* explains bradykinesia and akinesia of Parkinson's disease.

Bradykinesia (in its most severe form akinesia) refers to slowness of movement and is the most characteristic clinical feature of PD. Bradykinesia is a hallmark of basal ganglia disorders and it encompasses difficulties with planning, initiating and executing movement and with performing sequential and simultaneous tasks.[19] This may include difficulties with tasks requiring fine motor control (e.g., buttoning, using utensils). Other manifestations of bradykinesia include loss of spontaneous movements and gesturing, dysphagia and drooling of saliva because of impaired swallowing, loss of facial expression (hypomimia), decreased blinking.

The bradykinesia and postural instability both contribute to walking or gait difficulties in PD. Reduced arm swing, slow small steps, shuffling gait known as festination, atendency to propel forward with rapid short steps known as propulsion will be seen while walking. Speech disorders are characterised by monotonic, hypo phonic, soft breathy speech, dysarthria and frequent word finding difficulties referred as "tip-of-the-tongue phenomenon."[20]

Dysphagia and drooling of saliva develops due to impairment of '*Annapravesha'* i.e, swallowing function of *Prana Vata*.[21] *Vagbhata* explained swallowing difficulty or *Kantarodha* as a feature of *Prakupitaudana Vata*.[22] *Vakpravritti* is the function of *Udana* [23] assisted by *Vyana Vata*.[24] Therefore *Vakgraha* or *Swaragraha* results from *Kaphavrita Udana*[25] and *Vyana*[26] and *Pranavritasamana*.

Assessment of bradykinesia usually includes having patients perform rapid, repetitive, alternating movements of the hand (finger taps, hand grips, hand pronation-supination) and heel taps observing not only slowness but also decrementing amplitude. The

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phenomenon (kinesia paradoxical) suggests that patients with PD have intact motor programmes but have difficulties accessing them without an external trigger.

Postural deformities

Vinamana i.e, bent body (stooped posture-spine bent) and different parts of the body like extremities (*Anganam Vinamanam*) is the feature of *Majjavrita Vata* as explained in *Ashtanga Hridaya*. This explains postural deformities of Parkinson's disease.

Rigidity of the neck and trunk (axial rigidity) may occur, resulting in abnormal axial postures (e.g., anterocollis, scoliosis). Flexed elbows and knees are often associated rigidity. Striatal hand is characterized by ulnar deviation of the hands, flexion of the metacarpophalangeal joints and extension of the proximal and flexion of the distal interphalangeal joints, striatal foot is characterized by extension or flexion of the toes.[27] Other skeletal abnormalities include extreme neck flexion ("dropped head" or "bent spine"), truncal flexion (camptocormia) and scoliosis.[28] Camptocormia is characterized by extreme flexion of thoracolumbar spine. The condition is exacerbated by walking and is relieved by sitting, lying. Another truncal deformity is the Pisa syndrome, which is characterized by a tilting of the trunk, particularly when sitting or standing.[29]

Postural instability

Loss of *Sthiratwa* property of *Kapha* and increased *Chala* property of *Vata* leads to postural instability or *Deha Bhramana*, a clinical feature of *Kampavata* as described by *Basavarajeeyam*. Falling with instability or orthostatic hypotension results from the association of *Pitta* and *Rajodosha* of *Manas* with the *Vata* to be understood as *Bhrama* as explained by *Madhava Kara*.

Postural instability results due to loss of postural reflexes.The pull test, in which the patient is quickly pulled backward or forward by the shoulders, is used to assess the degree of retropulsion or propulsion respectively. Taking more than two steps backwards or the absence of any postural response indicates an abnormal postural response. Several other factors also influence the postural instability, like orthostatic hypotension, age related sensory changes and the inability to integrate visual, vestibular and proprioceptive sensory input (kinaesthesia).[30]

Freezing

Freezing, referred to as motor blocks, is a form of akinesia (loss of movement) and is one of the disabling symptoms of PD. It typically manifests as a sudden and transient inability to move. This include hesitation with 5 subtypes i.e, start hesitation, turn hesitation, hesitation in tight quarters, destination hesitation and open space hesitation.

