

Connection Between the Course of Neurodermitis and Oral Focus Finding

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Summary

A case of a patient suffering from neurodermitis, and with a finding of oral focus is described. The worsening of neurodermitis was concomitant with a finding of oral focus. After elimination of the focus, neurodermitis regressed from the severe form.

Key words: *neurodermitis, oral focus*

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CASE REPORT

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Introduction

Kotschau (1972) defined focus as a separate, local, inflammatory and intermittently acting anatomical change in the oral cavity, which can induce morphological or pathological changes on distant tissues or organs. The entity called focalosis represents response of an organism to a long distance acting focus. Focal infection is a disease of a distant tissue or organ which has been caused by a focus (1). The pathological process which acts as a focus must be clearly circumscribed from the surrounding environment, strongly perfused with blood and infected. In the oral cavity oral foci could be periapical processes (ostitis, cysts), foreign bodies such as dental materials or instruments, unerupted infected teeth, parodontal pockets deeper than 5 mm while probing, tonsillitis and sinusitis maxillaris. Oral foci can spread into extraoral tissues through three pathways (2):

- 1) metastatic infection
- 2) metastatic injury due to bacterial toxins

- 3) metastatic inflammation due to immunological injury caused by bacteria from the oral cavity

Oral foci mostly induce pathological changes on cardiovascular, dermatological, locomotory, neurological and ophthalmological systems (3,4,5,6). Diseases of the skin mostly associated with foci are some forms of eczema, erythema exudativum multiforme, dermatitis atopica, alopecia areata, acnae vulgares, dermatitis herpetiformis, erythema nodosum and lichen ruber planus. Oral foci induce massive bacteremia which cannot only induce pathological changes on distant organs but can also lead to worsening of the basic disease (7,8,9). Kogoj (10) suggests that numerous exogenous and endogenous factors have a very important role in the development of neurodermitis, such as endocrine-vegetative dystony, focal infection, gastrointestinal diseases and climate. The same author concludes that sensitization in neurodermitis is caused by various microorganisms, mostly streptococci and staphylococci that are either in the patient's environment or spread from foci. In the literature

connection between *Staphylococcus aureus* and neurodermitis is mostly described. Bacterial toxins act as superantigens and are lined up with MHC II antigens and can directly stimulate massive proliferation of about 20% T-cells while usual antigens activate only 0.1% T-cells. T-cells then lead to cytokine release (11).

Case report

A 35-year-old patient, BS, was referred to our Department with diagnosis of neurodermitis generalisata and suspected of having an oral focus. The patient had skin changes for three years, which appeared after her father's death which has induced severe stress in our patient. Four years ago conization was performed because gynaecological Papa finding was IV. After which her health was good. At the age of seven years she had undergone tonsillectomy due to recurrent pharyngeal infections. She did not have any other systemic disease. When testing with epicutaneous patch tests positive results to allergens, such as BREZA, grass, weed, *D.pteronissei*, olive oil, ascaris, eggwhite, milk, pork, beef, gluten and feather were found. Blood tests AST, ALT, γ -GT and complete blood count were found to be within normal ranges, while values of IgE were several times higher than 5000 kIU/L (normal range up to 114 kIU/L). Slightly elevated gamma globulin values were 21.6% (normal range 11.2-19.9%) while albumin values were 57.4% (normal range 59-70.6). Finding of AST, ASTA, Waaler-Rose and Latex-rheuma factor were all negative. Oral examination revealed the need for oral rehabilitation. X-ray finding showed a potential foci localized on teeth 12 (Figure 1), 27 and 46. The patient also needed initial parodontal therapy. While testing the teeth for foci we applied Wannemachers test. Erythrocyte sedimentation rate (ESR) was determined before testing which was 11. Wannemacher test consists of treating (brushing) the teeth with a gum polishing device for a duration of 2 minutes, resulting in bacteremia. Within 24 hours blood was taken again from the patient and ESR value was 38. ESR value elevated more than double suggested that the tested tooth was a focus. Teeth 27 and 46 did not appear to be foci when tested. After treating tooth 12, neurodermitis improved.

Discussion

Several pathogenic mechanisms may be involved in the proposed exacerbation of atopic eczema by staphylococcal superantigens (12). Superantigens may also be involved in other skin diseases. Several inflammatory skin diseases are known to be colonized with *S.aureus*, e.g. atopic dermatitis, contact dermatitis and psoriasis. It is common by found that infection and even colonization with *S.aureus* may lead to exacerbation of skin diseases. After superantigens have been released from the streptococcal infection and transported into blood they bind to antigen-presenting cells in the skin (epidermis and dermis), which in turn activate T-cells, a mechanism described in psoriasis (13). Halbert, Weston and Morelli (14) suggested that superantigens can bind to class II MHC molecules on epidermal Langerhans cells, macrophages and monocytes and cause the release of proinflammatory mediators such as IL-1 and tumor necrosis factor alpha. Through cross-linking MHC class II molecules on antigen presenting cells and the variable domain of TCR-beta, T-cells can be stimulated to proliferate and secrete a range of inflammatory cytokines. Sehgal and Jain (15) reported that colonization by *S.aureus* may be either primary or secondary. It probably initiates inflammation by liberating toxins that in turn activate the keratinocytes to express ICAM-1 and secrete IL-1, IL-3 and other cytokines. *Staphylococcus aureus* may also liberate biologically active substances by degranulating the mast cells through antistaphylococcal IgE antibodies or by directly adhering to the skin or mucosa through specific binding sites such as protein A, teichoic acid, fibronectin and laminin. Certainly we have to admit that there is a connection between oral and systemic health as well as oral and systemic disease. For some evidence is strong, for others less so and for others intriguing. Only our research together with that of other medical colleagues will enlighten (16).

We can conclude that worsening of neurodermitis is connected with a finding of oral foci. Although complete regression of neurodermitis was not observed a marked reduction of symptoms was noticed following treatment of the oral focus - the tooth 12.