

“Urustambha” – Aortoiliac occlusion with Metabolic syndrome?

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Abstract

Urustambha is a grave condition, in which the patient's thighs become painful, numb and immobile. *Urustambha* is a disease which is not amenable to *panchakarma* (five evacuative procedures) treatment. Till date, there is no clear understanding of the concept of *Urustambha* and its clinical application. This article is aimed to understand the concept of *Urustambha* and its correlation with relevant modern pathology or disease. *Urustambha* is a lifestyle disease and it is commonly seen in higher socioeconomic status. *Urustambha samprapti* resembles with atherogenesis. *Diva swapna* and *raatri jaagarana* explained in *Urustambha nidaana* may indicate obstructive sleep apnea (OSA). Clinical presentation of *Urustambha* may be unilateral or bilateral or both. *Charaka's* version of *Urustambha* indicates vascular pathology like “aortoiliac occlusion” with an underlying “metabolic syndrome (MS),” whereas *Sushruta's* version of *Urustambha* indicates inflammatory pathology of spinal cord like “acute transverse myelitis” or “inflammatory myelopathy” or “infectious myelitis.” Principles of *Urustambha* are applicable for the prevention and management of the conditions like atherosclerosis, MS, OSA, aortoiliac occlusion, diabetes mellitus, obesity, cardiovascular pathology, acute myelopathy and other ischemic and inflammatory spinal diseases.

Key words: Aortoiliac occlusion, atherosclerosis, metabolic syndrome, myelopathy, obstructive sleep apnea, *Urustambha*

INTRODUCTION

The word “*Urustambha*” is made up of “*Uru* (thigh)” and “*Stambha* (cramping/spasticity/rigidity/stiffness).” *Urustambha* is a grave condition, in which the patient thighs become painful, numb, and immobile.^[1] *Urustambha* is a disease which is not amenable to *panchakarma* (five evacuative procedures) treatment.^[2] In this condition, the *panchakarma* procedures are useless and should not be administered.^[1]

The deranged *vayu* (which controls the functions of nervous and musculoskeletal systems), surcharged with the local fat and *kapha* (liquid or nourishing material of the body) gives rise to a painful condition in the region of the thigh which is known as *Urustambha*; others designate it as “*Adhya vata*” (disease of wealthy people). This disease is marked by lassitude, aching pain in the limbs and sensation of coldness, heaviness, numbness, and unsteadiness of thighs.^[3]

There is a conversation found between disciple *Agnivesha* and his preceptor *Acharya Punarvasu Atreya* in *Charaka samhita* and

according to that, *Agnivesha* asked a question to *Acharya Atreya* as, “Oh! Lord, all the five purification therapies (*panchakarma*) are described to treat various diseases in *Ayurveda*. Is there any curable disease where *panchakarma* procedures are contraindicated?” for this question *Acharya Atreya* replied as “there is such a disease for which *panchakarma* is contraindicated and it is called *Urustambha* (spasticity of thighs).” *Agnivesha* again enquired about the etiology, symptomatology, and treatment of *Urustambha* and preceptor gave a detailed explanation to all the questions asked by *Agnivesha*.^[4]

The detailed description of *Urustambha* is available in “*Charaka samhita*,” 27th chapter of *chikitsa sthana*, “*Urustambha chikitsitam adhyaya*,”^[4] whereas in “*Sushruta samhita*” description regarding *Urustambha* can be seen

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in the 5th chapter of *chikitsa sthaana*, “*maha vata vyadhi chikitsitam adhyaya*.”^[3] In “*Ashtanga hridaya*,” *Urustambha* is described in 15th chapter of *nidaana sthaana*, “*vata vyadhi nidaana adhyaya*.”^[5] According to *Sushruta Acharya*, “*Aadhya vata* (commonly seen in higher socioeconomic class),” “*Urustambha* (stiffness of thighs),” and “*Kapha medo avrita vata* (*vata* obstructed by *kapha* and *medas* [fat])” are used synonymously.^[3]

Till date, there is no clear understanding of the concept of *Urustambha* and its clinical application. The previous studies considers *Urustambha* as, “paralysis of thighs,”^[6] “spastic paraplegia,”^[7] “myopathy/muscular fatigue,”^[8] “transverse myelitis,”^[9] and “chronic rheumatoid arthritis of hip joint.”^[10] There are plenty of causes like neurological (spinal cord injury/inflammation/infection), muscular, articular (hip joint pathology) and vascular (occlusion of iliac artery) which may affect the thighs and causing impairment. Among these, cause/condition/pathology which one is more suitable to *Urustambha* is not clear. The present article aims at the better understanding of *Urustambha* with modern correlation.

REVIEW METHODOLOGY

Ayurvedic material related to “*Urustambha*” collected from major *Ayurvedic* texts with their commentaries such as *Charaka samhita*, *Sushruta samhita*, *Ashtanga sangraha*, *Ashtanga hridaya*, and *Madhava nidaana*. Electronic databases “Google scholar search” and “Google search” were searched for relevant studies and reviews published until August 2016, irrespective of their appearance/publication year. The keywords used for search were “*Urustambha*,” “atherosclerosis,” “aortoiliac occlusion,” “metabolic syndrome (MS),” “antiobesity,” “antidiabetes,” “acute transverse myelitis (ATM),” “myelopathy,” “obstructive sleep apnea (OSA)” “atherogenesis,” and “inflammatory spinal diseases.” Abstracts and full-text articles which are freely downloadable and in English language were only included in the study.

Similarity between *Urustambha* and Aortoiliac Occlusion (AIO) with Metabolic Syndrome

There is no direct or exact correlation of *Urustambha* with any modern disease or condition available but it was found that there are so many similarities in various aspects like etiology, pathology, symptomatology, course and prognosis and management in between the two conditions, “*Urustambha*” and “AIO with MS.”

Nidaana (Etiology) of *Urustambha* and Its Similarity with Atherogenic Food/Factors

Excessive and regular intake of food items such as *snigdha* (unctuous/fatty), *ushna* (hot/spicy), *laghu* (light), *sheeta*

(cold), *drava* (liquid) and *sushka* (dry) *ahaara* (food) and eating various incompatible foods especially when the previous meal is not digested; *dadhi* (yoghurt), *ksheera* (milk), etc., dairy products and meat of *graamyas* (domesticated animals), *anupa* (animals inhabiting marshy land) and *audaka* (aquatic animals); intake of *pishtaanna* (food items prepared by flour/less fiber diet) and *madya* (alcohol); various other factors such as *diwaswapna* (sleeping during day), *prajaagara* (awakening during nights), *langhana* (fasting), *adhyashana* (excessive food intake), *aayaasa* (over exertion/stress), *bhaya* (fear), and *vega dhaarana* (suppression of natural urges) are the etiological factors of *Urustambha*.^[4]

Aahaara (dietary factors)

The diet explained in *Urustambha nidaana* is similar with “atherogenic diet”/“high calorie diet”/“diet causing MS.” Atherosclerosis is a generic term used to define the thickening of arteries by the formation and deposition of an atherosclerotic plaque. The plaque is a fatty fibrous growth which becomes calcified ultimately and leads to blockage of an artery.^[11]

Atherogenic diet which is high in saturated fats, cholesterol, processed foods and the factors such as dyslipidemia, hypertension, obesity, and physical inactivity are the major risk factors for causing vascular diseases.^[12] Saturated fats are considered as “bad” fats because they increase low-density lipoprotein (LDL) cholesterol, fatty cuts of lamb, pork, beef fat, lard, bacon, whole milk and whole milk dairy products such as butter, cheese and yoghurt, etc.; food contains saturated fats.^[12] Transient reductions in endothelial function have previously been reported after a high-fat meal.^[13]

Madya (alcohol)

Excessive intake of alcohol is a known factor to cause atherosclerosis. Regular consumption of excessive amount of alcohol is a prominent risk factor for early atherogenesis, surpassing even the effect of heavy smoking. Adverse and beneficial effects of alcohol in causing arterial disease are mediated in part by dose-dependent promotion or deceleration of atherogenesis.^[14]

Bhaya and *Aayaasa* (psychological factors)

