Case Report

Panchakarma in autoimmune pancreatitis: A single-case study

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Abstract

Autoimmune pancreatitis (AIP) is the pancreatic manifestation of a systemic fibro-inflammatory disorder. AIP is a unique form of pancreatitis in which autoimmune mechanisms are suspected to be involved in the pathogenesis. AIP is a rare disorder, its exact cause is unknown, but it is thought to be caused by the body's immune system attacking the pancreas and it responds to steroid therapy only. In Ayurveda, although there is no synonym for AIP, but has a resemblance in clinical features of *Grahani Dosha* (derangement of duodenum and intestine). The cause of *Grahani Dosha* is *Mandagni* (hypofunctioning of *Agni*) and *Panchakarma* therapy increases *Agni*. As per Charaka Samhita, treatment for *Grahani Dosha* amongst the *Panchakarma* therapy is *Virechana* (therapeutic purgation) and *Basti* (medicated enema). The present case report is of a 30-year-old female, diagnosed as case of AIP with multisystem involvement with increased level of immunoglobulin G (IgG), glycosylated heamoglobin (HbA1c), cholesterol, triglycerides, low-density lipoprotein (LDL) and body mass index (BMI). The patient was on anticholinergic agents, antacids, levothyroxine, multivitamin along with iron and antihistamine drugs since 1 year, but with not much relief. Patient was treated with classical *Virechana* and *Madhutailika Basti*. It was observed after the completion of therapy, that there was decrease in IgG, HbA1c, S. cholesterol, S. triglyceride, low density lipoprotein (LDL) and body mass index (BMI). This shows that *Virechana* and *Basti* play a significant role in patient with AIP associated with other disorders.

Keywords: Autoimmune pancreatitis, *Grahani Dosha*, *Madhutailika Basti, Panchakarma, Virechana*

Introduction

Autoimmune pancreatitis (AIP) is a rare form of chronic pancreatitis that has only recently been recognized as a separate type of pancreatitis in the last two decades. The histopathological features of this distinct form of pancreatitis was first described as early as 1961 when Sarles *et al*.[1] described a type of sclerosing pancreatitis associated with hypergammaglobulinemia. Subsequently, most of the early literature about AIP came from Japan where the concept of AIP was first proposed in 1995.[2] Thereafter, many authors had reported a form of chronic pancreatitis associated with Sjögren's-like syndrome. The definition of AIP was widely accepted and AIP was differentiated from other types of chronic pancreatitis. An increasing awareness and further research of AIP have indicated that it is a heterogeneous disorder with variations in pathophysiology, genetic predisposition, and extra-pancreatic manifestations compared to chronic pancreatitis.[3,4] In 2001, scientist reported increased serum levels of immunoglobulin G (IgG4) in patients with AIP.[5] Subsequently, in 2004, a critical milestone was reached when the researcher found intensely positive IgG4 cells in extrapancreatic organ systems in AIP patients.[6] Thus, the concept of IgG4-related systemic disease emerged. Type 1 AIP is now considered to be a pancreatic manifestation of IgG4-related disease whereas Type 2 AIP appears to be a pancreas specific disorder. The estimated prevalence in Japan, where AIP was first described, is 0.82/100,000 persons.[7] Japanese series have estimated the prevalence of AIP in patients with chronic pancreatitis to be between 5% and 6%. Several series in the United States have reported that 2%–3% of pancreatic resections had evidence of AIP at pathologic

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analysis.[8] Type 1 AIP is the most common form worldwide, accounting for almost all cases in Japan and Korea and more than 80% of cases in Europe and the United States.[9] Diabetes mellitus (DM), usually Type 2, is often (41% or 76% of cases) associated with it.[10,11] In many cases, the diagnoses of DM and AIP are made simultaneously; some patients show exacerbation of pre existing DM with the onset of AIP.

