

Case Report

Reducing allergic symptoms through eliminating subgingival plaque

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ABSTRACT

Background: Elimination of subgingival plaque for prevention and treatment of periodontal diseases through scaling is a routine procedure. It is also well-known that periodontal disease is related to systemic diseases. Nevertheless, the idea how scaling procedures also able to reduce allergic symptoms i.e. eczema and asthma, is not easily accepted, because it is contradictory to the “hygiene hypothesis”. However, since allergic symptoms also depend on variable factors such as genetic, environmental and infection factors; every possible effort to eliminate or avoid from these factors had to be considered. Subgingival plaque is a source of infection, especially the Gram-negative bacteria that produced endotoxin (lipopolysaccharides, LPS), a potential stimulator of immunocompetent cells, which may also related to allergy, such as mast cells and basophils. In addition, it also triggers the “neurogenic switching” mechanism which may be initiated from chronic gingivitis. **Objective:** This case report may explain the possible connection between subgingival plaque and allergy based on evidence-based cases. **Case:** Two adult siblings who suffered from chronic gingivitis also showed different manifestations of allergy that were allergic dermatitis and asthma for years. They were also undergone unsuccessful medical treatment for years. Oral and topical corticosteroids were taken for dermatitis and inhalation for asthma. **Case Management:** Patients were conducted deep scaling procedures, allergic symptoms gradually diminished in days even though without usual medications. **Conclusion:** Concerning to the effectiveness of scaling procedures which concomitantly eliminate subgingival plaque in allergic patients, it concluded that this concept is logical. Nevertheless, further verification and collaborated study with allergic expert should be done.

Key words: allergic symptoms, scaling, subgingival plaque

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INTRODUCTION

Allergic diseases may manifest in variety of symptoms i.e. rhinitis, asthma and eczema. Additionally, a strong association between sensitization and symptoms of allergic diseases is exists. Children with asthma, eczema or rhinitis are more likely to be sensitized to one or more allergens than those without these diseases. The “allergic march” alludes to the natural history of allergic diseases in childhood whereby eczema and sensitization to ingested allergens in infants and toddlers often improve by preschool age, when asthma and rhinitis become more prevalent, along with sensitization to aeroallergens.¹ Children who are sensitized to any allergens early in life have an increased risk of subsequently developing wheeze, airway hyper-responsiveness or rhinitis. There is also evidence

that the prevalence of allergic sensitization increases progressively during school years and early adulthood, then often decreases throughout this time.¹ Nevertheless, it is interesting that in this case report, allergic symptoms which already reduce with age, but in middle adulthood (in the 30s), it recurrent and became more severe. Additionally, even though they were sibling, it manifests in different symptoms, one has asthma and the other eczematous dermatitis (eczema).

The oral focal infection theory had been studied almost for 100 years, and had been experienced the “fall” and “rise” era. It had been disregarded since 1930s after failures of symptoms relieving following extractions of suspected teeth. Nevertheless, it was “reborn” in the 1990s by Taubmann et al. who begin with the scientific research related to the focal infection theory.² Li *et al.*³

also contributed new theories about oral focal infection and revealed the importance lipopolysaccharides (LPS), the endotoxin of Gram-negative bacteria. Gram-negative bacteria are abundant in the subgingival plaque. Elimination of subgingival plaque reduce the risk of several disease i.e. stroke and diabetes mellitus. However, the exact mechanism how elimination of focal infection may reduce allergic symptoms is not clearly understood.