Fear, anger, and grief like factors precipitate myocardial ischemia and infarction. Stress is associated with cardiovascular morbidity and mortality by inducing vasoconstriction. Hostility like psychological stress carries an increased risk for atherosclerotic vascular disease. Acute mental stress induces endothelial dysfunction and atherosclerotic vascular disease.^[15] Exaggerated cardiovascular reactivity to mental stress is hypothesized

to increase atherosclerotic risk.^[16] Psychological stress accelerates the atherosclerotic process.^[17] Chronic stress is associated with accelerated progression of atherogenesis by impairing endothelium dependent vascular homeostasis. Mental stress is known to result in rapid changes in systemic hemodynamics mediated by sympathetic activation. Even short-lived episodes of mental stress encountered frequently during normal daily life leads to a vascular abnormality relevant to early atherosclerosis.^[13]

Vega Dhaarana (suppression of natural urges)

Suppression natural urges like urination or defecation may increase bladder pressure and/or intra-abdominal pressure (IAP). Increased IAP is associated with cardiovascular, renal and pulmonary dysfunction. Respiratory derangement result as the elevated diaphragm due to increased IAP which decreases functional residual capacity and increases airway pressure. Cardiovascular compromise results from decreased venous return from compression of heart and the inferior vena cava due to increased IAP. Increased IAP may be a cause for systemic hypertension in central obesity.^[18,19] Thus, it can be predicted that *vega dhaarana* indirectly may play a role in vascular pathology.

Whether *Diwaswapna* and *Prajaagara* Mentioned in *Urustambha Nidaana* Indicates OSA?

OSA is a common condition characterized by recurrent episodes of cessation of respiratory airflow caused by upper airway inspiratory collapse during sleep with a consequent hypoxemia and decrease in oxygen saturation as well as sleep fragmentation and sleep deprivation.^[20] OSA is associated with endothelial damage/dysfunction, atherosclerosis, inflammation, oxidative stress, vascular smooth muscle activation, platelet activation, hypercoagulability, and thrombosis.^[21] Excessive daytime sleepiness and decreased sleep duration are associated with metabolic abnormalities. Sleep deprivation may raise blood pressure, diabetes, glucose intolerance, overweight, obesity, activate systemic inflammatory response, and insulin resistance. Sleep deprivation indirectly (through obesity) or directly implicated as a risk factor for MS.^[20]

OSA may accelerate atherosclerosis by exacerbating key atherogenic risk factors. OSA is associated with surrogate markers of premature atherosclerosis.^[22] The weight of evidence suggests that OSA and MS coexists. Relationship between sleep and the MS goes beyond OSA and extends to other forms of sleep disturbances especially sleep deprivation.^[20]

In *Urustambha nidaana*, *diwaswapna* (excessive daytime sleepiness) and *prajaagara* (night time awakening or disturbed sleep during nights) are mentioned. The person may be getting excessive sleep during day time may be

because of disturbed sleep during nights. The disturbed night sleep indicates OSA which again is associated with MS and atherosclerosis. It seems that *diwaswapna* and *prajaagara* play a key role in causing MS with atherosclerosis.

Why *Urustambha* is called as “*Aadhya Vata*?”

The word “*Aadhya*” means “rich/wealthy/opulent.” *Urustambha* is commonly seen in wealthy people or higher socioeconomic class, as they can afford the high-calorie diet which is mentioned in *Urustambha nidaana*.^[3] This might be the reason for calling *Urustambha* as “*Aadhya roga/Aadhya vata*.”

Excessive consumption of pro-atherogenic foods such as total visible fat, milk and milk products, meat, eggs, and also sugar were significantly increased in higher social classes. Mean body mass index (BMI), obesity, overweight, central obesity and sedentary lifestyle were also more significantly found in higher social classes compared to lower social classes in India. Hypertension and coronary artery disease risk increases in higher social classes and these problems are more common among wealthier groups with sedentary occupations consuming high-fat diets. It is possible that the higher cost of these foods was not within the limits of poor socio-economic groups.^[23] According to the rapid pace of economic and demographic changes in India has ushered marked nutritional and lifestyle changes. The diets in the urban and semi-urban areas contain more calories and saturated fats with less fiber. Obesity and the MS are becoming increasingly prevalent in the urban areas of India. These changes are conducive to the development of early-onset type 2 diabetes mellitus and accelerated atherosclerosis.^[24] Middle to high socioeconomic status significantly contributed to increased risk of MS.^[25] As MS and atherosclerosis are most commonly seen high socioeconomic class, *Urustambha* might also more prevalent in affluent societies by observing this fact *Ayurvedic Acharya's* might have called *Urustambha* as “*Aadhya roga*.”

Metabolic syndrome (MS)

MS is characterized by abdominal obesity (increased waist circumference), elevated triglycerides, decreased high-density lipoprotein (HDL cholesterol), high blood pressure, and increased fasting glucose. Other abnormalities have also been noted in individuals with MS which includes systemic inflammation, endothelial dysfunction, oxidative stress, and hypercoagulability.^[20]

By considering all the above facts, it can be said that various pro-atherogenic factors which are described in *Urustambha nidaana* when clustered together in the same individual may cause MS with atherosclerosis. There is marked similarity found between *Urustambha nidaana* and pro-atherogenic factors in terms of etiology [Table 1].

Table 1: Similarity between *Urustambha nidaana* and pro-atherogenic food/factors

<i>Urustambha Nidaana</i> ^[4]	Factors causing <i>Urustambha</i> ^[4]	Pro-atherogenic food/factors; Factors causing metabolic syndrome
Synonym	<i>Aadhya roga</i>	Sedentary life style/metabolic syndrome/ atherosclerosis most commonly seen in higher socioeconomic class ^[23-25]
<i>Aahaara</i>	<i>Snigdha, ksheera, dadhi, pishtanna, graamya, anupa, audaka mamsa Laghu, sheeta and sushka ahaara</i>	High calorie diet/diet rich in saturated fat/dairy products/pro-atherogenic diet/diet lacking fiber ^[12,13] Leads to <i>vata prakopa</i> (aggravation of <i>vata dosha</i>) which may induce sclerosis (hardening) of arteries/hypertension?
<i>Madya</i>	<i>Langhana</i> (fasting) and <i>Adhyashana</i> (excessive food intake)	Excessive food intake with fasting may leads to obesity/diabetes/insulin resistance/metabolic syndrome?
<i>Diwaswapana and Prajaagara</i>	<i>Vyapanna madya/dushita madya</i> (spoilt wine)/ <i>ati madya sevana</i>	Excessive alcohol consumption leads to atherosclerosis ^[14]
<i>Aayaasa and Bhaya</i>	Excessive day time sleep and night awakening	Sleep disturbances and sleep deprivation are associated with metabolic syndrome and atherosclerosis; obstructive sleep apnea is associated with metabolic syndrome and atherogenesis ^[20-22]
<i>Vega dhaarana</i>	Excessive stress/strain, fear/mental stress	Mental stress is known to cause ischemia, vasoconstriction, endothelial dysfunction and atherosclerosis ^[13,15-17]
	Suppression of natural urges	Which may increase intra abdominal pressure and leads to systemic hypertension/ atherosclerosis? ^[18,19]
All the above factors together will cause <i>Urustambha</i> ^[4]		All these factors may leads to metabolic syndrome (characterized by obesity, diabetes, hyperlipidemia, hypertension, hypercoagulability, endothelial dysfunction and atherosclerosis)

Samprapti (Pathogenesis) of *Urustambha* and Its Similarity with Atherogenesis

Urustambha is defined as, *kapha* associated with *medas* (fat) influences *vata* and *pitta* (which is related to heat and metabolism) to cause spasm/spasticity of the thighs. Due to unctuousness, the *Ama* (a product of altered digestion and metabolism) located in the gastrointestinal tract in association with fat obstructs the flow of *vata* (*vyana vata* which is responsible for blood flow). Because of heaviness, it descends to the thighs through the downward moving vessels. Being provoked by powerful *medas* (fat) these *dosha's* (morbid material) fill up the lower limb/limbs including the thighs and calf region to cause spasm and immobility in these parts. A pond which is large, deep and full of water, remains motionless, stable and unagitated, similarly the *kapha* which is located in thighs remains motionless, stable and unagitated.^[4]