In Ayurveda, *Agni* (factors for digestion and metabolism) is given prime importance in pathogenesis of the disease. *Grahani* (small intestine) is an anatomical structure situated between *Amashaya* (stomach) and *Pakwashaya* (large intestine). Its physiological importance is due to its interdependence on *Agni*.[12] *Grahani Dosha* refers to *Grahanyashrita Agnidosha* (mal functioning of *Agni* at intestine level). In *Grahanyashrita Dosha* indigestion of food occurs and symptoms like *Vishtamba* (improper defecation), *Praseka* (nausea), *Arti* (pain), *Vidaha* (burning sensation) and *Aruchi* (dyspepsia) and *Gaurava* (heaviness) develops.[13]

In light of the Ayurvedic principles, the present case was treated for the management of AIP with multisystem disorder not responding to conventional therapy by utilizing the two arms of *Panchakarma*, i.e., *Virechana* (therapeutic purgation) and *Basti* (therapeutic enema). In diseases of the gastrointestinal tract, *Virechana* is reported to be more effective, as it eliminates aggravated *Pitta* and *Kapha Dosha* from the body. *Basti* is being used to pacify *Vata*, it also impart mild cleansing effect on the gastrointestinal tract.

Patient Information

A 30-year-old female, a home maker of Indian origin residing in America approached *Panchakarma* OPD of Institute for Post Graduate Teaching and Research in Ayurveda, Gujarat Ayurved University, Jamnagar, Gujarat, India, on December 30, 2015 with the episodes of recurrent abdominal pain. The pain increased after taking fatty meal that would persist for at least 5–6 h, usually at night, and usually preceding with episodes of vomiting and 5–7 loose stools. The pain was so severe that patient was unable to take a sip of water and would just sit in the bending position for several hours. There was difficulty in carrying out the daily chores due to weakness and fatigue. Also, she had anorexia and fear of taking fatty food. On physical examination, the patient was obese, anxious, pale with dry skin, had coated tongue and pedal edema. On abdomen examination, there was mild tenderness over the umbilical region. The patient had *Pitta* predominant *Kapha Prakriti* and had a past one year history of chronic cholecystitis with cholelithiasis, for this cholecystectomy was done. Subsequently, she was diagnosed for type 2 diabetes, metabolic syndrome and hypothyroidism with Vitamin B_{12} and iron deficiency. The patient was nonalcoholic and nonsmoker. There was no significant family history. When the patient came for consultation, the patient was on anticholinergic agents, antacids, levothyroxine, multivitamin along with iron, and antihistamine medicines since 1 year, but with not much relief [Table 1].

Ayurvedic management

The patient was assessed as per *Dashavidha Parikshya Bhava* (ten examination tools) before planning the *Panchakarma* treatment. No concurrent conventional medication was administered during this period except for hypothyroid. Ayurvedic management started with *Deepana-Pachana* (carminative and digestive) for 3 days with *Phaltrikadi* decoction,[14] *Shivakshara Pachana* powder,[15] and *Dhanyaka Siddha Jala* (medicated water prepared with coriander). On 4th day *Snehapana* (internal oleation) with *Tiktaka Ghrita*[16] in increasing dose was administered for 4 days. After assessing *Samyaka Snigdha Lakhsna* (signs of proper oleation), 3 days gap was given. In gap days, *Sarvanga Abhyanga* (external oleation) with *Kshirabala Taila*[17] and *Swedana* (fomentation) was done. On the 11th day, V*irechana* was administered with *Avipattikara* powder[18] after performing the ritual of prayer. The patient reported total 14 *Vegas* (stool frequency) along with *Samyaka Virechana Lakshana* (signs of proper purgation). *Samsarjana* (special light diet) was advised for 5 days according to type of *Shuddhi* (purification). After *Samsarjana*, *Madhutailika Basti*[19] was administered with classical *Putaka* (a brass nozzle attached to polythene bag) method for 5 days [Table 2].

Outcome

The patient was followed up telephonically for a period of 6 months. Effect of therapy was assessed based on physical symptoms, laboratory parameters, and quality of life (QoL) parameters. After therapy, the patient did not report the recurrence of pain till 6 months. Overall condition of the patient improved as there was weight loss, improvement in digestion, no pedal edema, and skin became radiant. The patient became self-dependent in carrying out routine of daily work without any lethargy. Body mass index decreased from 32.8 to 31.22 kg/m^2. Laboratory investigations showed significant changes like decrease of glycosylated hemoglobin from 6.4 to 5.98 within 3 weeks. IgG4 also showed slight decrease in level which was 4.65 before the treatment and 4.19 after the treatment. Lipid levels also showed a significant decrease within 3 weeks of Ayurveda treatment. QoL parameters[20] showed improvement after *Panchakarma* therapy as per SF-12 scale [Table 3].

