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Clinical Evaluation of Patoladi Kwatha Gandusha in the Management of Sheetada (Gingivitis): An Ayurvedic Perspective

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ABSTRACT

Background: Sheetada, described in classical Ayurvedic texts as a disease of the gingival region, closely resembles gingivitis in contemporary dentistry. It is characterized by gingival bleeding, discoloration, swelling, halitosis, and softness of gums, primarily attributed to vitiation of Kapha and Rakta dosha. Modern etiopathogenesis correlates gingivitis with plaque-induced inflammatory changes confined to gingival tissues.

Aim: To evaluate the therapeutic efficacy of Patoladi Kwatha administered in the form of Gandusha, along with Pratisarana and Raktamokshana, in the management of Sheetada with special reference to gingivitis.

Material & Methods: A case report of patient of 38 years old women who approached Shalakya Tantra OPD of Mansarovar Ayurvedic Medical College Hospital and Research Centre Bhopal with presenting with classical features of Sheetada underwent Raktamokshana on day one, followed by daily Pratisarana and Patoladi Kwatha Gandusha for seven days. Clinical parameters including gingival bleeding, halitosis, discoloration, moisture, and gingival softness were assessed using a scoring system before treatment and on the 8th day.

Results: Complete remission of clinical symptoms was observed within seven days. Gingival bleeding, halitosis, discoloration, and other inflammatory signs were reduced to nil by the follow-up visit. The TV formulation demonstrated anti-inflammatory, antimicrobial, wound-healing, and dosha-pacifying effects, contributing to restoration of gingival health.

Conclusion: Patoladi Kwatha Gandusha, combined with Pratisarana and Raktamokshana, proved to be an effective and safe therapeutic approach for the management of Sheetada (gingivitis). The intervention supports both classical Ayurvedic principles and contemporary understanding of inflammatory periodontal pathology.

Keywords: Sheetada; Gingivitis; Patoladi Kwatha; Gandusha; Pratisarana; Raktamokshana; Kapha-Pitta shamana; Periodontal inflammation; Ayurvedic dentistry; Herbal therapeutics.

INTRODUCTION

In the field of Ayurvedic clinical science, Sushruta Samhita is regarded as an authoritative treatise. Acharya Sushruta has described Sheetada as one among the fifteen diseases affecting the gingival region (Danthamoolagata roga). [1] Sheetada represents the early stage of periodontal pathology and, if left untreated, may progress to more advanced conditions such as Dantaveshta and Upakusha.

Unhealthy dietary patterns, including frequent consumption of fast foods, and deleterious habits such as smoking and betel chewing, along with improper oral hygiene practices, contribute to the vitiation of Kapha and Rakta dosha. [2] This pathological state manifests clinically as spontaneous gingival bleeding (akasmika raktasrava), discoloration of gums (krishnata), excessive moisture (prakledana), gingival softness (mriduta), swelling (shotha), and halitosis (mukha daurgandhya). In advanced stages, suppuration (paka), gingival recession (danta mamsa shiryamanata), and tooth mobility (chalata) may develop.

From the contemporary dental perspective, marginal gingivitis is primarily associated with the accumulation of plaque and calculus at the gingival margin due to inadequate oral hygiene. If neglected, this condition may progress to periodontitis, characterized by gingival firmness, altered contour, tooth mobility, spacing, and drifting. Local etiological factors include microbial plaque, calculus, food impaction, faulty restorations, mouth breathing, and malpositioned teeth. Systemic factors such as nutritional deficiencies (vitamins, minerals, proteins), drug hypersensitivity (e.g., phenol, silver nitrate, aspirin), endocrine disturbances, puberty, pregnancy, and menstruation also contribute to disease progression. Conventional management includes scaling, root planing, irrigation, and gingivoplasty. Early prevention and control are therefore essential for a favorable prognosis.