Several case reports revealed that elimination of oral focal infection relief allergic asthma symptoms in children⁴ or adult.⁵ Focal infection may originate from pulpal and/or periodontal infection. Periodontal infection is caused by subgingival plaque. Furthermore, a collaborated clinical research conducted by dental practitioner and pediatrician revealed that elimination of dental plaque reducing allergic asthma symptoms.⁶ Nevertheless, researches regarding the connection of focal infection and asthma in adult mostly were epidemiological studies and did not support this concept. Friedrich et al. study revealed that adult periodontitis had inverse association with respiratory allergy.⁷ Additionally, Arbes et al study showed that higher immunoglobulin G (IgG) to *porphyromonas gingivalis* were significantly associated with lower prevalences of asthma, wheeze, and hay fever.⁸ These literatures supports the "hygiene hypothesis". Hygiene hypothesis stated in laymen's term that "cleanliness makes people more allergic".^{7,8} Therefore, elimination of focal infection that caused by subgingival plaque for reducing allergic symptoms is not easily accepted by medical personnel. Thus, explanation of the possible etiopathogenesis which may lead to the development of a new concept regarding the connection between subgingival plaque and allergy should be reviewed.

The objective of this case report is to elucidate the possible etiopathogenesis of subgingival plaque-related allergy based on evidence-based cases which may beneficial for the development of a new concept and researches. Through researches, this concept will be made clear and established.

CASE

Case 1: Male, 36 years old, lived in Surabaya came to the dental clinic after informed by his relative about the possible connection between oral infection and asthma. He suffered from allergic asthma when he was 6, and after had swimming lesson, the symptoms disappeared. His parents are allergic, especially to dairy product (i.e. milk). When he was in the late 20s, he worked in Bandung for 6 years, and asthmatic symptoms recurrent. Latest medication was bronchodilator (inhaler), which at the time was used until 8 puffs/day; this was considered the severe persistent asthma according to the classification of asthma.

Extraorally, he looked tired and slow. Intra-oral examination showed abundant calculus and severe chronic gingivitis with pseudopockets in 18 17 16 and 26 27 28.

He said that when brushing his teeth, the upper gingiva was easily to bleed.

Case 2: Male, 34 years old, his younger brother came to the dental clinic after told by her brother that his asthmatic symptoms were disappeared. He had been suffered from eczematous dermatitis (eczema) for seven years in both feet; nevertheless, the left foot had more severe symptoms after moving from Surabaya to Banjarmasin. When he was a child he suffered from otitis media and had been conducted paracentesis for 3 times. Eczema was first treated with oral corticosteroid for years; unfortunately it caused osteoporosis, then it was stopped. Subsequently, it was substituted with topical corticosteroid, nevertheless the symptoms still very annoying.

Extra oral examination looked normal. Intra orally, he had only mild chronic gingivitis. The 48 and 38 had pseudopockets, nevertheless 38 was buccoverversion and had deeper lingual and distal pseudopockets.

CASE MANAGEMENT

At the first visit, the management of these patients was merely deep scaling; before scaling procedures, the patients were told to rinse with hexetidine 0.1% for 30 second. Deep scaling was done with piezoelectric scaler followed by hand instrument, a thin universal scaler, which focused on the chronic gingivitis and pseudopockets area. During deep scaling, especially after scaling with hand instrument the gingival bleed easily. However it was beneficial for drainage of the pro-inflammatory mediators, thus reducing the inflammation. They were also prescribed hexetidine gargle 0.1% twice/day for daily maintenance, and were scheduled for evaluation one week later.

At the second visit, the asthmatic patient was relieved from most of the symptoms, he only use inhaler once/day. Two weeks later he moved to Dubai, however when evaluated, her mother said that all of the asthmatic symptoms were disappeared. The second patient came two weeks after his first visit, he also felt that the symptoms were diminished. Since the possible cause of eczema was oral focal infection from 48 38, especially 38 which had deeper pseudopocket, he was advised to checked regularly to a dental practitioner. However, for convenience, those teeth had to be conducted gingivectomy or to be extracted.

The latest evaluation of the patients in this case report was approximately one year and 10 months later respectively. The asthmatic symptoms did not recurrent, so did the eczema in the right foot, however in the left foot may have a mild symptom if consuming dairy product (i.e. yoghurt).