Atherosclerosis in the peripheral arteries is a chronic and slowly progressive condition (just like a pond full of water is stable, motionless, unagitated) which causes narrowing of the arteries. While many patients will remain asymptomatic throughout life on the degree of narrowing a range of severity

of symptoms may occur. Atherosclerosis occurs much less frequently in the arteries of upper extremity compared with lower extremity. Obesity and high total cholesterol are independently related to an increased risk of lower extremity artery disease.^[26] The atherosclerotic plaque is a fatty fibrous growth that ultimately becomes calcified and leads to blockage of the artery.^[11] Atherosclerosis is a dynamic disease process characterized by vessel wall remodeling that occurs over decades. Obesity, diabetes, hyperlipidemia have a major impact on the progression of atherosclerosis.^[22] The natural history of aortoiliac occlusive disease is slow progression proximally and distally overtime to end in complete occlusion of the aorta and iliac arteries.^[27]

Considering the above facts, the atherogenesis process in aortoiliac arteries is similar to *Urustambha samprapti* [Table 2].

Lakshana's (Signs and Symptoms) of *Urustambha* and Its Similarity with Aortoiliac Occlusion

Excessive fatigue of the calf muscles and thighs, constant pain with burning sensation, feeling pain while putting the

Table 2: Similarity between *Urustambha samprapti* and atherogenesis

<i>Urustambha samprapti</i>	Thrombosis/Aortoiliac occlusion
“ <i>Snehat cha amam</i> (<i>ama</i> due to its unctuousness) <i>chitam koshte</i> (accumulated at gut)”	Hypercoagulability/platelet aggregating factors/post prandial hypercoagulability/dysfunctioning of various coagulation factors?
“ <i>Medasaan saha vataadeen</i> ” (the above accumulated <i>ama</i> along with <i>medas</i> , <i>kapha</i> , <i>vata</i> and <i>pitta</i>)	<i>Medas</i> Hyperlipidemia/dyslipidemia/elevated triglycerides? Fatty fibrous growth inside a vessel? <i>Kapha</i> Increased fasting glucose? Hypercoagulability states? <i>Pitta</i> Inflammation of endothelium? Oxidative stress? <i>Vata</i> Fibrosis? Sclerosis? Hypertension? Calcification of atherosclerotic plaque? Inducing vasoconstriction?
“ <i>Gauravaat uru yaati adhogai siraadibhi</i> ” (due to heaviness the above morbid matter descends towards thighs through downward going vessels)	<i>Gauravaat yaati</i> Atherosclerosis occurs more frequently in lower limbs compared to upper limbs; <i>Adhogai siraadibhi</i> Descending aorta/abdominal aorta/iliac artery/femoral artery/popliteal artery?
“ <i>Rudhwaa</i> ” and “ <i>poorayan sakthi jangha uru</i> ” (obstructing and filling lower limb, calf and thigh regions)	<i>Rudhwaa</i> Obstructing the vessel/occlusion? <i>Poorayan sakthi</i> Aorta+common iliac/aortoiliac occlusion? <i>Poorayan uru</i> Aortoiliac/external iliac occlusion? <i>Poorayan jangha</i> Femoro-popliteal occlusion?
“ <i>Medo bala utkata</i> ” (predominance of <i>medas</i>)	Formation of thrombus/occlusion of an artery by thrombosis/ atherosclerosis/occlusion of an aortoiliac artery?
“ <i>Tishtati sthiram</i> ” (stable) “ <i>Akshobhyam</i> ” (unagitated) “ <i>Stimitam</i> ” (silent)	Atherosclerosis Chronic progressive condition Dynamic disease process occurs over decades Asymptomatic (symptomatic only when total occlusion of an artery or collateral circulation deficit) Calcification of the atherosclerotic plaque and complete or partial occlusion of an artery

feet on the ground, insensitivity to cold touch, lack of control over the functions like standing, pressing the feet against the ground and movement of the lower limb. The patient feels that his limb/limbs propelled by someone else (they does not belong to him) and suffers with severe pain as they are broken. *Dosha's* situated at thighs produce stiffness and coldness. Patient suffers with heaviness, fatigue, stiffness, burning sensation, pain, numbness, tremor, tearing/breaking pain, pulsatile pain, and pricking pain at thigh or calf or whole lower limb in *Urustambha*. *Dosha's* along with *medas* will cause loss of control and walking difficulty.^[4]

Atheroma is the main cause for chronic arterial occlusion. Pain is the key symptom of arterial occlusion irrespective of its site. Intermittent claudication (“Claudio” means “I limp”) is the most common complaint of the limb due to chronic arterial occlusion. Some degree of atherosclerosis is always seen in aortoiliac occlusion.^[28] Intermittent claudication is a

cramping pain and it is a symptom of atherosclerosis causing inadequate blood flow to the leg muscles.^[29] In intermittent claudication buttocks, thighs and calves get involved. Claudication may be symmetric or asymmetric depending on the pattern of involvement of the iliac arteries.^[28]

Atherosclerosis is the major cause for peripheral artery disease (PAD). Symptoms of PAD can be typical (claudication) or atypical. The severity of symptoms of claudication depends on the amount of stenosis, collateral circulation. Patient with aortoiliac occlusive disease present with buttock, hip and thigh claudication and may be associated with weakness of hip and thigh on walking. Functional capacity gets diminished in the patients of PAD.^[30]

Acute aortic occlusion (AAO) present with predominantly neurological symptoms due to spinal cord ischemia. AAO patient can be presented as symmetrical paraplegia with

absent reflexes, numbness with severe weakness of both legs. In AAO, paraplegia is caused by occlusion of the aorta either above or below the level of an artery Adamkiewicz, leading to serious cord ischemia and infarction or causing ischemia of the peripheral nerves and musculature distal to the occlusion, respectively. AAO is generally mistaken as neurological disorders and is missed up to 50% of cases presenting with paraplegia.^[31]

Complete occlusion of an artery without adequate collaterals is characterized by the six Ps, pain, pallor, pulselessness, paresthesia, paralysis, and prostration.^[32] The classic five P's, pain, pallor, pulselessness, paralysis, and paresthesia can be diagnostic of AAO.^[31] Even though pallor and pulselessness are not found in *Urustambha lakshanaa*, it can be assumed that predominance of *kapha* and *medas* will cause pallor and pulselessness also along with pain, paresthesia, and paralysis.

By considering the above facts, it seems that signs and symptoms of aortoiliac occlusion or AAO are similar to *Urustambha lakshanaa* [Table 3].

DIFFERENTIAL DIAGNOSIS

There is a lot of confusion prevailing regarding the concept of *Urustambha* and its clinical application. The previous studies compared *Urustambha* as, “paralysis of thighs,”^[6] “spastic paraplegia,”^[7] “myopathy/muscular fatigue,”^[8] “transverse myelitis,”^[9] “chronic rheumatoid arthritis of hip joint,”^[10] and “spasticity of thighs.”^[11] While comparing *Urustambha* with a modern condition, previous studies concentrated only on similarity of the symptomatology and ignored to consider similarity in etiology, pathogenesis, and treatment

while comparison. Most of the studies, jumped to concluding *Urustambha* as paraplegia or transverse myelitis based on similarity of clinical presentation (loss of function and pain in both lower limbs) only without considering the etiological, pathological similarities and also without differentiating it from *vata vyadhi* which effects both lower limbs such as “*Pangu vata*,” “*Adharaanga vata*,” “*mamsa gata vata*,” “*saama vata*” and “*avrita vata*”.

Pangu Vata

Vata gets vitiated and affects one major *kandara* (tendon) in the leg. This causes very severe pain and spasm/spasticity of muscles. The affected person cannot walk properly and starts limping. This condition is called “*Khanja vata*.” When both lower limbs involved, the person becomes paralyzed and this condition is known as *Pangu vata* (paraplegia/spastic paraplegia?).^[33]

Is *Urustambha* Unilateral?/Bilateral?

