NISHAADA GRAHONMADA: BEHAVIORAL AND PSYCHOLOGICAL SYMPTOMS OF DEMENTIA? / FRONTOTEMPORAL DEMENTIA? / HEBEPHRENIA?

NISHAADA GRAHONMADA: DEMANSIN DAVRANIŞSAL VE PSİKOLOJİK BELİRTİLERİ? / FRONTOTEMPORAL DEMANS? / HEBEFRENİ?

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Abstract

Nishaada grahonmada (NG) is one among 18 types of bhootonmada / grahonmada (psychiatric problems caused by the possession of evil spirits or super natural powers or extra terrestrial forces). Description of NG is found in ‘Ashtanga samgraha’ and ‘Ashtanga hridaya’ (Ayurvedic classical texts written by Vriddha Vagbhata and Vagbhata). In ‘Asthtanga samgraha’, ‘Kaakhorda grahonmada’ (KG) term is used synonymously for NG. The description of both NG and KG is similar. NG is characterized by Nagnam dhaavantam (nude or naked appearance / sexual disinhibition), gruheetva kaashta loshtaadi bhramantam (aggressiveness / restlessness / wandering / impulsivity / agitation), cheera vaasasam & trina vibhooshanam (inappropriate or bizarre clothing / disorganized behaviour), uthrasta drishtim (abnormal eye movements), parusha abhidaayinam (hostile speech / verbal aggression), smashaana shunya rathya nishevanam (staying alone / social isolation / staying at inappropriate places), tilaanna madya maamsa sakta drishtim (fond of sweets prepared with sesame, alcohol and meat) etc clinical features. Till date no studies have been conducted on NG and the concept as well as clinical application of NG is not clearly understood. The present study is focused to understand NG in a better way by correlating it with the modern psychiatric condition. Clinical features of NG shows similarity with various psychiatric / neuropsychiatric conditions like, BPSD (Behavioral and psychological symptoms of dementia), FTD (Frontotemporal dementia) and Hebephrenia.

Keywords: nishaada grahonmada, behavioral and psychological symptoms of dementia, frontotemporal dementia, hebephrenia, ayurveda, kaakhorda grahonmada

Özet

Nishaada grahonmada (NG), 18 çeşit bhootonmada / grahonmada (kötü ruhlara veya süper doğal güçler veya dünya dış güclere sahih olmanın neden olduğu psikiyatrik sorunlar) arasında yer almaktadır. NG’nin tanımı ‘Ashtanga samgraha’ ve ‘Ashtanga hridaya’ (Vriddha Vagbhata ve Vagbhata tarafından yazılan Ayurvedik klasik metinler) ’de bulunur. “Asthtanga samgraha” da, “Kaakhorda grahonmada” (KG) terimi NG için esaslı olarak kullanılmıştır. NG’nin ve KG’nin tanımları benzerdir. NG Nagnam dhaavantam (çiplak veya çiplak görünüm / cinsel disinhibisyon), gruheetva kaashta loshtaadi bhramantam (saldırganlık / hızursuzluk / dalgalık / dürtüsellik / ajitasyon), cheera vaasasam ve trina vibhooshanam (uygunsuz veya garip kıyafet / düzensiz davranış), uthrasta drishtim (anormal göz hareketleri), parusha abhidaayinam (hostil konuşma / sözel saldırganlık), smashaana shunya rathya nishevanam (yalnız / sosyal izolasyon / uygunsuz yerlede kalma), tilaanna madya maamsa sakta drishtim (susam, alkol ve et ile hazırlanan tatlılar) vb. klinik özellikleri ile başdaştırılır. Bugüne kadar NG ile ilgili herhangi bir çalışma yapılmamıştır ve NG’nin kavram ile klinik uygulaması açık bir şekilde anlaşılmamıştır. Bu çalışma, NG’yi modern psikiyatrik durumla ilişkilendirek daha iyi anlamba odaklanmıştır. NG’nin klinik özellikleri, BPSD (demansın davranışsal ve psikolojik belirtileri), FTD (Frontotemporal demans) ve HEBEPHRENİ gibi çeşitli psikiyatrik / nöropsikiyatrik durumlarla benzerlik göstermektedir.