DISCUSSION

The diversity of allergic symptoms throughout the life is likely, because according to allergic march, infants

who had atopic dermatitis may have rhinitis or asthma in the following years. The etiopathogenesis of allergy is multifactorial i.e. genetics, environmental and allergens factors.¹ However, it is still unclear why oral focal infection may involve in allergic development and symptoms. Since oral focal infection usually related to toxins from oral pathogenic bacteria, the possible involvement of LPS from Gram-negative bacteria is possible. Additionally, recent investigations showed that LPS stimulation plays a synergistic role with antigen⁷ and increase the immunoglobulin E (IgE) level.⁹

The oral infection theory revisited was conducted by several researchers such as Taubman and Slots in 1992, and Li *et al.*³ Nevertheless, in their researches, the main cause of systemic effects of oral focal infection was the immunological reaction of the host to bacteria and toxin.² It was in accordance with Pejic *et al.*,¹⁰ in 2006 that oral focal infection can play a part in the creation of respiratory infections that are manifested as sinusitis, tonsillitis, pneumonias, bronchial asthma etc. These diseases can be caused by the microorganisms from the oral cavity, following a direct inhalation from saliva and dental plaque, or by blood dissemination.

There have also been numerous other descriptions of the mechanism where oral bacteria have been included in the pathogenesis of respiratory infections, i.e. *porphyromonas gingivalis* and *actinomyces actinomycetemcomitans* which can aspire into the lungs and cause infection (droplets infection); then the host's and bacterial enzymes from the saliva can dissolve saliva pellicula on pathogens and allow them to adhere to the surface of mucous membrane; and also cytokines derived from the periodontal tissue can damage the respiratory epithelium by causing an infection via respiratory pathogens. Damage of respiratory epithelium may lead to increase its sensitivity to respiratory allergens or stimulation.^{11,12}

However, the possible involvement of oral focal infection in the etiopathogenesis of asthmatic symptoms is not merely immunological reaction. The subgingival biofilm which contains Gram-negative bacteria releases LPS which may induce immunogenic and neurogenic inflammation that is proposed termed as the "neurogenic switching mechanism" by Meggs in 1993 which explain why local inflammation is able to propagate and stimulates inflammation in distant organs (Figure 1).¹² Actually, according to Lundy and Linden¹³ this mechanism also occurs in periodontal disease via mast cell stimulation by LPS which releases histamine, enzymes and cytokines. These enzymes and mediators then stimulate nerve endings to produce neuropeptides (i.e. substance P) that in turn induce mast cell activation.

It is interesting that since allergy is an inherited disease, and in this case report the patients were sibling. Coincidentally, it is well established that these periodontal pathogens are transmitted from parent to child (especially the mother in infant), from sibling to sibling, and between spouses. Transmission is most likely through contact with

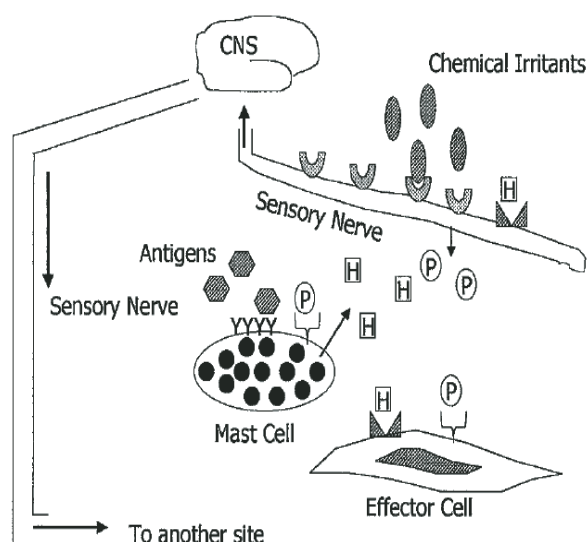


Figure 1. Neurogenic switching. The site of inflammation is switched from the site of inoculation in both allergy and chemical sensitivity by pathways through the central nervous system (CNS).¹²

saliva and the sharing of objects such as cups, spoons, and toothbrushes.^{14, 15} Therefore, it is also interesting to conduct researches for investigating the connection between the transmission of periodontopathic bacteria and allergy that could be valuable to prevent allergic diseases.