In Ashtanga Hridaya (Uttara Tantra, Chapter 2), Acharya Vagbhata has outlined several therapeutic modalities for the management of Sheetada. These include Pratisarana (therapeutic rubbing), Visravana (bloodletting), Pralepa (topical application of medicated paste), Kavala (oral rinsing), and Gandusha (retention of medicated liquid in the oral cavity). [3]

In the present context, Prachhana karma (therapeutic scraping) was initially performed to evacuate vitiated blood from the gingival tissues. Subsequently, Patoladi Kashaya Gandusha was selected for intervention. Gandusha was administered using a lukewarm decoction prepared from Patola, Shunthi, Triphala, Vishala, Tikta drugs, Haridra, Daruharidra, and Amrita, with the addition of 5 ml honey. The patient was instructed to retain the decoction in the oral cavity for 3–5 minutes. Patoladi formulation is described as Kapha-Pitta shamaka, possessing anti-inflammatory (shothahara), antimicrobial (krimighna), purificatory (shodhana), wound-healing (ropana), and rejuvenative (rasayana) properties, making it suitable for the management of Sheetada.

Pratisarana was performed using medicated powder applied locally over the gums. As tooth brushing is contraindicated in certain inflammatory oral conditions according to Acharya Nimi, Pratisarana was adopted as an alternative therapeutic measure. These procedures aid in removal of food debris and plaque, enhance local blood circulation, strengthen gingival defense mechanisms, and stimulate gingival fibers, thereby promoting healing. The combined therapeutic approach aims at interrupting the pathogenesis of the disease, restoring gingival integrity, and maintaining periodontal health.

Since Rakta dosha plays a predominant role in the etiopathogenesis of Sheetada, these interventions primarily facilitate dosha shamana and local tissue restoration. Additionally, the patient was advised to take Avipattikara churna (2 g at bedtime) for its anulomana (mild laxative and digestive regulatory) property. [4]

Considering the growing global interest in plant-based therapeutics as alternatives to modern pharmacological agents, the present study was designed to evaluate a scientifically validated Gandusha formulation for the effective management of Sheetada.

ETIOPATHOGENESIS OF GINGIVITIS

Gingivitis is an inflammatory condition of the gingival tissues, most commonly caused by bacterial infection. In contrast to periodontitis, gingivitis is not associated with clinical attachment loss or apical migration of the junctional epithelium. The pathological changes remain confined to the gingival epithelium and underlying connective tissue. [5]

Among periodontal diseases, gingivitis is the most prevalent form. It may be classified based on clinical presentation, duration, severity, and etiology. However, chronic plaque-induced gingivitis represents the most common variant. Clinically, gingival tissues appear erythematous, oedematous, tender, and exhibit a smooth, glossy surface. Bleeding on gentle probing is a characteristic feature. Spontaneous bleeding is uncommon, and pain is typically absent; therefore, many individuals remain unaware of the condition and do not seek timely treatment. [6]

Etiology

The primary etiological factor in gingivitis is the accumulation of microbial dental plaque in or adjacent to the gingival sulcus. Several bacterial species have been implicated in its pathogenesis, including Streptococcus, Fusobacterium, Actinomyces, Veillonella, and Treponema. Other organisms such as Bacteroides, Capnocytophaga, and Eikenella may also contribute to disease development. Local and systemic factors may enhance plaque accumulation or increase tissue susceptibility to microbial invasion. [7]

Based on etiology, gingivitis can be categorized into various types, the most common being plaque-induced gingivitis.

Plaque-Induced Gingivitis

Plaque-induced gingivitis is the most frequent form of gingival inflammation. Dental plaque is a thin biofilm that forms on the tooth surface due to inadequate oral hygiene. If not removed regularly, plaque may mineralize to form calculus, which further facilitates microbial colonization. The presence of a high bacterial load triggers an inflammatory response in the gingival tissues.

Certain local factors predispose individuals to plaque accumulation. Crowding and malalignment of teeth hinder effective plaque removal, thereby increasing the risk of inflammation. Orthodontic appliances and improperly fitted or inadequately finished dental prostheses may act as plaque-retentive sites.