Discussion

AIP with hypothyroidism, dyslipidemia, type 2 diabetes, fatty liver and obesity can be considered as multisystem involvement. In Ayurveda, it can be considered under umbrella cover of *Grahani Dosha*.

Agni has an important role in the physiological functioning of body. *Agni* by the virtue of *Sukshma Guna* (subtle in nature) converts *Ahara Dravya* (food particles) into *Ahara-Rasa* (essence of food) and with the help of *Dhatvagni* (tissue metabolism), the *Poshakansha* (nourishing part) is made available to body and thus digestion, absorption and assimilation is maintained which is important for the maintenance of life.[21]

Table 1: Course of the disease

Years	Incidence
April 2014	Patient had complaints of sudden, acute and intense pain in the upper right abdomen, nausea and vomiting. Patient was admitted in Dayton Gastroenterology Hospital, Sylvania Drive, Beavercreek, Ohio, USA. computerized tomography (CT) scan -abdomen and pelvis was done which revealed chronic cholecystitis with cholelithiasis and fatty infiltration of liver
May 2014	An episode of abdominal pain and admitted for further management in nuclear medicine (NM) gallbladder scan indicated as abnormal study, as the gallbladder is visualized but it has abnormal response to cholecystokinin (CCK), suggestion of gallbladder disease, liver function normal, billiary tract patent (May 9, 2014)
	Laparoscopic cholecystectomy was done (May 10, 2014)
	Patient had a relapse, physician advised procedure EUS (esophagogastroduodenoscopy) with biopsy and EUS (upper gastro-intestinal tract) finding revealed-pancreas showed several hyperechoic foci and strands with lobularity of parenchyma in head and body region. Pancreatic duct showed normal caliber with few hyperechoic margins in body region
	Postoperative diagnosis was chronic pancreatitis versus resolving acute pancreatitis (May 28, 2014)
June 2015	Patient developed sudden pain in abdomen, diarrhoea, nausea and was hospitalized. CT (abdomen/pelvis) non-contrast showed: no acute intra-abdominal or pelvic abnormality. Gall bladder was surgically absent. Liver, spleen, pancreas, kidney demonstrated a grossly normal appearance, complete blood count (CBC) with differential count was within normal limit, basic metabolic panel-normal, liver profile panel was normal, lipase -119 UL (normal range −0-60) IgA was within normal limit. IgG (subclass 4) - 116.3 mg/dl (4.0-86.4) and managed with conservative treatment
July 2015	Patient suddenly developed generalised pain in abdomen with cramps and constipation. Diagnosed as unspecified pancreatitis
	Lipase was high −82 U/L (normal 0-60) and amylase was normal. BMI was 31.76 kg/m^2
	Managed with conservative treatment and discharged
October 2015	Symptoms of acute pancreatitis reappeared and managed with lipase protease amylase and tetracycline. Oesophageal reflux was present. Patient was also diagnosed with type 2 diabetes. Mild rash developed on whole body due to allergy to these drugs
	BMI - 32.61 kg/m^2, IgG (subclass 4) - 106.9 mg/dl
December 2015	Acute pain in abdomen developed and was admitted to hospital and managed symptomatically. Patient was unwilling for admission due to repeated frequent relapses and so was looking for an alternative solution

CT: Computed tomography, NM: Nuclear medicine, CCK: Cholecystokinin, CBC: Complete blood count, BMI: Body mass index, EUS: Endoscopic ultrasound, IgG: Immunoglobin G