Even though the previous explanation of the mechanism of asthmatic symptoms which related to the neurogenic switching mechanism could be happened in non-allergic asthma (non IgE-mediated); the effects of LPS from different strain of periodontopathic bacteria also play an important role in the development of allergy. According to Kato *et al.*,¹⁶ induction of neonatal Balb/c mice with low dose of LPS from *actinomyces actinomycetemcomitans* (AaLPS) and *porphyromonas gingivalis* (PgLPS) gave different cytokines profile. The AaLPS produces T-helper1 (Th1) profile cytokines, whereas PgLPS produce T-helper2 (Th2) which related to allergy. In addition, IgE level in the control mice were lower than mice induced by PgLPS.

It is clear that the removal of subgingival plaque by deep scaling also reduce LPS and pro-inflammatory mediators which may involved in the neurogenic switching mechanism and allergic reaction. Nevertheless, according to Friedrich *et al.*,⁷ this treatment could be contradictory for allergic diseases, because periodontitis patients were considered allergy-resistant and in accordance with the hygiene hypothesis. The plausible answer of this ambiguous question is that there are several types of periodontitis with different etiologies that are periodontitis-only, often with severe resorption (Th1 disease), and periodontitis with gingivitis (Th2 disease). The patients in these case report suffered from periodontitis with gingivitis, thus elimination of sub gingival plaque for reducing allergic symptoms was likely.

Another contradictory study which conducted by Arbes *et al.*⁸ showed that higher IgG to *porphyromonas gingivalis* was related to the reduced prevalence to allergy. It could be explained that lower concentration of PgLPS which existed in chronic gingivitis or chronic periodontitis with gingivitis stimulates the toll-like receptor2 (TLR2), which increase the production of Th2 cytokines profile, i.e. interleukin-4 (IL-4) which related to allergy. Moreover, low PgLPS is antagonist to the TLR4 that resulted in less Th1 cytokine production, the interferon γ (IFN- γ). Higher PgLPS concentration is related to TLR4 stimulation and more severe periodontitis.^{17,18}

Moreover, according to Burt,¹⁹ periodontitis with gingivitis also have more prostaglandin E2 (PGE2) in the pseudopocket than periodontitis-only; this resulted in the increase of sensory nerve ending sensitivity, or lowering of pain threshold, especially the trigeminal nerve.²⁰ Nevertheless, it is interesting that in chronic periodontal disease this condition do not manifest as pain. According to Wadachi and Hargreaves,²¹ it was the effect of pain receptor cleavage by proteases from periodontopathic bacteria. Hence, the corresponding nerve is still in lower threshold which more easily stimulates by cold, capsaicin, glutamate etc.²² In the first case, patient moved to Bandung, a city with more rainy weather, humid and colder climate. Chronic stimulation of environmental factors to the asthmatics may result in airway remodeling that increase asthma severity.²³

Sensitization of the maxillary nerve, the second branch of the trigeminal nerve in the oral cavity which may propagate antidromically (opposite to the direction of regular impulse) to the nasal cavity is able to stimulate parasympathetic nerve directly via the sphenopalatine ganglion (SPG),²⁴ or indirectly via the trigeminal nucleus caudalis which acts as a relay center.²⁵ Since nasal cavity also innervated by the maxillary nerve; stimulation of this nerve can reflexively influence nasal engorgement, respiration rate, nasal secretion, and sneezing. Since most trigeminal stimulants were lipid soluble, such as volatile chemicals; the stimulations were likely. Stimulated trigeminal nerve and the sphenopalatine ganglion (SPG) may referred to multiple chemical sensitivity syndrome (MCS) which initiated in the nasal cavity.^{26,27}

The connection between periodontal disease and eczematous dermatitis is somewhat difficult to explain. The traditional etiopathogenesis of eczema is related to food allergy, especially in children.²⁸ Nevertheless, according to Pejicic *et al.*,¹⁰ skin diseases could also related to oral focal infection, which most frequently occur as the consequence of transmission of microbes from dental foci are allergic diseases (urticaria, eczema etc.), lichen planus, acnae vulgaris, etc. It was in accordance with Li *et al.*³ that oral microbes and toxins and also responsible for the release of histamine from mast cells or creation of circulating immune complex which resulted in skin problems.