In children, tooth eruption is often associated with transient gingival inflammation, termed eruption gingivitis. During the transitional phase of exfoliation of primary teeth and eruption of permanent teeth, maintaining oral hygiene becomes challenging, leading to localized plaque accumulation and gingival inflammation.

Thus, gingivitis represents a reversible inflammatory condition primarily initiated by microbial plaque, whose progression can be prevented through effective oral hygiene and timely professional intervention.

Dietary Gingivitis

Dietary gingivitis may occur due to nutritional deficiencies, particularly vitamin C deficiency (ascorbic acid). Inadequate intake of vitamin C compromises collagen synthesis and capillary integrity, thereby increasing susceptibility to gingival inflammation and bleeding.

Modern dietary patterns characterized by high consumption of refined carbohydrates and an increased omega-6 to omega-3 fatty acid ratio have been shown to promote inflammatory processes. [8] Diets with a high glycaemic index contribute to systemic and local inflammation by activating nuclear factor kappa B (NF-kB) and inducing oxidative stress. [9,10] These molecular pathways enhance the production of pro-inflammatory cytokines, thereby exacerbating gingival tissue response to plaque biofilm.

Hormonal Gingivitis

Hormonal fluctuations significantly influence gingival tissue response to plaque accumulation. During pregnancy, elevated levels of ooestrogen and progesterone, along with increased vascular permeability and dilation, intensify the inflammatory reaction of gingival tissues even in the presence of minimal plaque deposits. The severity of gingival inflammation has been correlated with circulating oestrogen levels, which modulate the host response to microbial biofilm at the gingival margin. [11,12]

Similarly, hormonal changes during puberty alter gingival tissue reactivity, resulting in puberty-associated gingivitis. Gingival tissues possess specific receptors for oestrogen and testosterone. Oestrogen receptors are predominantly located in the basal and spinous layers of the gingival epithelium, while both oestrogen and androgen receptors are present in gingival fibroblasts and endothelial cells of small blood vessels. This receptor distribution establishes gingiva as a target organ for steroid hormones, predisposing adolescents to exaggerated inflammatory responses. Epidemiological observations indicate that gingivitis tends to manifest earlier in females (11–13 years) than in males (13–14 years). [13]

Drug-Induced Gingivitis

Several systemic medications are known to induce gingival enlargement and inflammation as adverse effects. These include phenytoin (used in epilepsy), calcium channel blockers (prescribed for hypertension and angina), anticoagulants, fibrinolytic agents, oral contraceptives, protease inhibitors, and vitamin A derivatives.

The underlying mechanism is believed to involve drug metabolites stimulating fibroblast proliferation. An imbalance between synthesis and degradation of extracellular matrix components results in excessive accumulation of immature proteins, particularly collagen, within the gingival connective tissue. This pathological alteration manifests clinically as gingival enlargement and secondary inflammation. [14]

Risk and Contributing Factors

In addition to the above etiological categories, several risk and modifying factors contribute to the development and progression of gingivitis. These include tobacco smoking and chewing, systemic illnesses, genetic predisposition (such as hereditary gingival fibromatosis), xerostomia, and dental crowding. Such factors may either increase plaque retention or alter host immune response.

Epidemiology

Gingivitis is the most prevalent periodontal disease across populations. Epidemiological data indicate higher prevalence among males compared to females, possibly due to comparatively lower adherence to oral hygiene practices among males. The condition is commonly observed in both children and adults.

Socioeconomic status significantly influences disease prevalence. Individuals from lower socioeconomic backgrounds exhibit higher rates of gingivitis, likely due to limited access to oral healthcare services and reduced awareness regarding oral hygiene maintenance.

Pregnancy is associated with increased prevalence and severity of gingivitis, attributed to hormonal influences and altered immune responses. [15]

Among the various clinical types, plaque-induced gingivitis remains the most frequently encountered form. Other types include hormonal gingivitis, acute ulcerative necrotizing gingivitis, drug-induced

gingivitis, and hyperplastic gingivitis. Nevertheless, plaque-induced gingivitis accounts for the vast majority of cases, exceeding the combined frequency of all other variants. [16]

Pathophysiology

The progression of periodontal disease was systematically described by Page and Schroeder in 1976. [17] Based on histopathological changes, gingivitis is categorized into three stages—initial, early, and established lesions—while the advanced lesion corresponds to periodontitis.