Table 2: Timeline of Ayurveda management

Period	Incidence/investigations	Interventions
December 30, 2015	Patient approached *Panchakarma* OPD of IPGT and RA Hospital, Jamnagar for exploring Ayurveda treatment option for her complaints. No acute features of pancreatitis were present, so planned for *Panchakarma* with precautionary measures	
December 31, 2015	On assessment of *Koshtha* (gut reactivity/behaviour), *Deepana-Pachana* (carminative-digestive) advised	*Deepana-Pachana* for 3 days with
	Investigations	*Phalatrikadi* decoction - 30ml twice a day (preferably 7 a.m.-7 p.m.) on empty stomach
	IgG - 4.56%, HbA1c - 6.4%, S. cholesterol- 160.2 mg/dl, serum triglycerides - 216.1 mg/dl, LDL - 70 mg/dl, HDL - 30 mg/dl, BMI - 32.8 kg/m^2	*Shivakshara Pachana* powder - 3 g twice a day after food with luke warm water *Dhanyaka Siddha Jala* (medicated water prepared with coriander seeds) to drink whenever patient feels thirsty
January 3, 2016	After *Deepana-Pachana, Snehapana* (internal oleation) was given	*Snehapana* with *Tiktaka Ghrita* Dose on 1st day - 30 ml, 2nd day - 45 ml, 3rd day - 60 ml and 4th day - 100 ml
January 7, 2016	On confirming *Samyaka Snehapana Lakshanas* (signs of proper oleation), 3 days of gap was given	*Abhyanga* (whole body massage) with *Kshirabala Taila* and *Swedana* (hot fomentation)
January 10, 2016	After completion of gap days on 4th day *Virechana* was administered	*Virechana* with *Avipattikara* powder (25 g) + honey
	Patient reported 14 *Vegas* (stool frequency) during *Virechana* and it was considered as *Madhyama Shuddhi* (moderate purification)	
January 11, 2016	According to type of *Shuddhi, Samsarjana Krama* (special light diet) was advised to the patient	Special diet of *Peya* (rice gruel), *Vilepi* (thick rice gruel), *Mudgayusha* (green gram gruel) and *Khichadi* (thick gruel prepared with rice and green gram lentil) was advised for 5 days gradually
January 16, 2016	On completion of *Virechana*, course of *Madhutailika Basti* for 5 days was administered	Ingredients of *Madhutailika Basti* are decoction of *Erandmoola* (roots of *Ricinus communis* L.) - 350 ml paste of *Guduchi* (*Tinospora cordifolia* (Thunb.) Miers.) - 15 g *Dhanvantara* oil - 45 ml Honey- 45 ml *Saindhava* (rock salt) - 5 g

Contd...

Table 2: Contd...

Period	Incidence/investigations	Interventions
January 21, 2016	There was weight loss, improvement in digestion, no pedal oedema and skin became radiant IgG - 4.19%, HbA1c - 5.98%, S. cholesterol - 155.75 mg/dl, S. triglycerides - 130.59 mg/dl, LDL - 99.12 mg/dl, HDL - 30.51 mg/dl BMI - 31.22 kg/m^2	*Samshamani Vati* (250 mg) 2 tablets twice a day after food for 3 months was advised with modifications in lifestyle

HbA1c: Glycosylated heamoglobin, LDL: Low density lipoprotein, HDL: High density lipoprotein, BMI: Body mass index, IgG: Immunoglobin G, S. Cholesterol: Serum cholesterol, S. triglycerides: Serum triglycerides

Table 3: Effect on quality of life parameters

QoL domains	Before treatment	After treatment
Physical health	32	60
Psychological	38	70
Social relationship	29	72
Environment	40	65

QoL: Quality of life

Grahani[12] is an anatomical structure situated above *Nabhi* (naval) and the physiological importance is due to its interdependence on *Agni*. Among various causes, improper lifestyle is the prime factor leading to impairment of *Agni* causing *Mandagni* (weak digestive power) which is the main pathology involved in *Grahani Dosha*.[22] *Agnimandya*, *Ama* (improperly processed food substance), and *Srotorodha* (obstruction in channels) are the basic events responsible for outbreak of any disease.[23] *Grahani Dosha* is the disease related with gastrointestinal disorder. This condition usually develops due to irregular dietary habit such as over-eating; more ingestion of cold, heavy, dry, fried and dehydrated food. A wide variety of gastrointestinal symptoms such as loss of appetite, abdominal pain, nausea, vomiting, and constipation is reported in *Grahani Dosha*.[24] When digested or indigested food is invariably voided. Through stool, the condition is described as *Grahani Roga*.[25] *Grahani Roga* is considered as the advanced stage of *Grahani Dosha*.

