**Pitru Grahonmada – Neuropsychiatric Presentation of Vitamin B12 Deficiency?**

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**Abstract**

*Ayurveda* is an ancient Indian system of medicine has been in practice since thousands of years. *Bhuta vidya* is one of the eight branches / specialities of Ayurveda which deals with various psychiatric and neuropsychiatric conditions. Pitru grahonmada (PG) is a type of bhutonmada or grahonmada (various psychiatric or neuropsychiatric conditions of idiopathic origin). PG is one among 18 types (deva, asura, rushi, guru, vruddha, siddha, pitru, gandharva, yaksha, rakshasa, sarpa, brahma rakshasa, pishacha, kushmanda, nishada, preta, maukirana and vetala) of bhutonmada or grahonmada. Bhutonmada is characterized by various patterns of abnormal behavior and psychomotor activity like abnormalities of speech, valor, potency, activities, intelligence, knowledge and strength. Bhutonmada is caused by affliction of evil spirits or super natural powers or some idiopathic factors. The present study aims at better understanding of PG and its clinical applicability. PG is characterized by *Aprasanna drishtim & Apashyantam* (vision loss or vision abnormalities), *Chala netra pakshmaanaam* (abnormal eye movements), *Shankitekshanam* (suspicious looks), *Apasavya vastram* (doing rituals in disorganized manner which denotes confusion, memory loss, disorientation), *Deena vadanam* (depression), *Ananabhaslisha / Arochaka / Avipaaka / Alpaagmi / Manda paavaka* (various gastro-intestinal tract abnormalities like loss of appetite, indigestion, malabsorption etc), *Prathitha vaacham / Skhalat vaacham* (speech abnormalities), *Nidraalu* (excessive sleepiness / fatigue), *Samsushta taalukam* (dry palate / oral manifestations of vitamin B12 deficiency), *Shaantaatma* (catatonia / depression / reduced psycho-motor activity), *Tila, guda, maamsa and paayasa priyam* (cravings for sugar and meat) etc features. These features of PG have shown striking similarity with vitamin B12 deficiency induced neuropsychiatric conditions. PG has shown similarity with various neuropsychiatric manifestations induced by vitamin B12 deficiency.

**Keywords:** Psychiatry, Depression, Neuropsychiatry, Vitamin B12, Fatigue, Catatonia

**INTRODUCTION**

The term ‘Unmada’ denotes, ‘a state of disturbed mental functions’ and it is characterized by the deviation of manas (mind), buddhi (decision), smriti (memory), sangya gyanam (orientation & responsiveness), bhakti (desire), sheela (habit), cheshta (activity) and achaara (conduct) (1). Unmada is classified in to two groups based on etiopathology and treatment, *doshaja unmada* (internal origin) and *bhutonmaada or grahonmaada* (external or idiopathic origin). Bhutonmaada is characterized by various patterns of abnormal behavior and psychomotor activity seen in a person such as abnormalities of speech, valor, potency, activities, intelligence, knowledge, strength etc; all of a sudden without any visible cause (idiopathic, sudden personality changes) (2). Acharya Vagbhata (one of the biggest contributor of Ayurveda) has described 18 types of grahonmada’s. They are deva, asura, rushi, guru, vruddha, siddha, pitru, gandharva, yaksha, rakshasa, sarpa, brahma rakshasa, pishacha, kushmanda, nishada, preta, maukirana and vetala. Pitru grahonmada (PG) is one among these 18 types of grahonmada (3).

According to Acharya Charaka (one of the biggest contributor of Ayurveda), PG is characterized by the features like *aprasanna drishtim* (abnormalities of vision), *apashyantam* (loss of vision), *nidraalu* (excessive sleepiness or fatigue), *prathitha vaacham* (obstructed speech or reduced speech), *ananabhaslisha* (anorexia), *arocakam* (loss of appetite) and *avipaakam* (indigestion) (4). According to Acharya Sushruta, *pretebhyo visruja-
ti pindaan (doing rituals), shaantaatma (reduced psycho-motor activity), apasavva vatra (performing rituals in a disorganized manner which denotes confusion or disorientation or memory loss) and maamsa, tila, guda & paayasa abhikaama (craving for meat, sesame, sugar and sweets) are the features of PG (5). In Ashtanga samgraha (written by vridha Vagbhat) along with the above features, chala netra pakshmaanaam (abnormal eye movements), shankitekshanam (suspicious looks / paranoid), deena vadanam (depression) and samsushka taalum (dry palate or oral manifestations) are also mentioned (6). The description of PG in ‘Ashtanga hridaya’ (7) and in ‘Madhava nidaana’ (8) is almost similar to the above texts.