Whether *Urustambha* occurs in one lower limb or both is not clearly mentioned in *brihatrayee* (three major *Ayurvedic* classical texts – *Charaka samhita*, *Sushruta samhita* and *Ashtanga hridaya*). Authors who have compared *Urustambha* with transverse myelitis,^[9] spastic paraplegia,^[7] paralysis of thighs,^[6] and myopathies/muscular fatigue,^[8] etc., were considered *Urustambha* as a bilateral condition (involvement of both lower limbs). In *Madhava Nidaana*, *Urustambha* is considered as a bilateral^[34] but in *Charaka samhita*^[4] and *Sushruta samhita*^[3] it is not clearly mentioned whether *Urustambha* is symmetrical or asymmetrical or both. *Urustambha* may affect one lower limb (*sakthi*) or either thigh (*uru*) and/or *jangha* (calf) according to *Charaka*

Table 3: Similarity between *Urustambha lakshanaa*'s and aortoiliac occlusion

<i>Urustambha lakshanaa</i> 's ^[4]	Aortoiliac occlusion ^[28] /AAO
<ul style="list-style-type: none"> • <i>Avidheya paris pandam</i> (loss of control over limb) • <i>Alpa vikrama</i> (walking difficulty) • <i>Aayaasa</i> (fatigue) • “<i>Chaalane aneeshwara</i>” (unable to walk) • <i>Ruk</i> (pain) • <i>Bheda</i> (tearing/breaking pain) • <i>Toda</i> (pricking pain) • <i>Sphurana</i> (pulsatile pain) • <i>Adaaha vedana</i> (pain without burning sensation/pain with excessive burning sensation) • “<i>Sambhagna iva vedana</i>” (breaking pain) • <i>Daha</i> (burning sensation) • <i>Supti</i> (numbness) • “<i>Sheetam sparsham na veti</i>” (insensitivity to cold touch) • <i>Gaurava</i> (heaviness of limb) • <i>Sankocha</i> (spasm/stiffness) • <i>Kampana</i> (tremors) • <i>Stambha</i> (stiffness/spasticity) • <i>Shaityata</i> (coldness) 	<p>Five P's: Paralysis</p> <p>Pain</p> <p>Paresthesia</p> <p>Pulselessness and pallor: Ischemia/ Claudication/aortoiliac disease/PAD/AAO due to spinal cord ischemia</p>

AAO: Acute aortic occlusion, PAD: Peripheral artery disease

Acharya.^[4] By considering this, it can be assumed that clinical presentation of *Urustambha* may be unilateral or bilateral or both.

Unanswered Questions in Previous Works

Whether the conditions such as “spastic paraplegia,”^[7] “transverse myelitis,”^[9] “myopathy,”^[8] and “rheumatoid arthritis of hip joint”^[10] can be seen only in higher socioeconomic class as *Urustambha* (*aadhya roga*)? Various etiological factors explained in *Urustambha nidaana* (high calorie diet, atherogenic factors, etc.) can produce the above conditions? Whether *Urustambha samprapti* (occlusion of a descending artery/vessel which is a slow, chronic and silent process as explained in *Urustambha samprapti*) is traceable in above conditions? Whether *Urustambha chikitsa* (exercise therapy or dry/rough procedures etc.) is suitable for the above conditions? Whether a person suffering with paraplegia or transverse myelitis or myopathy can do the vigorous exercises which are explained in *Urustambha chikitsa* (such as sand walking, swimming, walking against tides, etc.)? How the above conditions like paraplegia or transverse myelitis or myopathy etc. are diagnosed as *Urustambha* and differentiated from “*pangu vata*,” “*adharaanga vata*,” “*mamsa gata vata*,” “*saama vata*” and “*avrita vata*” etc.? In absence of early intervention *Urustambha* leads to death (complication); whether the above conditions are fatal if not treated early? *Urustambha* is unilateral?/Bilateral?/Both?

Urustambha Chikitsa (Treatment)

As *kapha* and *ama* both are predominant in the pathogenesis of *Urustambha*, the treatment should be focused mainly on *kshapana* (complete extraction) and *shoshana* (absorption/drying of the liquid fraction). The patient of *Urustambha* should receive *rooksha* (dry/ununctuous) treatment regularly. Foods such as *Yava* (*Hordeum vulgare*) and *Shyamaka* (millet) cooked along with vegetables without salt are indicated in *Urustambha*. Administration of *Kshara* (alkali preparations), *Arishta* (medicated wines) and *Hareetaki* (*Terminalia chebula*) along with honey and *Pippali* (*Piper longum*) are indicated in *Urustambha chikitsa*. Various external procedures such as *utsaadana* (rubbing of dry/medicated powder/massage), *pralepana* (external application of medicated paste), and *parisheka* (sprinkling/pouring of decoction over the affected area) are also explained in *Urustambha chikitsa*.^[3,4]

Contra-indication of *Panchakarma* in *Urustambha*

Panchakarma procedures (five major cleansing procedures) such as *vamana* (therapeutic emesis), *virechana* (therapeutic purgation), and *vasti* (medicated enema's) are contra-indicated in *Urustambha*. Therapies like *snehana* (unctuous) and *vasti* (oil enema) aggravates *kapha*. *Virechana* is also

ineffective to remove *kapha* which is localized in the thighs. In *Urustambha*, *ama*, *kapha* and *medas* which are lodged/firmly located in thighs, it is impossible to eliminate them by the above mentioned *panchakarma* procedures. Because of continuous *rooksha* (dry/rough/ununctuous) *chikitsa*, if pain and stiffness aggravates in a *Urustambha* patient, procedures such as *sneha* (oil massage), *sweda* (fomentation), and *vasti* (oil enema) may be done according to the condition even though these procedures are contra-indicated in *Urustambha chikitsa*.^[3,4]

Exercise Therapy in *Urustambha*

Various exercises are mentioned in the management of *Urustambha*. To alleviate *kapha*, the patient of *Urustambha* should be engaged in vigorous physical exercise and patients are made to walk over the ground covered with gravel and sand in the mornings. The patient should swim against tides/water currents in a river or pond. Swimming should be done frequently in a pond which is having clean and stable water and also free from dangerous aquatic animals. All these exercises should be performed according to the patient's physical strength and stamina.^[4]

Exercise Therapy in “Intermittent Claudication”/“MS”/“PAD”

Exercise is useful in increase HDL levels, to reduce triglycerides and LDL, to reduce blood pressure (both systolic and diastolic), to reduce hemoglobin A1C levels, to reduce weight, and also to reduce cardiovascular risk factors.^[35] In aortoiliac occlusive disease daily exercise regimen can significantly alleviate the symptoms and helps to regain functional capacity. An increased tolerance to demand ischemia may be a probable mechanism for such observed improvement.^[27] In PAD, a supervised exercise program is recommended for a minimum period of 30-45 min at least 3 times a week for a minimum of 12 weeks.^[30] Daily exercise to the point of claudication not only increases the walking tolerance but also enhances collateral circulation. If walking is not feasible, a similar indoor exercise may be advised.^[28] Exercise (walking to the level that causes pain) can relieve intermittent claudication for many people. Exercise found even better than the angioplasty and other forms of surgery in intermittent claudication.^[29]

Physical activity is an effective management for patients with claudication. Exercise training increases the average walking distance to pain onset and also improvement in the average distance to maximum tolerated pain. The greatest improvement by exercise training occurred when patients trained to maximum tolerated pain for at least a period of 6-month duration by keeping walking as the primary mode of exercise. Exercise training is found superior than peripheral angioplasty in claudication and also it improves the walking distance better than the widely used medicines.^[35]

Urustambha chikitsa resembles with the management of the conditions like Atherosclerosis, MS, PAD and aortoiliac occlusive disease with intermittent claudication [Table 4].