Anahtar Kelimeler: nishaada grahonmada, demansın davranışsal ve psikolojik belirtileri, frontotemporal demans, hebephrenia, ayurveda, kaakhorda grahonmada

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1. Introduction

‘Bhuta vidya’ (Ayurvedic psychiatry) is one of the eight branches or specialties of Ayurveda. The word ‘bhuta’ has different meanings in different contexts such as ‘super natural power’ or ‘demon’ or ‘extra terrestrial force’ or ‘paranormal force’. ‘Bhutonmada’ / ‘Grahonmada’ is a psychiatric condition characterized by, abnormal behavior and psychomotor activity seen in a person with sudden onset and idiopathic nature (Mamidi & Gupta, 2015). There are 18 types of bhutonmada’s (deva, asura, rushi, guru, vruddha, siddha, pitru, gandharva, yaksha, rakshasa, sarpa, brahma rakshasa, pishacha, kushmanda, nishada, preta, mukirana and vetaala) and ‘Nishaada grahonmada’ (NG) is one among them (Prasad & Kshama, 2015).

There is no description found regarding NG in Charaka samhita (Agnivesha, 2008), Sushruta samhita (Sushruta, 2009) and Madhava nidana (Madhavakara, 2012). Only lakshana’s (signs and symptoms) of NG are explained in Ashtanga samgraha (Vriddha Vagbhata, 2012) and Ashtanga hridaya (Vagbhata, 2005). The description of NG is similar in both texts (Ashtanga samgraha and Ashtanga hridaya). In ‘Ashtanga samgraha’, ‘Kaakhorda grahonmada’ (KG) term is used synonymously for NG (Vriddha Vagbhata, 2012). The description of both NG and KG is similar (Vriddha Vagbhata, 2012; Vagbhata, 2005).

NG is characterized by Nagnam dhaavantam (nude or naked appearance / sexual disinhibition), gheeetva kaasha loshtaa bhramantam (aggressiveness / restlessness / wandering / impulsivity / agitation), cheera kaashta loshtaadi bhramantam (aggressiveness / naked appearance / sexual disinhibition), gruheetva nishada (hostile speech / verbal aggression), smashaana shunya rathya nishevanam (staying alone / social isolation / staying at inappropriate places), tilaanna madya maamsa sakta drishtim (fond of sweets prepared with sesame, alcohol and meat) etc clinical features (Vriddha Vagbhata, 2012; Vagbhata, 2005). Till date no studies have been conducted on NG and the concept as well as clinical application of NG is not clearly understood. The present study is focused to understand NG in a better way by correlating it with the modern psychiatric condition. Clinical features of NG shows similarity with various psychiatric / neuropsychiatric conditions like, FTD (Frontotemporal dementia), BPSD (Behavioral and Psychological Symptoms of Dementia), Hebephrenia.

2. Etiology, Pathogenesis, Course and Prognosis of NG

Causative factors are not visible or traceable in grahonmada. Pragyaparaadha (intellectual blasphemy) and karma (idiopathic) plays an important role in the pathogenesis of bhutonmada. The onset of bhutonmada is sudden and instantaneous without significantly affecting the body physiology. The signs and symptoms of bhutonmada are also infinite. The course and prognosis of bhutonmada is unpredictable (Prasad & Kshama, 2015). There is no special description regarding etiology, pathogenesis, course and prognosis of NG is available in Ayurvedic classical texts. The common etiology, course and prognosis mentioned for grahonmada is applicable for NG also (Agnivesha, 2008; Sushruta, 2009; Madhavakara, 2012; Vriddha Vagbhata, 2012; Vagbhata, 2005).

The mechanism by which BPSD occurs in some patients with AD (Alzheimer’s dementia) is not completely understood. Studies on BPSD in subtypes of AD appear to be least understood and least explored in India (Musthaq et al., 2016). BPSD are associated with lowered functional abilities and poorer prognosis (Brodaty et al., 2003). BPSD is associated with a more rapid rate of cognitive decline and greater impairment in activities of daily living and there are variations in severity of BPSD at different cognitive levels. Underlying neurobiology of BPSD is still unclear (Kar, 2009). Available data indicate that BPSD occurs due to both anatomical and biochemical changes within the brain. Pathological changes in the cholinergic system cause BPSD via the denervation of the frontal and temporal cortices. Alterations in adrenergic and serotonergic systems also contribute to the development of these symptoms. Higher levels of norepinephrine in the substantia nigra and lower levels of serotonin in the presubiculum are seen in patients with BPSD. BPSD are also heritable, with certain symptoms occurring more frequently in family members with dementia (Tampi et al., 2011).