In treatment of *Grahani*, if *Pakwashayasth Doshas* remain in *Leenavastha* (the deep seated *Doshas*) then *Sramsana* (mild laxative) should be adopted with *Deepana Dravya* (carminative drugs). If *Sama Rasa Lakshanas* are produced, then *Langhana* (reducing therapy), *Deepana-Pachana,* and *Virechana* should to be adopted. After ascertaining symptoms of *Nirama Lakshana* (without association of *Ama*), *Agni* is stimulated with *Snehana* (oleation) therapy and *Niruha Basti* (type of medicated enema) can be administered.[26]

Due to unhealthy eating habits, *Agni* present in the body is unable to properly digest the food. This undigested food may convert to *Ama* which when combines with *Dosha* (*Vata, Pitta* and *Kapha*) leads to *Grahani Dosha*. Prolonged faulty eating habits can lead to the recurrent episodes of abdominal pain leading to chronicity of the condition. In AIP, *Dosha* (*Vikriti*) may stay in *Leenavastha* (inactive state) during non-acute condition.[26] To break the *Samprapti* (pathology) of the disease, the drugs must have *Deepana-Pachana* properties predominantly. Due to accumulation of *Dosha* in the *Grahani* in *Leenavastha*, it may not be treated by *Shamana* (medicines) only. The *Vikriti Doshas* are to be expelled from the body which may be achieved by *Virechana*[26] as the main stay of the *Panchakarma* therapy. In the present case study, *Virechana* was planned as the initial mode of the treatment.

Considering the *Dashavidha Pariksha Bhavas,* the combination of drugs for *Deepana-Pachana* was taken so that *Agni* may get stimulated on one hand with *Shivakshara Pachana* powder as it is having digestive and carminative property and has *Tridosha Shamaka* (pacification) effect.[15] On the other hand to remove the accumulated *Kleda*, *Phalatrikadi* decoction was administered as it is having *Kledahara* (removes impurities) property.[14] Medicated coriander water was advised during *Deepana-Pachana* as coriander is having *Pitta Shamaka* (alleviating *Pitta*) and *Deepana-Pachana* properties to prevent any acute attack of pancreatitis.[27] Primary goal to stimulate the *Agni* was achieved with *Deepana-Pachana* therapy due to which patient felt lightness in the body and had increased hunger.

Tiktaka Ghrita was selected for the *Snehapana* due to its virtue of alleviating *Pitta* and *Kleda*.[16] For *Abhyanga*, *Kshirabala Taila* was applied for proper oleation of the skin. *Avipatikara* powder was administered for *Virechana* as it is a mild, safe purgative, alleviating *Pitta* and has no reported adverse effect.[28] As the patient reported 14 *Vegas* during the *Virechana*, assessing *Shuddhi*, on the same day, *Peya* (rice gruel) was advised as first diet. On next day, two diets of *Vilepi* (thick rice gruel) were advised. On the 3rd day *Akrit Mudgayusha* (plain green gram gruel) twice a day was advised. On the 4th day, *Krit Mudgayusha* (green gram gruel simmered with *Ghrita*, cumin and salt) was advised as a diet. On the 5th day, *Khichadi* (thick gruel prepared with rice and green gram simmered with *Ghrita*, cumin and salt) was given. This procedure was followed to gradually increase *Agni* to bring the digestive system in a normal state.

Madhutailika Basti is a type of *Niruha Basti* and as per text can be given at any time to the persons with delicate physique, to remove *Dosha* as it has no complications, increases the strength and improves the complexion. Further no definite regimen needs to be followed during this course of *Basti*.[29] *Shatapushpa* (*Anethum sowa* Roxb. ex Fleming) *Kalka* (paste) in *Madhutailika Basti* was replaced with *Guduchi* (*Tinospora cordifolia.*(Thunb.) Miers.) *Kalka* to make the *Basti* milder so that the purpose of mild

Shodhana (purification) with *Pitta Shamana* can be achieved, as *Guduchi* is having *Snigdha Guna* (unctuous property), *Ushna Virya* (hot potency), and *Shamshamaniya* properties.[30] *Dhanwantaram* oil used in *Madhutailika Basti* has *Kapha-Pitta Shamaka* and *Kledahara* properties.[31]

As a result of all these, there was weight loss, improvement in digestion, and complexion of skin with feeling of lightness in the body. It is also worth mentioning that during the 21 days of *Panchakarma* therapy, the patient did not complain of any symptoms and tolerated the therapy without any adverse events.