Sushruta's Version of *Urustambha*

Acharya Sushruta has explained *Urustambha* in the 5th chapter of *chikitsa sthaana*, “*Maha vata vyadhi chikitsitam adhyaya*.” Sushruta used the terms “*Kapha medo avrita vata*” and “*Aadhya vata*” for *Urustambha* synonymously. *Urustambha* according to Sushruta, resembles with the condition of an “acute spinal cord disorders.” “*Jwara*” (fever) explained in

Urustambha of Sushruta's version indicates inflammatory pathology of spinal cord.^[3]

Inflammatory Myelopathy/Acute Myelopathy

ATM, an inflammatory myelitis, is one of the causes of acute transverse myelopathy. The five groups of disorders that present as acute myelopathy are demyelination, infections, inflammatory disorders, vascular, and neoplastic/paraneoplastic. The first three are considered as inflammatory disorders. Idiopathic ATM is characterized by sensory, motor and autonomic dysfunction (involvement of spinothalamic

Table 4: Similarity between *Urustambha chikitsa* and management of atherosclerosis/PAD/MS/intermittent claudication

<i>Urustambha Chikitsa</i> ^[3,4]	Management of atherosclerosis/MS/PAD/intermittent claudication
Diet:	
Yava (<i>Hordeum vulgare</i>)	Inhibits platelet aggregation, anti-inflammatory, useful in cardiovascular diseases, protects against diabetes, obesity, atherosclerosis and stroke, improves insulin resistance, antioxidant ^[36]
Shyamaka (<i>Setaria italica</i>)	Antidiabetic ^[37]
Kodrava (<i>Paspalum scrobiculatum</i> Linn.)	Antidiabetic, having low glycemic index, high dietary fiber and controls cholesterol and high blood sugar levels ^[37]
Karavellaka (<i>Momordica charantia</i>)	Hypoglycemic, antidiabetic ^[38,39]
Nimba (<i>Azadirachta indica</i>)	Anti-inflammatory, hypotensive, antiarrhythmic, diuretic, useful in coronary artery disease ^[40]
Aragvadha (<i>Cassia fistula</i>)	Hypoglycemic, improves glucose tolerance ^[41]
Arishta's (medicated wines)	Antithrombotic effect, inhibits atherogenic action of high levels of LDL cholesterol ^[14]
Panchakarma procedures:	
Utsaadana	Improve collateral circulation?
Pralepa/Parisheka	To reduce pain/stiffness?
Snehana	
Swedana	
Internal medicines:	
Guggulu (<i>Commiphora mukul</i>)	Prevents platelet aggregation, antithrombotic, hypocholesterolemic, hypolipidemic, anti-atherosclerotic, antioxidant ^[40]
Shilajit	Reduces blood sugar levels, beneficial effects on lipid profile, antidiabetic ^[42] Analgesic, anti-inflammatory, antioxidant ^[43]
Hareetaki (<i>Terminalia chebula</i>)	Antiatherosclerotic, hypocholesterolemic, hypotensive, anti-oxidant, anti-inflammatory ^[40] Antidiabetic ^[44]
Cow's urine	Antioxidant, antiobesity, vasodilator, fibrinolytic, blood purifier, lowers cholesterol levels, maintains structural integrity of corpuscles ^[45]
Pippali (<i>Piper longum</i>)	Antiplatelet, antihyperlipidemic, antioxidant, anti-inflammatory, analgesic, cardio protective, coronary vasodilatation ^[46]
Bilva (<i>Aegle marmelos</i>)	Hypoglycemic, improves glucose tolerance ^[40]
Daru haridra (<i>Berberis aristata</i>)	Antiangina, antihypertensive, antiarrhythmic, anti-inflammatory, prevents myocardial infarction ^[40] Reduces serum cholesterol, triglycerides, LDL levels and increases thrombin and fibrinogen time ^[47]
Haridra (<i>Curcuma longa</i>)	Fibrinolytic, antiatherosclerotic, antithrombotic, antioxidant, hypolipidemic, antiobesity, anti-inflammatory ^[40] Curcumin acts as anticoagulant, antidiabetic, antifibrotic and hypotensive ^[48]

(Contd...)

Table 4: (Continued)

<i>Amlaki (Embllica officinalis)</i>	Hypolipidemic, cardio protective, adaptogenic, antioxidant, anti-inflammatory ^[40]
<i>Jatamansi (Nardostachys jatamansi)</i>	Anti-arrhythmic, hypotensive, tranquilizing ^[40]
<i>Shunthi (Zingiber officinale)</i>	Antioxidant, antiplaque, cardio protective, antiobesity, hypocholesterolemic, anti-inflammatory, useful in coronary artery disease ^[40]
<i>Vacha (Acorus calamus)</i>	Hypolipidemic, tranquilizer, useful in heart diseases ^[40] Antioxidant ^[49]
<i>Vibheetaki (Terminalia bellirica)</i>	Antioxidant, anti-inflammatory, hypotensive, smooth muscle relaxant ^[40]
<i>Bhallataka (Semecarpus anacardium Linn.)</i>	Anti-atherosclerotic, reduces serum hyper lipidemia, useful in coronary heart disease ^[50]
<i>Chitraka (Plumbago zeylanica)</i>	Prevents platelet adhesion, prolongs bleeding time by altering platelet adhesion and coagulation ^[51]
<i>Musta (Cyperus rotundus)</i>	Antiplatelet aggregating activity ^[52]
<i>Katukarohini (Picrorhiza kurroa)</i>	Antithrombic, anti-inflammatory ^[53]
<i>Devadaru (Cedrus deodara)</i>	Antihyperglycemic ^[54]
<i>Samangaa (Rubia cordifolia)</i>	Antithrombic, antioxidant, blood purifier, regulates blood pressure and vaso constriction ^[55]
<i>Shalmali (Salmalia malabarica)</i>	Hypoglycemic, antihyperglycemic, analgesic ^[56]
<i>Udeechya (Pavonia odorata)</i>	Antidiabetic, hypoglycemic ^[57]
<i>Chandana (Santalum album)</i>	Antioxidant, NO scavenging activity, reduces angina pain, anti-inflammatory ^[58] Anti-hyperglycemic, antihyperlipidemic, cardio protective ^[59]
<i>Dhataki (Woodfordia fruticosa)</i>	Anti hyperglycemic, antioxidant ^[60] Anti-inflammatory, anti-nociceptive ^[61]
<i>Talisa (Abies webbiana)</i>	Inhibits platelet aggregation, useful in thrombo-embolic conditions ^[62]
<i>Kakamachi (Solanum nigrum)</i>	Antinociceptive, anti-inflammatory, antipyretic ^[63] Antidiabetic ^[64] Antioxidant, hypolipidemic ^[65]
<i>Lodhra (Symplocos recemosa)</i>	Antioxidant, anti-inflammatory, antidiabetic ^[66]
<i>Padmaka (Prunus pudum)</i>	Analgesic, antioxidant, diuretic ^[67]
<i>Gunja (Abrus precatorius)</i>	Antidiabetic ^[68] Antithrombic ^[69] Antiplatelet, antiviral, antioxidant, anti-inflammatory ^[65]
<i>Madanaphala (Randia dumetorum)</i>	Analgesic, anti-inflammatory ^[70] Antidiabetic, antihyperlipidaemic, anti-hyperglycemic, antioxidant ^[71]
<i>Danti (Baliospermum montanum)</i>	Antidiabetic, decreases blood sugar levels and serum cholesterol levels ^[72]
<i>Kutaja (Holarrhena antidysenterica)</i>	Antidiabetic, decreases serum cholesterol, triglycerides and increases liver glycogen ^[73]
<i>Chavya (Piper retrofractum)</i>	Antihypertensive ^[59]
<i>Murva (Marsdenia tenacissima)</i>	Antiobesity ^[74]
<i>Patha (Cissampelos pareira)</i>	Antidiabetic ^[75]
<i>Karanja (Pongamia pinnata)</i>	Antihyperglycemic ^[76]
<i>Nyagrodha (Ficus bengalensis)</i>	Antidiabetic, raises serum insulin ^[77]
<i>Naga keshara (Mesua ferrea)</i>	Anti-inflammatory ^[78] Antioxidant ^[79]
<i>Kshara prayoga</i>	Antiobesity (Yava kshara) ^[80] Thrombolytic/anti-platelet aggregative factors/statin therapy
Exercise therapy:	
Swimming	Exercise increases HDL levels, decreases LDL levels and serum triglycerides, decreases blood pressure and cardiovascular risk factors ^[35]
Walking on gravel/sand	
Vigorous physical exercise	Enhances collateral circulation ^[28]
Walking against tides/water current	Relieves pain in claudication ^[29]