The pathogenesis of NPS (Neuropsychiatric symptoms) or BPSD has not been clearly delineated but it is probably the result of a complex interplay of psychological, social, and biological factors. Recent studies have emphasized the role of neurochemical, neuropathological, and genetic factors underlying the clinical manifestations of BPSD. Altered glucose metabolism has also been associated with anxiety, apathy, agitation, and disinhibition in AD involving multiple regions: apathy was related to the bilateral anterior cingulated gyrus, medial orbitofrontal cortex, and medial thalamus; agitation / disinhibition were related to frontal and temporal lobes. Behavioural variant – FTD (bv-FTD) begins with atrophy of the orbitofrontal, anterior cingulated, and anterior insular cortex and quickly involves the basal ganglia (Dillon et al., 2013). Thus, it seems that the patho-physiology of BPSD or FTD is not fully understood or unclear or not traceable causative factor (externally visible) just like the etiopathology of grahonmada.

3. Behavioral and Psychological Symptoms of Dementia (BPSD)

BPSD is a term used to delineate diverse range of psychological responses, psychiatric symptoms, and behaviours occurring in any type of dementia. AD patients have increased verbal and physical aggression. Anxiety is often associated with other NPS such as depression, psychosis, aberrant motor behaviour, disinhibition, euphoria, irritability and agitation (Musthaq et al., 2016). The management of dementia is complicated by BPSD, such as psychosis, depression, agitation, aggression and disinhibition (unrestrained behaviour resulting from a lessening or loss of inhibitions or a disregard of cultural constraints). BPSD is an umbrella term for a heterogeneous group of non-cognitive symptoms that are almost ubiquitous in dementia. Moderately severe
behavioural and psychological symptoms characteristic of people include major depression, verbal aggression, low-level (non-dangerous) physical aggression, psychosis, sexual disinhibition and wandering (Brodatty et al., 2003). Individuals with dementia commonly manifest BPSD such as aggression, agitation, psychotic symptoms, sleep disturbance and wandering (Haw et al., 2009). BPSD such as agitation, aggression, calling out repeatedly, sleep disturbance, wandering and apathy affect up to 90% of people with dementia (Feast et al., 2016). Symptom clusters that emerge during the study of the psychopathology of persons with dementia include the mood disorders cluster (depression, anxiety, and apathy/indifference), psychotic cluster (delusions and hallucinations), aberrant motor behaviors cluster (pacing, wandering, and other purposeless behaviors), and inappropriate behavior cluster (agitation, disinhibition, and euphoria). Pacing, wandering, rummaging, picking, and other stereotyped, purposeless behaviors, which are more common in moderate-to-severe stages of dementia. Finally, disinhibition characterized by tactlessness and impulsivity occurs in 36% of patients with dementia (Tampi et al., 2011). Different neuropsychiatric pathologies produce abnormalities in eye movements, which can be registered and measured (Bioni et al., 2017).

NPS are now regarded as an intrinsic aspect of dementia and they are common hallmarks of all types of dementia, independent of etiology. Patients with mild cognitive impairment (MCI) present with a higher rate of NPS. Patients with mild behavioral impairment (MBI) show a notably increased risk of progression to degenerative dementia. NPS also known as BPSD according to the ‘International Psychogeriatric Association’, represent a heterogeneous group of non-cognitive symptoms and behaviours occurring in subjects with dementia. Common NPS can include agitation, anxiety, irritability, illusion and delusions, apathy, depression, disinhibition, aberrant motor & obsessive–compulsive behaviours, and sleep disorders. These manifestations can be present at any stage of dementia and non-dementia–cognitive impairment. NPS is the first indicator of impending dementia and it is most common in patients with FTD and AD. MBI was defined as a behavioural disturbance characterized by agitation, anxiety symptoms, apathy, aspontaneity, delusion symptoms, depressive symptoms, disinhibition, emotional lability, euphoria, impulsivity, indifference, irritability, lack of empathy, loss of insight, loss of personal hygiene, loss of social contact, oral / dietary changes, perseverant behaviour and sleeping disorders. MBI, specifically in the absence of cognitive symptoms, probably represents an FTD prodrome. Depression, anxiety, irritability, aggressiveness, apathy, euphoria, sleep and appetite disturbances, motor restlessness, hallucinations, delusions, and paranoia are typical features of fulminate AD. The presence of NPS in MCI patients is an indicator of increased risk of progression to dementia (Dillon et al., 2013).