Previous works on ‘Rakshasa’, ‘Daitya’ and ‘Yaksha Grahavesha’ have shown striking similarity with various psychiatric conditions like antisocial personality disorder with psychotic mania, antisocial/narcissistic/borderline personality disorder, and bipolar disorder with obsessive-compulsive disorder respectively (9-11). Till date no studies have been conducted on PG and it is an unexplored concept in the field of Ayurvedic psychiatry. The present study is focused at better understanding of PG by correlating it to the modern psychiatric conditions. The clinical picture of PG shows similarity with neuropsychiatric conditions induced by vitamin B_{12} deficiency.

VITAMIN B_{12} and ITS DEFICIENCY

B_{12} deficiency often goes undetected, with manifestations that range from asymptomatic to a wide spectrum of hematologic and / or neuropsychiatric features. The most common etiology of vitamin B_{12} deficiency is food-cobalamin malabsorption resulting from gastric dysfunction (12). Vitamin B_{12} deficiency has been associated with neurologic, cognitive, psychotic, mood symptoms as well as treatment-resistance. It is estimated that up to 40% of older adults have vitamin B_{12} (cobalamin) deficiencies. Cobalamin is synthesized by anaerobic bacteria and is found in foods of animal origin (e.g., fish, meat, dairy products and eggs) and in fortified cereals. Defects at any step of the absorption process can cause cobalamin deficiencies of varying degrees; cobalamin stores in the body are located in the liver. These stores help delay (often for up to 5 years), the onset of clinical symptoms due to insufficient cobalamin absorption (13).

Neuropsychiatric symptoms due to vitamin B_{12} deficiency have been described since the early 1900s. Commonly described neuropsychiatric manifestations associated with vitamin B_{12} deficiency include motor, sensory & autonomic symptoms, cognitive impairment, mood and psychotic symptoms. Other neuropsychiatric symptoms among individuals with vitamin B_{12} deficiency are paresthesias, ataxia, proprioception & vibration loss, memory loss, delirium, dementia, depression, mania, hallucinations, delusions, personality changes and abnormal behaviour. Neurologic symptoms have been the hallmark of vitamin B_{12} deficiency, which include abnormal reflexes, bowel / bladder incontinence, optic atrophy, orthostatic hypotension, and autonomic disturbances. The association of psychotic symptoms and cobalamin deficiency has been described for more than a century. Reported symptoms include suspiciousness, persecutory / religious delusions, auditory & visual hallucinations, tangential / incoherent speech, and disorganized thought-process. An association of vitamin B_{12} deficiency and depressive symptoms in elderly patients has also been documented (13).

ETIOLOGY, PATHOGENESIS, CLINICAL COURSE and PROGNOSIS of PG and VITAMIN B_{12} DEFICIENCY

There is no specific etiology, pathogenesis and prognosis explained for PG in Ayurvedic texts. The samaanya nidaana, sampraapti and saadhyasaadhyata (common etiology, pathogenesis & prognosis) explained for bhutonmada is also applicable for PG. Grahavesha (affliction by supernatural power), prgnaaparaadha and / or karma (deeds of present life or past life) are explained as causative factors for bhutonmada. In bhutonmada the symptoms occurs suddenly without any reason (insidious onset) or triggered by chidra kaaal (various predisposing factors) and the course of the disease is also unpredictable. The prognosis of bhutonmada is also unpredictable (3).

The clinical picture of vitamin B_{12} deficiency is highly polymorphic and of varying severity, ranging from asymptomatic to rare and severe disorders (14). The prognosis of neuropsychiatric conditions induced by vitamin B_{12} deficiency depends on symptom severity, duration and clinical diagnosis. Treating deficiencies in the early stages yields better results, as structural and irreversible changes in the brain may also occur if left untreated (13). The clinical picture, course and prognosis of vitamin B_{12} deficiency induced neuropsychiatric conditions are highly polymorphic and variable as found in grahonmada.
SIMILARITY BETWEEN THE SIGNS and SYMPTOMS of PG and VITAMIN B\textsubscript{12} DEFICIENCY

There is striking similarity found between the signs and symptoms of PG and various neuropsychiatric conditions caused by vitamin B\textsubscript{12} deficiency. This similarity has been explored in the following sections.