Nevertheless, since in this patient the removal of subgingival biofilm in the pseudopockets especially in

38 also reduce eczematous symptoms in the left foot; the possible involvement of the neural system and not merely immunological was possible. The neurogenic switching mechanism is able to give an appropriate explanation. Furthermore, according to the somatosensory system or the somatosensory homunculus of the brain, the skin sensitivity could be unilateral to the trigger area because it is located in the same somatosensory cortex (Figure 2).²⁹ This mechanism could be mimicking to the referral pain system i.e. temporomandibular pain is originated from shoulder pain (Figure 3).²⁴

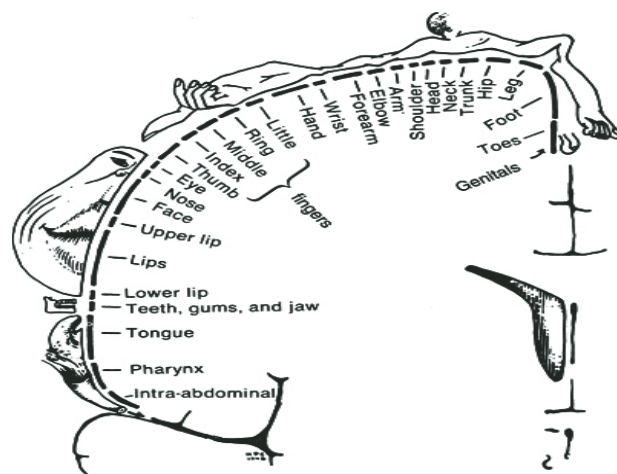


Figure 2. 'Sensory homunculus', illustrating the projection of various body regions on the sensory cortex.²⁹

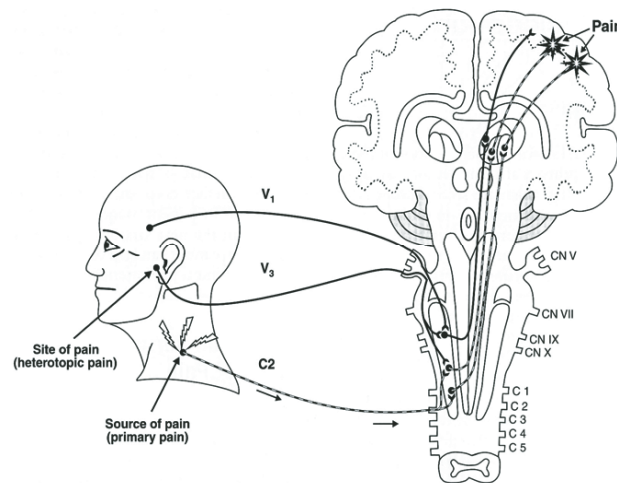


Figure 3. Pain impulse from the shoulder (source of pain) may perceive as pain in the site of pain, because they have adjacent converging neuron in the sensory cortex. This phenomenon is termed as heterotopic or referral pain.²⁴

For the concluding remarks, it is the important role of LPS which is able to stimulate TLR2, thus shifting Th1 cytokines profile to Th2 locally, which may propagate to systemic and also stimulate the periodontal afferent nerve endings which initiates the "neurogenic switching"

mechanism. Therefore, our concept that elimination of subgingival plaque may reduce allergic symptoms is logical. However, since these evidence-based cases are only examples from uncontrolled allergy by conventional medication, and that allergy is related to multifactorial etiologies; collaborated studies with medical personnel are mandatory. In addition, since transmission of periodontopathic bacteria also related to parent, sibling and spouses, other research could be done to investigate its possible correlation with the spread of allergic disease.

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