LDL: Low-density lipoprotein, HDL: High-density lipoprotein, NO: Nitric oxide, MS: Metabolic syndrome, PAD: Peripheral artery disease

Table 5: Similarity between *Sushruta's* version of *Urustambha* and spinal cord ischemia/inflammatory myelopathy/acute transverse myelitis

<i>Urustambha</i> ^[3]	Spinal cord ischemia/inflammatory myelopathy/acute transverse myelitis ^[81,82]
<i>Urustambha</i> explained in “ <i>Maha vata vyadhi</i> ” context	Indicates acuteness? Acute onset? Need of quick intervention?
<i>Angamarda</i> (body ache) <i>Jwara</i> (fever)	Inflammatory pathology of spinal cord like acute viral myelitis?
<ul style="list-style-type: none"> • <i>Ruja</i> (pain) • “<i>Na sva iva cha manyate</i>”/“<i>Sparsha agyanaat</i>” (loss of sensation) • <i>Staimitya</i> (rigidity/immobility/stillness) • <i>Stabdhatta</i> (spasm/spasticity/rigidity) • <i>Gurutva</i> (feeling heaviness of lower limbs) • “<i>A sthiraau – A kathinau</i>” (flaccid/lack of control/paresis) • <i>Sheetala</i> (coldness) • <i>Roma harsha</i> (goose flesh/pilo motor reflex) 	Sensory disturbance/paresthesia/loss of proprioception (involvement of spino thalamic tracts)
<ul style="list-style-type: none"> • <i>Staimitya</i> (rigidity/immobility/stillness) • <i>Stabdhatta</i> (spasm/spasticity/rigidity) • <i>Gurutva</i> (feeling heaviness of lower limbs) • “<i>A sthiraau – A kathinau</i>” (flaccid/lack of control/paresis) 	Motor disturbance/paresis/paralysis either symmetrical or asymmetrical (involvement of cortico spinal tracts)
<ul style="list-style-type: none"> • <i>Sheetala</i> (coldness) • <i>Roma harsha</i> (goose flesh/pilo motor reflex) 	Autonomic dysfunctions like temperature fluctuations/loss of temperature sensation (involvement of autonomic fibers)
All the above features together characteristic of <i>Urustambha</i> according to <i>Sushruta Acharya</i>	Various sensory, motor and autonomic disturbances along with paraplegia and fever indicate “acute transverse myelitis”/“inflammatory myelopathy”/“inflammatory spinal disease”/“infectious myelitis”

tracts, corticospinal tracts, and autonomic fibers, respectively) attributable to the spinal cord. Bilateral signs and/or symptoms (even though not necessarily symmetric) and also clearly defined sensory level are also the characteristic features of idiopathic ATM.^[81] ATM usually presents with paraplegic symptoms accompanied by or immediately following a febrile (viral) infection.^[82] The inflammatory myelopathy is similar to *Urustambha* of *Sushruta's* version [Table 5].

COMPLICATIONS OF URUSTAMBHA

The patient suffering with *Urustambha*, if further afflicted with severe burning sensation, severe pain and tremors, it leads to death. If such signs and symptoms are absent, such a patient is treatable.^[4] The severe pain (rest pain) with hyperesthesia may indicate “pre-gangrenous state” due to chronic arterial occlusion.^[28] Atherosclerosis is rarely fatal. It is thrombosis, superimposed on a ruptured or eroded atherosclerotic plaque, which precipitates life-threatening clinical events such as acute coronary syndrome and stroke.^[83] Clinical practice has demonstrated that the multisystemic involvement of vascular disease is common and various epidemiologic studies have shown that up to 50% of patients with PAD also have symptoms of cerebro-vascular or heart disease.^[84]

CONCLUSION

Urustambha is a lifestyle disease and it is commonly seen in higher socioeconomic status. *Urustambha samprapti* resembles with atherogenesis. *Diva swapna* and *raatri jaagarana* explained in *Urustambha nidaana* may indicate

OSA. Clinical presentation of *Urustambha* may be unilateral or bilateral or both. *Charaka's* version of *Urustambha* indicates vascular pathology like “aortoiliac occlusion” with an underlying “MS” whereas *Sushruta's* version of *Urustambha* indicates inflammatory pathology of spinal cord like “ATM” or “inflammatory myelopathy” or “infectious myelitis.” Principles of *Urustambha* are applicable for the prevention and management of the conditions such as Atherosclerosis, MS, OSA, aortoiliac occlusion, diabetes mellitus, obesity, various cardiovascular pathologies, acute myelopathy, paraplegias and other ischemic and inflammatory spinal diseases.

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REFERENCES

1. Valiathan MS. The Legacy of *Charaka*. Numb and Immobile Thighs (*Urustambha*). 1st ed. Ch. 61. Chennai: Orient Longman Private Limited; 2003. p. 478-81.
2. Agnivesha, elaborated by *Charaka*. *Charaka samhita* – Text with English translation. In: Sharma PV, editor. *Chikitsa Sthana, Urustambha Chikitsitam Adhyaya* 27/1-20. Vol. 4. Varanasi: Chaukhambha Orientalia; 2012. p. 208-9.
3. Sushruta. *Susruta samhita*, commentary by Dalhana. In: Vaidya Jadavji Trikamji Acharya, Narayana Ram