Saada, which is one of the *Prakupita Vata Lakshana* is described as '*Anganamkriya Asaamarthyam*'[31] will explain the off-feature or freezing seen in PD.

Non-motor features[32]

Autonomic dysfunction : features include orthostatic hypotension, sweating dysfunction, sphincter dysfunction and erectile dysfunction. '*Mutra* and *Shakrita Pravartana'* loss or inability to pass urine or bowels is the presentation of *Kaphavritaapana Vata*.[33] This explains the constipation, sphincter dysfunction and erectile dysfunction resulting out of autonomic dysfunction in Parkinsonism.

Cognitive and neurobehavioral abnormalities

Matiksheena is a feature of *Kampavata*. Features of dementia occurs due to functional impairment of *Pranavata*, which is responsible for normal functioning of *Buddhi, Shirohridaya, Manas* and *Indriya. Sadhakapitta, Tarpakakapha* also contributes for its normal functioning. Abnormality of *Smriti* will be contributed by a abnormal *Udana Vata*, as *Udana Vata* is responsible for normal *Smriti*. *Chittopaplava* (disturbed mind) a feature of *Prakupitavyana Vata* explains its role in the normal functioning of *Manas*.

Dementia, depression, anxiety, apathy, hallucinations, unable to remember words, particular periods of time and people, inability to focus on or complete a task in the manner that they desired. There may be OCD, and impulsive behaviour such as craving repetitive

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handling, examining referred as "hedonistic homeostatic dysregulation".[34]

Sleep disturbances: patient may experience difficulties falling asleep, staying asleep (nonrestorative). *Nidrabhramsha* is the feature of *Prakupita Vata* assisted by *Pitta*.

Sensory abnormalities: Loss of smell, the olfactory dysfunction, and muscle ache, oral and genital pain may be seen. *Vata* is considered as '*Sarvaindriyaudyojaka'*. Specifically *Prana Vata Vikriti* leads to '*Indriyaupaghata'* or improper perception from senses.

Melanoma[35] : PD patients have an increased risk of developing melanoma, a type of skin cancer.

Emotional burden of living with PD makes them feel frustrating and incredibly scary, as they need to rely on care givers. Motor symptoms, lack of energy, anxiety will lead to social isolation. Periods of "off-time" described as unpredictable exacerbation of symptoms during which medications were less effective: stress and cold weatherare considered to be aggravating factors. This various off-and-on states makes this disease so hard to live with.

Diagnostic criteria have been developed by the UK Parkinson 's Disease Society Brain Bank and the National Institute of Neurological Disorders and Stroke (NINDS). A number of rating scales are used for the evaluation of motor impairment and disability in patients with PD but the commonly used is Hoehn and Yahr scale. The unified Parkinson's disease Rating scale (UPDRS) is the most well established scale for assessing disability and impairment.

Investigations

- Magnetic resonance imaging (MRI) - to rule out other conditions
- DaT scan- an imaging test that measures dopamine function in the brain.
- Trans cranial ultrasound
- Genetic testing
- Dopaminergic response test

Differential diagnosis[36]

Absence of rest tremor, early occurrence of gait difficulty, postural instability, dementia, hallucination, and presence of dysautonomia, optholmoparesis, ataxia and other atypical features, coupled with poor or no response to levodopa, suggest diagnosis other than PD.

Essential tremor

A positive family history, symmetrical postural kinetic tremor unlike resting tremor of PD, suggests essential tremor.

Progressive Supranuclear Palsy (PSP)

Mental and physical slowness, unsteady gait and early falling within the first year of illness due to postural reflex impairment are the cardinal features. Neck posture in extension instead of the typical flexion seen with the most Parkinsonian syndromes.

They also complain of vague alterations in vision, difficulty looking down, trouble seeing food on plate or trouble seeing steps. There will be incomplete pursuit eye movements, slowed saccades and the presence of square wave jerks. Speech and swallowing complaints are seen early.

There is relative loss of resting tremor with mild to moderate Parkinsonism evident. Another characteristic feature is poor response to dopaminergic agents. The progression to disability is much faster in PSP.