Major manifestations of BPSD are in the areas of motor behavior, social interactions, speech, personality changes and somatic symptoms. Inappropriate behaviors have been divided into four main subtypes: physically aggressive behavior (hitting, kicking or biting), physically nonaggressive behavior (paced or inappropriately handling objects), verbally non aggressive agitation (constant repetition of sentences or requests) and verbal aggression (cursing or screaming). Agitation manifests with restlessness, pacing, complaining, repetitive sentences, negativism, requests for attention, cursing and verbal aggression etc. Hyperphagia has been reported though uncommonly. Mood disturbances like depression, anxiety, fear, irritability, anger, emotional lability, explosive emotional outbursts, weeping and laughing are seen. Apathy, a syndrome (one of the most common NPS reported) characterized by decreased initiation and motivation, decreased social engagement, emotional indifference, diminished reactivity and lack of persistence. Changes in personality like, increasing passivity, coarsening of affect, decreased spontaneity, inactivity, feelings of insecurity, less cheerfulness and responsiveness etc are found. Loss of socialization, companionship, self-centred behaviour, irritability, reduced initiative and drive, grossly insensitive behaviour, lack of restraint, disinhibition, sexual misadventure, indolence, foolish jokes and pranks are also seen. Psychotic features in patients with dementia are usually paranoid in nature. Behavioural symptoms occur more frequently as the severity of dementia increase. Aggression, wandering, incontinence, apathy, rage, hyper metamorphosis, binge eating, sexual disinhibition or hyperorality is reported in a study (Kar, 2009).

4. Similarity Between BPSD and NG

Various features of NG like Nagnam dhaavantam, gruheetva kaasha loshtaa bhramantam, cheera vaasasam & trina vibhooshanam, uthrasta dhrishtham, parusha abhidaayinam, smashaana shunya rathya nishevanam, and tilaanna madya maamsa sakta dhrishtim etc resembles with features like sexual disinhibition / hypersexuality, agitation / aggression / irritability / impulsivity / motor restlessness / aberrant motor behaviour / rage / wandering, purposeless behaviour / grossly insensitive behaviour / lack of personal hygiene / loss of insight, abnormal eye movements, verbal aggression, loss of social contact / indifference / apathy / depression / social withdrawal, and hyperphagia / binge eating / hyperorality etc of BPSD or NPS or prodrome of FTD or AD or Dementia. But in NG there is no symptom suggesting of memory impairment which is the most common feature of dementia. The answer for this is the clinical picture of NG may indicates either prodrome of FTD or AD or BPSD in ‘Vascular cognitive impairment – No dementia (VCI-ND)’ patients.

5. Vascular Cognitive Impairment – No Dementia (VCI-ND)

Apart from frank cases of vascular dementia (VaD), there exists a subgroup of patients with cerebrovascular disease who demonstrate cognitive deficits not fulfilling the traditional definitions of dementia. This subgroup has been designated “Vascular cognitive impairment – No dementia (VCI-ND).” VCI-ND may be considered to be the prodromal stage of VaD. Diagnosis of VaD mandates
the documentation of memory impairment. However, several patients have cognitive dysfunction attributable to vascular brain insults where memory may not be impaired as commonly as other domains like executive function. In a study, comparing the prevalence of NPS across the declining memory continuum in AD (i.e., normal controls, subjective cognitive impairment, mild cognitive impairment and mild AD), the prevalence of BPSPD was found to be higher at the higher end of the spectrum, i.e., mild AD. The total NPI (Neuropsychiatric Inventory) scores also were found to increase along the continuum (Gupta et al., 2013). It seems that there may be patients of dementia without memory impairment, which exactly resembles with the clinical picture of NG.

6. Frontotemporal Dementia (FTD)

FTD is another neurodegenerative condition characterized by disinhibition, euphoria, irritability, agitation, and aggressive behaviour. Symptoms like psychomotor agitation and aggressive behaviour have been associated with fronto-temporal pathology not only in patients with FTD but also AD (Almeida, 2004). Appetite changes can be quantitative (anorexia or hyperphagia) or qualitative (preference for particular foods associated or not to changes in taste). The preference for sweets is particularly frequent in fronto-temporal dementia. Fronto-temporal lobar degeneration (FTLD) is the prototype of a neurodegenerative disorder where changes in behavior, rather than in cognitive function, are the presenting feature and dominate the clinical picture throughout the disease course. Stereotypic behaviour, appetite changes and loss of social awareness are characteristic of FTLD with complex ritualized behaviours occurring more frequently in patients with FTD and semantic dementia than in AD. Loss of basic emotions, food cramming, pacing a fixed route, and an absence of insightfulness differentiated FTD from other dementias (Cerejeira et al., 2012).