Initial Lesion

The initial lesion is characterized by an acute exudative inflammatory response. It is marked by increased gingival crevicular fluid flow and migration of neutrophils from the vascular plexus within the gingival connective tissue into the gingival sulcus. Structural alterations occur in the perivascular connective tissue matrix, leading to localized fibrin deposition.

This stage becomes evident within 2–4 days of plaque accumulation. Collagen destruction begins due to the release of collagenase and other proteolytic enzymes from neutrophils. Approximately 5–10% of the gingival connective tissue is infiltrated by inflammatory cells during this phase. [16]

Early Lesion

The early lesion corresponds to a delayed-type hypersensitivity reaction and typically develops approximately one week after plaque accumulation. Clinical signs of gingivitis, including erythema and bleeding on probing, become apparent at this stage.

Lymphocytes predominate among the inflammatory infiltrate, accounting for nearly 75% of the cellular population, along with macrophages. A small number of plasma cells may also be observed. Inflammatory cell infiltration involves approximately 5–15% of the gingival connective tissue, accompanied by 60–70% collagen loss in the affected area.

Fibroblasts exhibit degenerative changes, and there is a continued increase in gingival crevicular fluid flow and leukocyte migration. Neutrophils and mononuclear cells accumulate within the junctional epithelium. The duration of the early lesion is variable and may persist for extended periods. [16]

Established Lesion

The established lesion represents a chronic inflammatory stage. It is characterized by enhanced collagenolytic activity and a marked increase in plasma cells and B lymphocytes, which become the predominant cell types. Macrophages and T lymphocytes are also present.

A gingival pocket lined by pocket epithelium may develop at this stage. The lesion demonstrates a high degree of organization and chronicity. Increased proportions of B cells and plasma cells, along with a relative decrease in T cells, are associated with greater severity of gingival inflammation.

The established lesion may remain stable for months or years or may progress to a destructive lesion, often associated with qualitative and quantitative changes in the microbial flora. Importantly, this stage is potentially reversible with effective periodontal therapy. Successful treatment leads to a shift toward a health-associated microbial profile and a corresponding reduction in plasma cells and lymphocytes within the gingival tissues. [16]

Advanced Lesion

The advanced lesion represents the transition from gingivitis to periodontitis. This stage is characterized by irreversible attachment loss. The inflammatory process extends beyond the gingiva to involve deeper supporting structures, including the periodontal ligament and alveolar bone.

Progressive connective tissue destruction and alveolar bone resorption occur, potentially resulting in tooth mobility and eventual tooth loss if untreated. [18,19]

HISTORY AND PHYSICAL EXAMINATION

CASE HISTORY

Patient Profile

Name : XYZ

Age / Sex: 32-year-old Female

Religion : Hindu

Address: Kolar Road Bhopal

Occupation: Office worker

Past history: No systemic illness

General Examination

- Body built - Average
- BP - 120/80 mm/Hg.
- Pulse - 74/bpm
- Temperature- Afebrile

1. Drug Profile and Pharmacological Actions

Intervention Drug: Patoladi Kashaya (decoction form)

The formulation consists of the following ingredients with their classical therapeutic properties:

Drug	**Property**
Patola	Krimighna (antimicrobial), alleviates all three dosha.
Shunti	Pacifies Kapha and Vata; improves digestion and reduces inflammation.
Guduchi	Tridosha balancing, reduces burning sensation, acts as a rejuvenative (rasayana).
Tikta group drugs	Pacify Kapha and Pitta; exhibit anti-inflammatory action.
Vishala	Pacifies Pitta and Kapha.
Haritaki	Tridosha balancing, rejuvenative, nutritive (bruhana).
Vibhitaki	Pacifies Kapha and Pitta; supports tissue health.
Amalaki	Rejuvenative, pacifies Rakta and Pitta, relieves itching and inflammation.
Trayanthi	Pacifies Kapha and Vata; purifies Pitta.
Haridra	Tridosha pacifying, mild purgative for Pitta, anti-inflammatory and antimicrobial.
Daruharidra	Tridosha pacifying; known for anti-inflammatory and antimicrobial properties.