\textit{Aprasanna drishtim & Apashyantam} (vision abnormalities or vision loss)

Optic neuropathy denotes a broad spectrum of ophthalmological or systemic conditions that can affect the optic nerve. The patho-physiological mechanisms of optic neuropathies vary widely including inflammatory, traumatic, ischemic, autoimmune, genetic or toxic mechanisms. In case of bilateral, symmetric, slowly progressive optic neuropathies, the three main causes to be investigated are hereditary, metabolic and toxic. If a vitamin deficiency is suspected, a dosage of B\textsubscript{1}, B\textsubscript{6}, B\textsubscript{12} and folate vitamins are useful (15). Optic atrophy is one of the neurological manifestations of vitamin B\textsubscript{12} deficiency (16,17). The typical clinical picture of vitamin B\textsubscript{12} deficiency induced optic neuropathy includes bilateral, progressive and painless visual loss, central or cecocentral scotoma, colour vision defects and inappropriate visual evoked responses (14). Optic neuropathy is a rare, but important, manifestation of vitamin B\textsubscript{12} deficiency that should be suspected in patients with risk factors for malnutrition. Vitamin B\textsubscript{12} optic neuropathy is a reversible, treatable cause of vision loss and may be a harbinger for other manifestations of the disease (18). \textit{Aprasanna drishtim} and \textit{Apashyantam} of PG denotes vision loss or vision abnormalities caused by underlying optic atrophy or optic neuropathy induced by vitamin B\textsubscript{12} deficiency.

\textit{Chala netra pakshamaanaam} (abnormal eye movements)

Eye movement disorders are rarely reported in vitamin B\textsubscript{12} deficiency. There is a case report which has described two cases, one with bilateral inter nuclear ophthalmoplegia and the other with downbeat nystagmus (19). In contrast to frequent optic nerve involvement, eye movement disorders in vitamin B\textsubscript{12} deficiency, has only been reported 11 times in the literature. Serum vitamin B\textsubscript{12} should be measured in any patient with unexplained eye movement disorder (20). Blepharospasm is a form of focal dystonia characterized by involuntary contractions of the ‘orbicularis oculi’ muscles resulting in bilateral closure of the eyes. According to a study, a patient with severe blepharospasm found to have low vitamin B\textsubscript{12} levels and elevated homocysteine levels. Blepharospasm can be a rare manifestation of vitamin B\textsubscript{12} deficiency, which is reversible with therapy. Vitamin B\textsubscript{12} levels and homocysteine levels should be tested in patients with blepharospasm in whom there is no obvious cause for blepharospasm (21). Isolated paralysis of upward gaze may be a feature of vitamin B\textsubscript{12} deficiency (22). ‘Wandering eyes’ has been reported in vitamin B\textsubscript{12} deficiency (23). Thus conditions like ophthalmoplegia, nystagmus, other abnormal eye movements and blepharospasm induced by vitamin B\textsubscript{12} deficiency are similar to ‘\textit{chala netra pakshamaanaam}’ of PG.

\textit{Shankitekshanam} (suspicious looks / paranoid)

Vitamin B\textsubscript{12} deficiency has been highly linked to several psychiatric disorders like impaired memory, irritability, depression, dementia, delirium, schizophrenia and psychosis. Published studies on vitamin B\textsubscript{12} deficiency have documented prevalence of 29%, 44% and 70.8% among patients with primary dementia and schizophrenia, depression respectively (24). Neurocognitive impairment resulting from vitamin B\textsubscript{12} deficiency is usually accompanied by other neurologic abnormalities and may present as paranoia, irritability, dementia, hallucinations, psychosis, depression or disorientation (25). The common psychiatric manifestations of vitamin B\textsubscript{12} deficiency are depression, agitation or violence, delirium with or without delusions & hallucinations and acute paranoid states (26). Vitamin B\textsubscript{12} deficiency is associated with mood and psychotic disorders. Delusions, hallucinations, personality changes and abnormal behaviour are the neuropsychiatric symptoms of vitamin B\textsubscript{12} deficiency. Suspiciousness, persecutory / religious delusions, auditory & visual hallucinations and disorganized thought process are seen in vitamin B\textsubscript{12} deficiency induced psychotic conditions (13). Low serum vitamin B\textsubscript{12} is prevalent among patients admitted with psychiatric illnesses especially those with schizophrenia. Schizophrenia, a long duration of psychiatric illness, and acute hospitalization were independently associated with low serum vitamin B\textsubscript{12} levels among psychiatric patients (27). Thus ‘\textit{shankitekshanam}’ described in PG denotes ‘suspiciousness’ or ‘persecutory delusions’ or ‘schizophrenia’ induced by vitamin B\textsubscript{12} deficiency.