Conclusion

The case report demonstrates clinical improvement in autoimmune pancreatitis (AIP) with *Panchakarma* therapy. As this is the single-case study, it may open a new path to the clinicians and researchers for exploring the *Panchakarma* option for the treatment of autoimmune pancreatitis(AIP).

Limitations

To establish the procedure as mode of treatment, large sample size should be taken. The procedure cannot be adopted in all patients as some patients may be averse to take *Ghrita*. This is an IPD procedure for which the patient needs to be admitted for 3 weeks which may not be feasible for all the patients. These therapies cannot be performed in acute conditions and *Basti* cannot be performed in patients with anal diseases.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for images and other clinical information to be reported in the journal. The patient understands that names and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

References

1. Sarles H, Sarles JC, Muratore R, Guien C. Chronic inflammatory sclerosis of the pancreas-an autonomous pancreatic disease? Am J Dig Dis 1961;6:688-98.
2. Yoshida K, Toki F, Takeuchi T, Watanabe S, Shiratori K, Hayashi N. Chronic pancreatitis caused by an autoimmune abnormality. Proposal of the concept of autoimmune pancreatitis. Dig Dis Sci 1995;40:1561-8.
3. Chari ST, Kloeppel G, Zhang L, Notohara K, Lerch MM, Shimosegawa T, *et al*. Histopathologic and clinical subtypes of autoimmune pancreatitis: The Honolulu consensus document. Pancreas 2010;39:549-54.
4. Kamisawa T, Chari ST, Lerch MM, Kim MH, Gress TM, Shimosegawa T. Recent advances in autoimmune pancreatitis: Type 1 and type 2. Gut 2013;62:1373-80.
5. Hamano H, Kawa S, Horiuchi A, Unno H, Furuya N, Akamatsu T, *et al*. High serum IgG4 concentrations in patients with sclerosing pancreatitis. N Engl J Med 2001;344:732-8.
6. Kamisawa T, Funata N, Hayashi Y. Lymphoplasmacytic sclerosing pancreatitis is a pancreatic lesion of IgG4-related systemic disease. Am J Surg Pathol 2004;28:1114.
7. Nishimori I, Tamakoshi A, Otsuki M; Research Committee on Intractable Diseases of the Pancreas, Ministry of Health, Labour, and Welfare of Japan. Prevalence of autoimmune pancreatitis in Japan from a nationwide survey in 2002. J Gastroenterol 2007;42 Suppl 18:6-8.
8. Kamisawa T, Takuma K, Egawa N, Tsuruta K, Sasaki T. Autoimmune pancreatitis and IgG4-related sclerosing disease. Nat Rev Gastroenterol Hepatol 2010;7:401-9.
9. Hart PA, Kamisawa T, Brugge WR, Chung JB, Culver EL, Czakó L, *et al*. Long-term outcomes of autoimmune pancreatitis: A multicentre, international analysis. Gut 2013;62:1771-6.
10. Horiuchi A, Kawa S, Hamano H, Hayama M, Ota H, Kiyosawa K. ERCP features in 27 patients with autoimmune pancreatitis. Gastrointest Endosc 2002;55:494-9.
11. Kim KP, Kim MH, Song MH, Lee SS, Seo DW, Lee SK. Autoimmune chronic pancreatitis. Am J Gastroenterol 2004;99:1605-16.
12. Acharya JT, editors. Sushruta Samhita of Sushruta, Uttar Tantra. Ch. 40, Ver. 169. 9th ed. Varanasi: Chaukhambha Orientalia; 2007. p. 709.