T2 weighted images in PSP will reveal Mickey Mouse sign and humming bird sign.

Multiple System Atrophy

This idiopathic neuro-degenerative process of adult-onset, characterised by varying proportions of cerebellar dysfunction, autonomic failure and Parkinsonism. There are three subgroups namely Olivo Ponto Cerebellaratrophy (OPCA), Striatal Nigral Degeneration (SND), Shy Dragger Syndrome.

The clinical features include rare resting tremor, progressive akinesia, rigidity with severe head flexion and postural instability.

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Autonomic dysfunction is characterised by postural hypotension manifested as dizziness, visual obscuration's, inspiratory stridor, cranial cervical discomfort (upon raising from a sitting or supine position they experience dull achiness in the posterior occipital region and across both shoulders.) and syncope. Genitor urinary dysfunction includes erectile dysfunction, urinary frequency and urgency and incontinence. Constipation of a severe nature can develop.

Cerebellar dysfunction as a part of MSA includes gait ataxia, dysarthria kinetic tremors in the upper extremity, hypotonia and rebound phenomenon. The ocular motor signs include nystagmus, jerking pursuit, ocular dysmetria and slowing of saccades.

Evidence of pyramidal dysfunction in MSA includes brisk and exaggerated deep tendon reflexes, Babinski sign, pseudo bulbar palsy and spasticity of the limbs.

The evolution of symmetric Parkinsonism in 90% of patients associated with autonomic failure in 80%, pyramid signs in 60% and cerebellar signs in 55% is a definite indication of MSA. There is usually rapid progression to being wheelchair bound. This is poorly responsive to levodopa therapy.

T2 weighted images of MSA will reveal 'hot cross bun sign'.

Cortico Basal Ganglionic Degeneration (CBD)

Symptoms include unilateral slowness and awkwardness of one hand. The involved hand and arm described as "applause sign" and "alien hand" will assume involuntary postures and gestures such as removing eye glasses or unbuttoning a shirt. These patients always exhibit severe rigidity, frequent dystonic postures and occasional myoclonus. There may be cortical sensory findings and apraxia as a prominent feature. There is often severe dysfunction of speech with hypophonia and dysarthria.

T1 weighted axial section images reveal 'pan cake' representation of the cortex.

Dementia with Lewybodies (DLB)

This is also called diffuse Lewy body disease, Parkinson's disease with dementia, or Alzheimer's disease (AD) with Parkinsonism. Onset of Parkinsonism and dementia should occur within 12 months of each other to establish the diagnosis of DLB.

Vascular Parkinsonism

It is also called as atherosclerotic Parkinsonism or lower half Parkinsonism. The advent of imaging has made it possible to identify multiple infarcts and lacunes scattered throughout the basal ganglia or extensive periventricular white matter disease.

These patients along with mild Parkinsonian findings have a great difficulty walking and often marked shuffling and a freezing magnetic gait. Postural reflexes are severely impaired and there is a tendency to fall. Patients show little response to dopaminergic agents even in high doses.

Apart from these disorders conditions like Normal Pressure Hydrocephalus (NPH), Drug induced parkinsonism, any kind of extrapyramidal lesion with Parkinsonian presentation should be considered for differential diagnosis.

CONCLUSION

PD is a progressive neurodegenerative disorder. This can be understood as *Kampavata* under the light of *Kaphaavarana* to all five types of *Vata*, especially *Prana, Udana* and *Vyana*. There is *Udanaavruta Vyana* and *Pranavruita Samana* type of *Anyonyavarana, Majjaavruta Vata, Snayuprapta Vata* and *Asthimajjagata Vata* as pathological processes depending upon the clinical presentation of the patient. Here *Vata* aggravates by 3 factors namely, *Vatakaraaharavihara*, *Avarana* and the resultant *Dhathukshaya*. A thorough understanding of the broad spectrum of motor and non-motorclinical manifestations of PD is essential for proper diagnosis of the disease and differentiating it from Parkinsonism. Future research may uncover disease specific biomarkers helpful for diagnosis, differential diagnosis and assessment of treatment.

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