\textit{Apasavya vastram} (confusion or disorientation or cognitive impairment)

‘Apasavya vastram’ means wearing towel on right shoulder instead of left while performing rituals. It
signifies that the person suffering with PG performs rituals in disorganized way or improper manner. This may due to confusion, disorientation, forgetfulness (memory impairment) or cognitive impairment. Vitamin B₁₂ deficiency has been linked to memory impairment, dementia, delirium, schizophrenia, psychosis and depression (24). A case report has revealed that, a patient with spinal cord injury associated with vitamin B₁₂ deficiency had clinical features of delirium such as alteration in consciousness, disorientation, memory impairment, disorganized thinking, visual hallucinations and disturbances in concentration, attention & thinking (25). Psychiatric disturbances caused by vitamin B₁₂ deficiency also include delirium, dementia and hallucinations (26). The association of vitamin B₁₂ deficiency and cognitive dysfunction has been well established. Vitamin B₁₂ deficiency has been associated with attention deficits, acute mental-status changes, and acute cognitive changes with EEG abnormalities (13). Psychiatric symptoms attributable to vitamin B₁₂ deficiency fall into several clinically separate categories like slow cerebration, confusion, memory changes, delirium, hallucinations and / or delusions, depression, acute psychotic states, manic and schizophreniform states (28). Irrational behaviour, confusion, forgetfulness or memory impairment and other psychotic features were observed in vitamin B₁₂ deficiency patients (26). Based on these observations it seems that ‘Apasavya vastram’ mentioned in PG denotes an underlying acute confusion or disorientation or memory, cognition, attention, concentration impairment caused by vitamin B₁₂ deficiency.

**Deena vadanam (depression)**

Deficiencies in vitamin B₁₂ can lead to various neurological and psychiatric disorders such as depression, dementia and brain atrophy (29). In elderly people, depression, dementia and mental impairment are often associated with vitamin B₁₂ and folate deficiency. Vitamin B₁₂ and folic acid are crucial for the transmethylation of neuroactive substances such as myelin and neurotransmitters. There are several theories concerning potential associations between depression and levels of vitamin B₁₂ and folate. Vitamin B₁₂ and folate are connected with the synthesis of monoamines such as dopamine and serotonin and are involved in the patho-physiology of neuropsychiatric disorders such as depression and psychosis. Assessment of B₁₂ levels should be included as a standard evaluation with treatment-resistant depressive disorders, dementia, psychosis, or among individuals with history of poor nourishment (30). As there is growing evidence that B₁₂ deficiency is associated with depression and organic psychosis lends credence to consideration of obtaining B₁₂ levels in patients who presents with these problems (31). Adequate vitamin B₁₂ levels may also play a role in depression treatment response (13). Depression often accompanies a medical condition. Among the various systemic diseases, vitamin B₁₂ deficiency and folic acid deficiency are particularly likely to cause depression (32). ‘Deena vadanam’ mentioned in PG denotes depression induced by an underlying vitamin B₁₂ deficiency.

**Anannaabhiilaasha, Arochaka, Avipaaka, Alpaagni, Manda paavaka** (various gastro-intestinal tract abnormalities like anorexia, indigestion, malabsorption etc)

According to a study, chronic atrophic gastritis leads to vitamin B₁₂ deficiency and increasing weakness, loss of memory, mental depression were common symptoms. Flatulent dyspepsia, epigastric pain were also reported in the same study (33). Malabsorption of vitamin B₁₂ in patients with tropical sprue leads to vitamin B₁₂ deficiency and clinically it is manifested as neuropathy. Malabsorption of vitamin B₁₂ was present in all patients who had deficiency of this vitamin (34). The most common etiology of vitamin B₁₂ deficiency is food-cobalamin malabsorption resulting from gastric dysfunction. Pernicious anaemia is characterized by fundic atrophic gastritis (type A) leading to atrophy of the fundus and achlorhydria. Abnormal gastric endoscopic findings appear to be correlated with B₁₂ levels. *Helicobacter pylori* infection is associated with cobalamin deficiency, implicating *Helicobacter pylori* as an etiological factor for B₁₂ deficiency (12).