- Acharya, editors. *Chikitsa Sthana, Maha Vata Vyadhi Chikitsitam Adhyaya*, 5/31-45. Varanasi: Chaukhamba Orientalia; 2009. p. 429-30.
4. Agnivesha, elaborated by Charaka and Dridhabala commentary by Chakrapani. In: Vaidya Jadavji Trikamji Acharya, editor. *Charaka Samhita, Chikitsa Sthana, Urustambha Chikitsitam Adhyaya*, 27/1-62. Varanasi: Chaukhamba Surbharati Prakashan; 2008. p. 613-5.
 5. Vagbhata. In: Bhishagacharya Harishastri Paradkara Vaidya, editor. *Ashtanga Hridaya*, Commentary by Arunadatta and Hemadri, Nidana Sthana, Vata Vyadhi Nidanam Adhyaya, 15/47-51. 9th ed. Varanasi: Chowkhamba Sanskrit Series Office; 2005. p. 534.
 6. Bhela. *Bhela samhita*: Text with English translation, commentary and critical notes by Krishnamurthy KH. In: Priyavrat Sharma, editor. *Chikitsa Sthana, Urustambha Chikitsa*. Reprint Edition. Ch. 14. Varanasi: Chaukhamba Vishvabharati; 2008. p. 384-7.
 7. Bhela. *Bhela samhita*. Edited and Commentary by Sri Abhay Katayan. *Chikitsa sthana, Urustambha Chikitsa*. 1st ed. Ch. 4. Varanasi: Chaukhamba Surbharati Prakashan; 2009; p. 389.
 8. Sharma AK, Yadav PS. *Urustambha. Kayachikitsa*. 1st ed., Vol. III. Ch. 49. New Delhi: Chaukhamba Orientalia; 2014. p. 134-5.
 9. Jindal N, Shamkuwar MK, Berry S. Importance of Rookshana Karma (dehydrating therapy) in the management of transverse myelitis. *Ayu* 2012;33:402-5.
 10. Mamidi P, Gupta K. *Ayurvedic* management of chronic rheumatoid arthritis with bilateral hip involvement: A case report. *J Pharm Sci Innov* 2015;4:329-32.
 11. The American Association of Physicians of Indian Origin (AAPI). In: Mishra R, editor. *Indian Foods: AAPI's Guide to Nutrition, Health and Diabetes*. 2nd ed. Ch. 1. Chennai: M/S Allied Publishers Pvt., Ltd.; 2011. p. 3-4.
 12. The American Association of Physicians of Indian origin (AAPI). *Indian foods: AAPI's guide to nutrition, health and diabetes*. In: Mishra R, editor. *Preventing Heart Disease in Asian Indians*. 2nd ed. Ch. 3. Chennai: M/s Allied Publishers Pvt., Ltd.; 2011. p. 17.
 13. Ghiadoni L, Donald AE, Cropley M, Mullen MJ, Oakley G, Taylor M, *et al*. Mental stress induces transient endothelial dysfunction in humans. *Circulation* 2000;102:2473-8.
 14. Kiechl S, Willeit J, Rungger G, Egger G, Oberhollenzer F, Bonora E. Alcohol consumption and atherosclerosis: What is the relation? Prospective results from the Bruneck Study. *Stroke* 1998;29:900-7.
 15. Spieker L, Noll G. Pathophysiologic cardiovascular changes in stress and depression. *Ther Umsch* 2003;60:667-72.
 16. Kamarck TW, Everson SA, Kaplan GA, Manuck SB, Jennings JR, Salonen R, *et al*. Exaggerated blood pressure responses during mental stress are associated with enhanced carotid atherosclerosis in middle-aged Finnish men: Findings from the Kuopio Ischemic heart disease study. *Circulation* 1997;96:3842-8.
 17. Brotman DJ, Golden SH, Wittstein IS. The cardiovascular toll of stress. *Lancet* 2007;370:1089-100.
 18. Bloomfield GL, Sugerman HJ, Blocher CR, Gehr TW, Sica DA. Chronically increased intra-abdominal pressure produces systemic hypertension in dogs. *Int J Obes Relat Metab Disord* 2000;24:819-24.
 19. Flageole H, Ouahed J, Walton JM, Yousef Y. Abdominal compartment syndrome secondary to chronic constipation. *Case Rep Pediatr* 2011;2011:562730.
 20. Wolk R, Somers VK. Sleep and the metabolic syndrome. *Exp Physiol* 2007;92:67-78.
 21. Drager LF, Bortolotto LA, Lorenzi MC, Figueiredo AC, Krieger EM, Lorenzi-Filho G. Early signs of atherosclerosis in obstructive sleep apnea. *Am J Respir Crit Care Med* 2005;172:613-8.
 22. Drager LF, Polotsky VY, Lorenzi-Filho G. Obstructive sleep apnea: An emerging risk factor for atherosclerosis. *Chest* 2011;140:534-42.
 23. Singh RB, Beegom R, Verma SP, Haque M, Singh R, Mehta AS, *et al*. Association of dietary factors and other coronary risk factors with social class in women in five Indian cities. *Asia Pac J Clin Nutr* 2000;9:298-302.
 24. Wasir JS, Misra A. The metabolic syndrome in Asian Indians: Impact of nutritional and socio-economic transition in India. *Metab Syndr Relat Disord* 2004;2:14-23.
 25. Prasad DS, Kabir Z, Dash AK, Das BC. Prevalence and risk factors for metabolic syndrome in Asian Indians: A community study from urban Eastern India. *J Cardiovasc Dis Res* 2012;3:204-11.
 26. Tendera M, Aboyans V, Bartelink ML, Baumgartner I, Clément D, Collet JP, *et al*. ESC Guidelines on the diagnosis and treatment of peripheral artery diseases. *European heart journal* 2011; 32(22):2851-2906.
 27. Al-Shafie T, Suman P. Aortoiliac occlusive disease-vascular surgery. In: Yamanouchi D, editor. *InTech*; 2012. p. 3-38. Available from: <http://www.intechopen.com/books/vascular-surgery/aortoiliac-occlusive-disease>. [Last accessed on 2017 Mar 04].
 28. A Concise Textbook of Surgery. Diseases of Arteries. 3rd ed. Ch. 13. Calcutta: Dr S Das 2001. p. 148-57.
 29. Leng GC, Fowler B, Ernst E. Exercise for intermittent claudication. DOI: 10.1002/14651858.CD000990.
 30. Boras J, Brkljacic N, Ljubic A, Ljubic S. Peripheral artery disease. *Diabetol Croat* 2010;39:67-77.
 31. Kilany A, Al-Hashel JY, Rady A. Acute aortic occlusion presenting as flaccid paraplegia. *Case Rep Neurol Med* 2015;2015:713489.
 32. Largiadèr J, Schneider E. Therapy of acute peripheral arterial occlusion. *Herz* 1991;16:456-62.
 33. Subhash Ranade. *Kayachikitsa. Majjavaha – Mamsavaha srotas: Khanja and Pangu*. 1st ed., Vol. III. Ch. 1. Section K. New Delhi: Chaukhamba Sanskrit Pratishthan; 2001. p. 403.
 34. *Madhavakara. Urustambha nidaanam*. In: Brahmananda Tripathi, editor. *Madhava nidaanam* with Sanskrit