Collectively, the formulation exhibits Kapha-Pitta shamaka, anti-inflammatory, antimicrobial, wound-healing, and rejuvenative actions, making it suitable for gingival inflammatory conditions.

Physical Examination Findings

Healthy gingival tissue appears pink (or physiologically pigmented in individuals with darker complexion), firm in consistency, and exhibits a stippled surface texture. There are no signs of erythema, edema, or spontaneous bleeding. On gentle periodontal probing, no bleeding is observed.

Periodontal examination reveals probing depths of less than 3 mm, indicating healthy sulcus depth. Radiographic evaluation shows no evidence of alveolar bone loss.

Treatment Plan

Setting: Outpatient department (OPD) basis

Drug for Intervention: Patoladi Kashaya

Dosage Form: Kwatha (decoction)

Dosage Schedule: Twice daily

Duration of Treatment: 7 days

Follow-up Assessment: On the 8th day

The treatment protocol was designed to reduce gingival inflammation, eliminate microbial load, promote tissue healing, and restore gingival health.

Treatment Protocol

The therapeutic procedures administered during the 7-day treatment period are summarized below:

Procedure	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Raktamokshan	Done	-	-	-	-	-	-
Pratisarana	Done	Done	Done	Done	Done	Done	Done
Gandusha	Done	Done	Done	Done	Done	Done	Done

Raktamokshana (bloodletting) was performed only on the first day. Pratisarana (local therapeutic rubbing) and Gandusha (medicated mouth retention) were carried out daily for seven consecutive days.

Assessment Criteria

Clinical signs and symptoms were evaluated using a scoring system before treatment and on the 8th day (post-treatment follow-up).

Symptoms	Before Treatment	After Treatment (Day 8)
Rakta srava (bleeding)	+++	Nil
Daurgandhya (halitosis)	++	Nil
Krishnata (discoloration)	++	Nil
Kledta (moistness/exudation)	+	Nil
Mruduta (softness of gums)	++	Nil

Observation

The patient exhibited complete remission of clinical features of Sheetada within 7 days of treatment. Significant reduction in gingival bleeding, halitosis, discoloration, moisture, and gingival softness was observed, with all symptoms resolving by the 8th day.

The patient was advised to maintain proper oral hygiene practices, including regular and appropriate tooth brushing. Additionally, cessation of tobacco chewing was strongly recommended due to its established deleterious effects on gingival and periodontal health.

Conclusion

The present study was undertaken to evaluate the therapeutic efficacy of Patoladi Kwatha administered in the form of Gandusha in the management of Sheetada, with special reference to gingivitis. The clinical findings indicate that the intervention was highly effective in alleviating the signs and symptoms of the disease.

Gandusha, as a therapeutic procedure, plays a significant role in maintaining oral health. It stimulates the salivary glands, helps maintain optimal oral pH, activates taste buds and sensory nerves, enhances local circulation and temperature within the oral cavity, and maintains intraoral pressure. Additionally, it facilitates mechanical cleansing of the oral cavity, thereby promoting improved local drug absorption and therapeutic action. [24]

Patoladi Kashaya contains ingredients possessing anti-inflammatory, antibacterial, anthelmintic, and wound-healing properties. These pharmacological actions contribute to reduction of gingival inflammation, control of microbial load, and promotion of tissue healing, resulting in marked symptomatic relief. [25]

Based on both theoretical principles and clinical observations, Patoladi Kwatha Gandusha can be considered an effective and safe therapeutic approach for the management of Sheetada (gingivitis).

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