Stomach plays an important role in the absorption of vitamin B₁₂. Gastritis and gastric atrophy leads to an increase in intrinsic factor secretion and malabsorption, eventually resulting in vitamin B₁₂ deficiency. Atrophic gastritis is one of the important causes of vitamin B₁₂ deficiency. It has been reported that *Helicobacter pylori* infection resulting in chronic gastritis, which plays a role in the development of gastric atrophy and intestinal metaplasia. Deficiency of vitamin B₁₂ seen in conditions like inadequate dietary intake, disorders related to secretion of gastric pepsin, disorders of pancreatic secretion and intrinsic factor secretion from gastric parietal cells, and ileum disease in which absorption is disrupted (35). *Helicobacter pylori* seem to be a causative agent in the development of adult vitamin B₁₂ deficiency. Eradication of *Helicobacter pylori* infection alone may correct vitamin B₁₂ levels and improves anaemia (36).
Anannaabhilaasha, Arochaka, Avipaaka, Alpaagni and Manda paavaka of PG denotes various factors like malabsorption, atrophic gastritis, Helicobacter pylori infection and other gastro-intestinal tract abnormalities commonly seen in vitamin B\textsubscript{12} deficiency.

Pratihata vaacham & Skhalat vaacham (speech abnormalities)

A case report has cited, ‘abnormal speech’ in a 9 year old boy with vitamin B\textsubscript{12} deficiency (37). Hoarseness of voice and vocal cord palsy has been documented in cases of vitamin B\textsubscript{12} deficiency (29). Tangential speech or incoherent speech is also seen in vitamin B\textsubscript{12} deficiency induced psychotic conditions (13). Pratihata vaacham & Skhalat vaacham of PG denotes vocal cord palsy or incoherent speech or mutism etc conditions induced by vitamin B\textsubscript{12} deficiency.

Nidraalu (excessive sleepiness or fatigue)

Sleepiness is an impairment of normal arousal mechanism and is characterized by a tendency to fall asleep. Persons who are sleepy are temporarily aroused by activity whereas fatigue is intensified by activity. Patients with sleepiness feels better after a nap, but patients with fatigue report a lack of energy, mental exhaustion, poor muscle endurance, delayed recovery after physical exertion and non restorative sleep (38). Malaise and lethargy are the depressive symptoms induced by vitamin B\textsubscript{12} deficiency (31). Damage to peripheral nerves due to vitamin B\textsubscript{12} deficiency, results in sleepiness, altered taste and smell, and optic atrophy (39). Levels of vitamin B\textsubscript{12} in the cerebrospinal fluid were significantly correlated with measures of fatigability and neurasthenia in the patients with chronic fatigue syndrome (CFS) and fibromyalgia together. A substantial amount of vitamin B\textsubscript{12} appears to be necessary to relieve the symptoms of CFS. Vitamin B\textsubscript{12} administration may relieve CFS symptoms by reversing the erythrocyte abnormalities leading to improved tissue oxygenation (40). Vitamin B\textsubscript{12} deficiency was found to be associated with fatigue and depressive symptoms. Depression and fatigue are known to be strongly correlated. Fatigue is a common symptom of depression. Vitamin B\textsubscript{12} induced anaemia, could be a mediating factor, since this may also induce fatigue. There is an association between vitamin B\textsubscript{12} deficiency and increased levels of fatigue and depression in lacunar stroke patients (41). ‘Nidraalu’ of PG denotes fatigue or excessive sleepiness induced by vitamin B\textsubscript{12} deficiency.