- commentary ‘*Madhukosha*’ by Vijayarakhshita & Shrikanthadatta. 1st ed., Vol. I. Ch. 24. Varanasi: Chaukhamba Surbharati Prakashan; 2012. p. 465-70.
35. Thompson PD. Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease. *Arterioscler Thromb Vasc Biol* 2003;23:1319-21.
 36. Gul S, Ahmed S, Kifli N, Uddin QT, Batool Tahir N, Abrar Hussain, *et al.* Multiple pathways are responsible for anti-inflammatory and cardiovascular activities of *Hordeum vulgare* L. *J Transl Med* 2014;12:316.
 37. Pandya MG. A review on role of vyayama (physical exercise) in the prevention and management of *Madhumeha* (diabetes mellitus Type 2). *Int J Ayu Pharm Chem* 2015;3:1-13.
 38. Mehrotra R, Bajaj S, Kumar D. Use of complementary and alternative medicine by patients with diabetes mellitus. *Natl Med J India* 2004;17:243-5.
 39. Rai MK. A review on some antidiabetic plants of India. *Anc Sci Life* 1995;14:168-80.
 40. Mehrotra NN, Ojha SK, Tandon S. Drug development for cardiovascular diseases from *Ayurvedic* plants. *Curr R D Highl* 2007;30:1-9.
 41. Espósito Avella M, Díaz A, de Gracia I, de Tello R, Gupta MP. Evaluation of traditional medicine: Effects of *Cajanus cajan* L. and of *Cassia fistula* L. on carbohydrate metabolism in mice. *Rev Med Panama* 1991;16:39-45.
 42. Trivedi NA, Mazumdar B, Bhatt JD, Hemavathi KG. Effect of *Shilajit* on blood glucose and lipid profile in alloxan-induced diabetic rats. *Indian J Pharmacol* 2004;36:373-6.
 43. Mittal P, Kaushik D, Gupta V, Bansal P, Khokra S. Therapeutic potentials of ‘*Shilajit rasayana*’ - A review. *Int J Pharm Clin Res* 2009;1:47-9.
 44. Rao NK, Nammi S. Antidiabetic and renoprotective effects of the chloroform extract of *Terminalia chebula* Retz. seeds in streptozotocin-induced diabetic rats. *BMC Complement Altern Med* 2006;6:17.
 45. Mohanty I, Senapati MR, Jena D, Palai S. Diversified uses of cow urine. *Int J Pharm Pharm Sci* 2014;6:20-2.
 46. Kumar S, Kamboj J, Suman, Sharma S. Overview for various aspects of the health benefits of *Piper longum* Linn. fruit. *J Acupunct Meridian Stud* 2011;4:134-40.
 47. Razzak FA, Alamkhan R, Feroz Z, Afroz S. Effect of *Berberis aristata* on lipid profile and coagulation parameters. *Afr J Pharm Pharmacol* 2011;5:943-7.
 48. Chattopadhyay I, Biswas K, Bandyopadhyay U, Banerjee RK. Turmeric and curcumin: Biological actions and medicinal applications. *Curr Sci* 2004;87:44.
 49. Manikandan S, Srikumar R, Jeya Parthasarathy N, Sheela Devi R. Protective effect of *Acorus calamus* Linn. on free radical scavengers and lipid peroxidation in discrete regions of brain against noise stress exposed rat. *Biol Pharm Bull* 2005;28:2327-30.
 50. Premalatha B. *Semecarpus anacardium* Linn. Nuts – A boon in alternative medicine. *Indian J Exp Biol* 2000;38:1177-82.
 51. Vijayakumar R, Senthilvelan M, Ravindran R, Devi RS. *Plumbago zeylanica* action on blood coagulation profile with and without blood volume reduction. *Vascul Pharmacol* 2006;45:86-90.
 52. Seo EJ, Lee DU, Kwak JH, Lee SM, Kim YS, Jung YS. Antiplatelet effects of *Cyperus rotundus* and its component (+)-nootkatone. *J Ethnopharmacol* 2011;135:48-54.
 53. Engels F, Renirie BF, Hart BA, Labadie RP, Nijkamp FP. Effects of apocynin, a drug isolated from the roots of *Picrorhiza kurroa*, on arachidonic acid metabolism. *FEBS Lett* 1992;305:254-6.
 54. Ahmad R, Srivastava SP, Maurya R, Rajendran SM, Arya KR, Srivastava AK. Mild anti hyperglycaemic activity in *Eclipta alba*, *Berberis aristata*, *Betula utilis*, *Cedrus deodara*, *Myristica fragrans* and *Terminalia chebula*. *Indian J Sci Technol* 2008;1:1-6.
 55. Samy RP, Pushparaj PN, Gopalakrishnakone P. A compilation of bioactive compounds from Ayurveda. *Bioinformation* 2008;3:100-10.
 56. Jain V, Verma SK. Assessment of credibility of some folk medicinal claims on *Bombax ceiba* L. *Indian J Tradit Knowl* 2014;13:87-94.
 57. Rayar A, Manivannan R. Evaluation of anti diabetic activity from the root extracts of *Pavonia odorata* wild in alloxan induced diabetic rats. *Int J Pharm Sci Invent* 2015;4:46-52.
 58. Sindhu RK, Upma KA, Arora S. *Santalum album* Linn: A review on morphology, phytochemistry and pharmacological aspects. *Int J PharmTech Res* 2010;2:914-9.
 59. Lenin, Rao MRK, Prabhu K, Bindu R, Elizabeth AA, Dinakar S. The study of anti oxidant activities of an *Ayurvedic* medicine Ayaskriti. *Der Pharm Lett* 2016;8:203-11.
 60. Neeraj V, Amresh G, Sahu PK, Rao CV, Pratap SA. Anti hyperglycaemic activity of *Woodfordia fruticosa* (Kurz.) flower extracts in glucose metabolism and lipid peroxidation in streptozotocin-induced diabetic rats. *Indian J Exp Biol* 2012;50:351-8.
 61. Baravalia Y, Vaghasiya Y, Chanda S. Brine shrimp cytotoxicity, anti-inflammatory and analgesic properties of *Woodfordia fruticosa* Kurz. Flowers. *Iran J Pharm Res* 2012;11:851-61.
 62. Yasin M, Hussain Janbaz K, Imran I, Gilani AU, Bashir S. Pharmacological studies on the antispasmodic, bronchodilator and anti-platelet activities of *Abies webbiana*. *Phytother Res* 2014;28:1182-7.
 63. Zakaria ZA, Gopalan HK, Zainal H, Mohd Pojan NH, Morsid NA, Aris A, *et al.* Antinociceptive, anti-inflammatory and antipyretic effects of *Solanum nigrum* chloroform extract in animal models. *Yakugaku Zasshi* 2006;126:1171-8.
 64. Meonah ST, Palaniswamy M, Immanuel ST, Rajkumar LA, Nandhini RU. Pharmacognostical and hypoglycaemic activity of *Solanum nigrum* Linn. *Plant. Int J Pharm Pharm Sci* 2012;4:221-4.

65. Ganesan K, Nair SK, Sinaga M, Gani SB. A review on the phytoconstituents and pharmacological actions in the medicinal plants of Bedauna forest, Jimma zone, South west Ethiopia reported effect on experimental models. *Eur J Biomed Pharm Sci* 2016;3:62-83.
66. Acharya N, Acharya S, Shah U, Shah R, Hingorani L. A comprehensive analysis on *Symplocos racemosa* Roxb.: Traditional uses, botany, phytochemistry and pharmacological activities. *J Ethnopharmacol* 2016;181:236-51.
67. Pallavi G, Gupta KL, Rishi R. Ethno pharmaco-botanical review of *Padmaka* – *Prunus puddum* Roxb. *Int J Ayu Herb Med* 2011;1:87-98.
68. Gurjar HP, Irchhaiya R, Verma A. Review on some medicinal plants with anti diabetic activity. *J Drug Deliv Ther* 2016;6:45-51.
69. Bhatia M, Siddiqui N, Gupta S. *Abrus precatorius* (L.): An evaluation of traditional herb. *J Pharm Res* 2013;3:3296-15.
70. Ritesh GP, Pathak NL, Rathod JD, Patel LD, Bhatt NM. Phytopharmacological properties of *Randia dumetorum* as a potential medicinal tree: An overview. *J Appl Pharm Sci* 2011;1:24-6.
71. Mishra PR, Panda PK, Chowdary KA, Panigrahi S. Anti diabetic and anti hyperlipidemic activity of *Randia dumetorum*. *Int J Res Pharm Chem* 2012;2:552-9.
72. Mohanraghupathy S, Silambujanaki P, Chitra V, Raju D. Effect of Hydro alcoholic extract of *Baliospermum montanum* roots against diabetic nephropathy on rats. *Int J Res Pharmacol Pharmacother* 2013;2:263-6.
73. Mana S, Singhal S, Sharma NK, Singh D. Hypoglycemic effect of *Holarrhena antidysenterica* seeds on streptozotocin induced diabetic rats. *Int J PharmTech Res* 2010; 2:1325-1329.
74. Chandrasekaran CV, Vijayalakshmi MA, Prakash K, Bansal VS, Meenakshi J, Amit A. Review article: Herbal approach for obesity management. *Am J Plant Sci* 2012;3:1003-14.
75. Piero NM, Eliud NN, Susan KN, George OO, Murugi NJ, David M, *et al.* *In vivo* anti diabetic activity and safety in rats of *Cissampelos pareira* traditionally used in the management of diabetes mellitus in Embu county, Kenya. *J Drug Metab Toxicol* 2015;6:184.
76. Punitha R, Manoharan S. Antihyperglycemic and antilipidperoxidative effects of *Pongamia pinnata* (Linn.) Pierre flowers in alloxan induced diabetic rats. *J Ethnopharmacol* 2006;105:39-46.
77. Bhushan MS, Rao CV, Ojha SK, Vijayakumar M, Verma A. An analytical review of plants for antidiabetic activity with their phyto constituents and mechanism of action. *Int J Pharm Sci Res* 2010;1:29-46.
78. Tiwari P, Nandy S. Screening of anti-inflammatory activity of *Mesua ferrea* Linn. flower. *Int J Biomed Res* 2010;3:245-52.
79. Udayabhanu J, Shanmugapriya K, Thangavelu T. Evaluation of phytochemical and anti oxidant contents of *Mesua ferrea*, *Hemionitis arifolia* and *Pimento dioica*. *Int J Adv Pharm Biol Chem* 2014;3:272-6.
80. Kodlady N, Patgiri BJ, Math PD. Role of *Kshara* (Alkalis) in the *Ayurvedic* therapeutics as per *Ashtanga Hrudayam*. *Indian J Anc Med Yoga* 2012;5:209-18.
81. Jacob A, Weinshenker BG. An approach to the diagnosis of acute transverse myelitis. *Semin Neurol* 2008;28:105-20.
82. Schwendreis P, Pennekamp W, Tegenthoff M. Differential diagnosis of acute and sub acute non-traumatic paraplegia. *Dtsch Arztebl Int* 2006;103:A2948-54.
83. Falk E. Pathogenesis of atherosclerosis. *J Am Coll Cardiol* 2006;47:C7-12.
84. Serrano Hernando FJ, Martin Conejero A. Peripheral artery disease: Pathophysiology, diagnosis and treatment. *Rev Esp Cardiol* 2007;60:969-82.

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