Behavioural disinhibition is a classic hallmark of bv-FTD. Patients may inappropriately touch or aggressively approach strangers. Patients may also disregard subtler social norms, encroach on the personal space of others, exhibit childish behaviour and a general lack of etiquette. Patients can be less socially engaged and fail to exhibit personal warmth, even with friends and family. Patients frequently exhibit altered food preferences, commonly craving sweets or carbohydrates, or expressing rigid preferences for particular foods. In some cases, patients may engage in binge-eating. bv - FTD patients have consistently been found to show higher scores of apathy, euphoria, disinhibition, aberrant motor behavior, and eating abnormalities compared to AD patients (Manoochehri & Huey, 2012). Patients of AD have an increased preference for sweet foods. Although disengagement was predictive of lower total food intake, increased “psychomotor regulation” disturbances, including irritability, agitation, and disinhibition, were primarily associated with higher carbohydrate and lower protein selection. The use of sweet, protein-dense foods, such as ice creams and puddings, especially in those displaying “psychomotor regulation” disturbances, may be of value, particularly at dinner when the drive toward high carbohydrate foods is most evident (Greenwood et al., 2005).

People with FTD often exhibit disinhibition, wandering, social inappropriateness, and apathy. Agitation (a broad category that includes excessive psychomotor activity such as pacing, trailing, restlessness, dressing and undressing, and emotional distress) is common, persistent, and may increase with disease severity (Kales et al., 2015). Clinically, bv-FTD is characterized by a cluster of behavioural symptoms in association with executive dysfunction. Changes in personality, social behaviour that include apathy and disinhibition, lack of initiation, setting aside personal responsibilities, and experience impairment of professional activities. Patients lose empathy for others and are not interested in what may occur in their environment. Disinhibition presents as inappropriate social behavior, loss of decorum, and inappropriate vocabulary. Lack of insight, denial of illness, dramatic changes in personal hygiene and dressing, repetitive motor acts, modifications in eating habits, hyperorality, and hypersexuality; Klüver-Bucy syndrome can even develop. Patients can also develop depression, emotional lability, but with an absolute indifference to their surroundings. Patients with bv-FTD exhibit impairment in their self criticism, self-perception, and social skills. Clinically, they are socially uninhibited and lack emotional recognition and empathy. They fail in the recognition of social behavior, and are not able to appropriately evaluate the severity of moral and social transgressions (Dillon et al., 2013).

7. Similarity Between FTD and NG

Various features of NG like Nagnam dhaavantam, grheeertva kaashtha loshtaai bhramantam, cheera vaasam & trina vibhooshanam, parusha abhidaayinam, smashaana shunya rathy naishvanam, and tilaanna madya maamsa sakta dirshtm etc resembles with FTD features like disinhibition, agitation / irritability, socially inappropriate behaviour, irritability / emotional lability / lack of empathy, apathy, and excessive preference to sweet and protein dense foods.

8. Hebephrenia

There are five types of schizophrenia: paranoid, disorganized, catatonic, undifferentiated, and residual. Disorganized type is characterized by disorganized speech and behavior, as well as flat or inappropriate affect. The onset of schizophrenia can be abrupt or insidious. Most patients undergo a prodromal phase marked by a slow and gradual development of symptoms, such as social withdrawal, loss of interest in school or work, deterioration in hygiene and grooming, unusual behavior, or outbursts of anger (Schultz et al., 2007). In 1881, Hecker described an entity of early onset, typically during adolescence, characterized by the emergence of mood symptoms before the onset of psychotic and disorganized symptoms, progressing quickly to severe functional impairment and cognitive deterioration. He termed this entity “Hebephrenia.” Hebephrenia had to satisfy the basic diagnostic criteria for schizophrenia and show prominence of at least two out of the following symptoms:
disorganized speech, disorganized behavior, and flat or inappropriate affect (Ortiz et al., 2013). Hebephrenic and catatonic schizophrenia are collectively referred to as “nuclear” or “kernel” schizophrenia, both involving severe personality and behavioral deterioration, an early onset, a poor prognosis, and a larger percentage of schizophrenic relatives than other types (Templer & Veleber, 1982). Early course of illness in hebephrenia is insidious, continuous, and distinct but nonreactive (Mc Glashan & Fenton, 1991).

9. Similarity Between Hebephrenia and NG

Various features of NG like Nagnam dhaavantam, gruhveeta kaasha loshtaadi bhramantam, cheera vaasaasaam & trina vibhooshanam, parusha abhidaayinan and smashahana shunya rathya nishhevanam etc resembles with features like disorganized behaviour, disorganized speech, deterioration in hygiene and grooming, unusual behaviour and flat or inappropriate affect of Hebephrenia.

10. Conclusion

‘Nishaada grahonmada’ is one among 18 types of grahonmada. The signs and symptoms of NG have shown similarity with various psychiatric / neuropsychiatric conditions like, BPSD (Behavioral and psychological symptoms of dementia), FTD (Frontotemporal dementia) and Hebephrenia.

References