Samsushka taalukam (dry palate or oral manifestations):

The most common causes of megaloblastic anemias are cobalamin (vitamin B\textsubscript{12}) and folate (vitamin B\textsubscript{9}) deficiency. Megaloblastic anemia progresses slowly, and symptoms include weakness, fatigue, shortness of breath and neurologic abnormalities. A wide range of oral signs and symptoms may appear in anaemic patients such as glossitis, angular cheilitis, recurrent oral ulcer, oral candidiasis, diffuse erythematous mucositis and pale oral mucosa etc., as a result of basic changes in the metabolism of oral epithelial cells. Soreness of the tongue & generalized ulceration, reduced taste sensitivity, generalized sore mouth and / or burning mouth has been reported in the literature. Candidiasis and angular cheilitis are common oral complaints of patients with megaloblastic anemia (42). The oral manifestations of glossitis, stomatitis and mucosal ulceration in vitamin B\textsubscript{12} deficiency are well established. These oral changes may occur in the absence of symptomatic anaemia (43). ‘Samsushka taalukam’ of PG denotes various oral manifestations as explained above due to vitamin B\textsubscript{12} deficiency induced megaloblastic anaemia.

Shaantaatma (reduced psycho-motor activity / catatonia / depression)

Catatonia has been associated with extreme vitamin B\textsubscript{12} deficiency (26). Catatonia is a condition which includes symptoms like catalepsy, stupor, rigidity, posturing, and waxy flexibility. Negativism, mutism, and catatonic excitement are other common symptoms of Catatonia. Vitamin B\textsubscript{12} deficiency has been linked to many psychiatric disorders. According to a case report ‘a patient has developed catatonia secondary to a low vitamin B\textsubscript{12} level, which improved after supplementation’. Vitamin B\textsubscript{12} deficiency induced catatonic patients have shown stupor, mutism, rigidity, posturing, and waxy flexibility (44). Low or reduced psycho-motor activity is also seen in depression, dementia and also in catatonia. ‘Shaantaatma’ of PG denotes depression or catatonia or reduced psycho-motor activity induced by vitamin B\textsubscript{12} deficiency.

Tila, guda, maamsa, paayasa priyam (cravings for sesame, meat, sugar and sweets)

Literature consistently demonstrates that individuals experiencing emotional distress, especially depression, report a craving and preference for sweet, carbohydrate and / or fat rich foods. Depressed individuals have shown increased preference to sweets, carbohydrate or fat rich foods. People with depression consume sweets,
carbohydrate and fat rich foods for mood enhancement (45). Depression is strongly associated with obesity, lower physical activity and higher caloric intake among obese individuals. Depression was associated with significantly higher daily caloric intake among those with BMI over 30 (46). Patients with Alzheimer’s disease have shown a greater preference than normal controls for relatively high-fat, sweet foods and for high-sugar, low-fat foods. Vascular dementia patients have also shown a similar pattern. Craving for sweet foods may be a significant part of the clinical syndrome of dementia (47). Depression and variation in dopamine related genes have both independently been associated with food consumption. Depressive symptoms could synergistically interact with genetic variation to influence food intake. Relationship between depression and food intake may vary as a function of genetic polymorphism (48).

Consumption of diets low in carbohydrate tends to precipitate depression. Neurotransmitter like serotonin and tryptophan which promotes the feeling of well being is triggered by carbohydrate rich foods. The neurotransmitter dopamine is made from the amino acid tyrosine and the neurotransmitter serotonin is made from the tryptophan. Lack of any of these two amino acids leads low mood and aggression. Deficiency of iron is known to cause fatigue and depression. Iron deficiency anemia is associated with apathy, depression, and rapid fatigue during exercise (49). *Tila, guda, maamsa, paayasa priyam* (fond of sesame, meat, sugar and sweet foods) of PG indicates cravings towards food rich in iron, calcium, aminoacids, proteins, sugars etc. Such type of cravings may denote deficiency of essential vitamins or minerals inside the body. These cravings denote anaemia, nutritional deficiencies (due to malabsorption), depression etc conditions induced by vitamin B_12_ and other deficiencies.

**CONCLUSION**

*Pitrugrahonmada* is one among 18 types of *grahonmada*. The signs and symptoms of PG have shown similarity with conditions which are induced by vitamin B_12_ deficiency like optic atrophy, optic neuropathy, ophthalmoplegia, nystagmus, abnormal eye movements, paranoia, disorientation, memory impairment, depression, atrophic gastritis, malabsorption, helicobacter pylori infection, speech abnormalities, glossitis or various oral manifestations, catatonia, fatigue and cravings towards sugars. PG shows similarity with various neuropsychiatric conditions caused by vitamin B_12_ deficiency. Even though the present article is a hypothetic equation of two different entities, not a proven diagnostic match and it would pave the way for further discussions regarding ancient Indian neuropsychiatric diagnoses and their actual definitions.

**REFERENCES**