13. Acharya JT, editors. Charak Samhita of Agnivesha, Chikitsa Sthana, Ch. 15, Ver. 51-4. Reprint ed. Varanasi: Chaukhambha Orientalia; 2006. p. 516.
14. Tripathi IndraDev, Commentator. Chakradutt. Ch., Ver. 7. 4th ed. Varanasi: Chaukhambha Sanskrit Sansthan; 2002. p. 79.
15. Krishanandaji S. Rasatantrasara and Siddhaprayog Sangraha, Part – I. 18th ed. Ajmer: Krishna Gopal Ayurved Bhavan (D.T.); 2010. p. 332.
16. Paradkar HS, editor. Ashtang Hridayam of Vagbhata, Chikitsa Sthana. Ch. 19, Ver. 8-10. 2nd ed. Varanasi: Chowkhamba Krishnadas Academy; 2006. p. 711.
17. Paradkar HS, editor. Ashtang Hridayam of Vagbhata, Chikitsa Sthana. Ch. 22, Ver. 44. 2nd ed. Varanasi: Chowkhamba Krishnadas Academy; 2006. p. 731.
18. Shastri RD, editor. Bhaisajyaratnavali of Shri Govind Das. Ch. 56, Ver. 25-29. 19th ed. Varanasi: Chaukhambha Prakashana; 2008. p. 922.
19. Acharya JT, editors. Sushruta Samhita of Sushruta, Chikitsa Sthana. Ch. 38, Ver. 96-101. 9th ed. Varanasi: Chaukhambha Orientalia; 2007. p. 547.
20. World Health Organization. Division of Mental Health. WHOQOL-BREF: Introduction, Administration, Scoring and Generic Version of the Assessment: Field trial Version. Geneva: World Health Organization; 1996. Available from: https://apps.who.int/iris/handle/10665/63529. [Last accessed on 2015 Dec 02].
21. Acharya JT, editors. Sushruta Samhita of Sushruta, Sutra Sthana. Ch. 35, Ver. 27. 9th ed. Varanasi: Chaukhamba Orientalia; 2007. p. 154.
22. Acharya JT, editors. Sushruta Samhita of Sushruta, Uttar Tantra. Ch. 40., Ver. 166., 9th ed. Varanasi: Chaukhamba Orientalia; 2007. p. 709.
23. Paradkar HS, editor. Ashtanga Hridayam of Vagbhatta, Nidana Sthana, Ch. 11, Ver. 1. 2nd ed. Varanasi: Chaukhamba Krishnadas Academy; 2006. p. 513.
24. Acharya JT, editors. Chakrapanidatta Commentary on Charaka Samhita of Agnivesha, Chikitsa Sthana. Ch. 15, Ver. 58-72. Reprint ed. Varanasi: Chaukhamba Orientalia; 2006. p. 518.
25. Dash B, Sharma RK, editors. Chakrapanidatta Commentary on Charaka Samhita of Agnivesha. Vol. 4. Ch. 15, Ver. 57. Reprint ed. Varanasi: Chaukhamba Sanskrit Series Office; 2012. p. 29.
26. Acharya JT, editors. Charak Samhita of Agnivesha, Chikitsa Sthana. Ch. 15, Ver. 73-78. Reprint ed. Varanasi: Chaukhamba Orientalia; 2006. p. 518.
27. Mishra BS, editor. Bhavaprakash Nighantu of Bhava Mishra, Haritakyadi Varga, Ver. 86-88. Varanasi: Chaukhamba Sanskrit Sansthan; 2004. p. 33.
28. Paradkar HS, editor. Ashtanga Hridayam of Vagbhatta, Kalpa Sthana. Ch. 2, Ver. 21-3. 2nd ed. Varanasi: Chaukhamba Krishnadas Academy; 2006. p. 743.
29. Acharya JT, editors. Sushruta Samhita of Sushruta, Sutra Sthana. Ch. 38, Ver. 96-99. 9th ed. Varanasi: Chaukhamba Orientalia; 2007. p. 547.
30. Mishra BS, editor. Bhavaprakash Nighantu of Bhava Mishra, Guduchyadi Varga. Ver. 6-10. 11th ed. Varanasi: Chaukhamba Sanskrit Sansthan; 2004. p. 269.
31. Paradkar HS, editor. Ashtanga Hridayam of Vagbhatta, Chikitsa Sthana. Ch. 12, Ver. 24. 2nd ed. Varanasi: Chaukhamba Krishnadas Academy; 2006. p. 679.