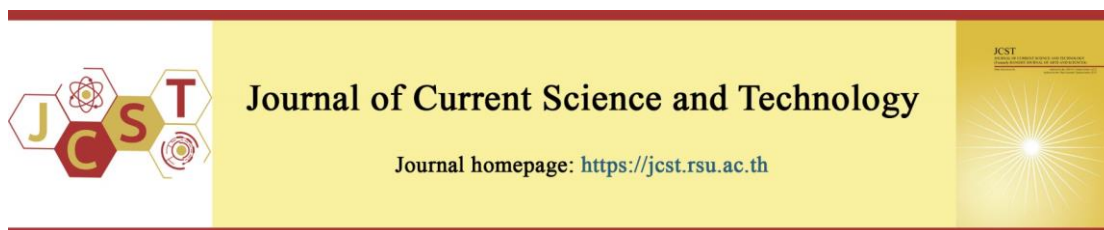


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Allergic contact dermatitis to nickel in ophthalmic frames

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Abstract

Contact dermatitis from a xenobiotic irritant or allergen, negatively impacts quality of life. Nickel is the most prevalent contact allergen in studies. In the late 18th and early 19th century nickel allergy was an occupational hazard, while today nickel exposure is predominantly from consumer goods. Nickel allergy is prevalent throughout the world, more frequently in women. Limiting chronic nickel exposure and preventing sensitization is the best treatment. Consumer items such as jewelry, especially earrings and other piercings, metal fasteners on clothing, watches, coin money and cell phones are common sources of exposure. Both the innate and adaptive immune systems have been shown to be involved in the complicated epidermal processing of nickel as an allergen. The optometric patient population frequently wears eyeglass frames contain nickel, raising the risk of nickel exposure, sensitization and contact dermatitis. Optometrists should be aware of nickel allergy from eyeglass frames and recommended treatment options.

Keywords: *adaptive immunity; contact dermatitis; innate immunity; nickel allergy; nickel in ophthalmic frames.*

1. Introduction

Allergic contact dermatitis (ACD), is a type IV delayed-type hypersensitivity reaction (DTH) initiated by the innate immune system and progressing to involve T cells of adaptive immunity with a more profound dermatitis (Silvestre, Sato, & dos Rios, 2018). Nickel is perennially the most common cause of clinical ACD. 17.5% of 5597 patients tested in North America, were positive for nickel ACD (DeKoven et al., 2018). Women show a significantly greater prevalence, 15.7% of 14,873 females while only 4.3% of 11,157 males exhibited nickel ACD (Alinaghi, Bennike, Egeberg, Thyssen, & Johansen, 2018). 5-15% of women in Thailand show sensitivity to nickel with most sensitized by their early teen years with risk factors being: young age, long exposure to metals, seafood and canned food consumption (Boonchai, Chaiwanon, & Kasemsarn, 2014). A more recent study found 19.4% of the Thai population show nickel sensitivity, with 22.6% of females and 11.7% of

males (Dararattanaroj, Poontongkam, Rojanawatsirijev, & Wongpiyabovorn, 2017).

Optometric patients often present with ACD in a characteristic distribution pattern where eyeglass frames touch the bridge of the nose, the cheeks, and where the temples rest on the side of the head, as metal ophthalmic frames commonly contain nickel (Nakada & Maibach, 1998). Patients have presented with typical nickel ACD from eyeglass frames while wearing 'nickel-free' titanium frames, or other frames labeled 'nickel-free' (Walsh & Mitchell, 2002). Some eyeglass frames, when tested, have demonstrated the presence of nickel despite 'nickel-free' labeling. The presence of nickel in eyeglass frames, jewelry or other metal can be demonstrated with the colorimetric dimethylglyoxime (DMG) test, with 1% DMG and 10% ammonium hydroxide combined on a cotton swab, when rubbed on a metal surface for up to 60 seconds, produces a pink precipitate on the swab if nickel is released at

greater than 0.5 $\mu\text{g}/\text{cm}^2/\text{week}$ (Thyssen et al., 2010). Patch-testing for nickel ACD is conducted with 2.5% nickel sulfate in the US and 5% in Europe and release of less than 0.5 $\mu\text{g}/\text{cm}^2/\text{week}$ of nickel is below an exposure threshold inducing ACD.

Eyeglass frame suppliers often acquire frame components from a series of manufacturers, finished elsewhere, and finally assembled in yet another country, resulting in a poor, or nonexistent, manufacturing trail or record of materials and compounds used. Manufacturers using multinational supply lines, proprietary processes and chemical treatments in the finish of eyeglass frames may be unwilling or unable to provide specific information on the exact composition of frame materials or contaminants. Chemicals or toxins commonly found on eyeglass frames include: alcohol, soaps, detergents, solvents, fiberglass, plasticizers, antistatic agents, lubricants, polishing creams and other irritants, (Shono, Numata, & Sasaki, 2018). Direct cellular damage from chemical irritants is responsible for the localized cutaneous inflammation and disruption of the skin's barrier function (Leonard & Guttman-Yassky, 2019) and may persist for 2 or 3 days after removal of the offending agent. Nickel exposure is most frequently from consumer goods, including: jewelry: earrings, finger rings, watches, watchbands, metal buttons, bracelets, necklaces, belt buckles, coins, mobile phones and eyeglass frames (Boonchai, Maneeprasopchoke, Suiwongsa, Kasemsarn, 2015). Buttons, fasteners, rivets and zippers containing nickel in clothing also contribute to nickel ACD. The thickened, horny barrier of skin on the palms of the hands is thought to mitigate nickel ACD when handling coins containing nickel, though some cashiers still develop ACD (Hamann, Hamann, Hamann, Thyssen, & Lidén, 2012). Chloride ions present in sweat facilitates production of nickel ions and ACD. Hot, humid climates increase the likelihood of sweating with an increase in chloride ions, facilitating the liberation of nickel ions which readily penetrates the epidermis.

Women whose ears were pierced after enactment of the nickel regulation showed decreased prevalence of nickel ACD at 11.4%, compared to women with ears pierced before regulation at 19.8%, (Ahlström, Menné, Thyssen, & Johansen, 2017). Thailand and the United States do not regulate consumer exposure to nickel, and the prevalence of nickel ACD was found to be 19.4% in

Thailand (Dararrattanaraj, et al., 2017) and 17.5% in the US (DeKoven et al., 2018).

2. Case Report

A 24-year old Hispanic female presented for a comprehensive eye examination. Her ocular history was unremarkable other than having worn glasses since middle school, to improve her distance vision. A low correction for myopia gave vision of 20/20 in either eye. Ocular examination findings were unremarkable and non-contributory. General observation of the patient revealed a localized acne-like rash on her cheeks, underlying where the metal eyewire rested. Less obvious was a similar, preauricular rash on both sides of her head where the frame temples rested. The patient noted the 'acne' had been there for more than a year. She acknowledged it was mildly irritating, mostly itchy. The patient was informed the malar and temple rash appeared to be nickel ACD, and is likely sensitized to nickel. Avoiding contact with nickel, including selecting eyeglass frames that did not rest on her cheeks, wearing a polymer or nickel-free metal frame were recommendations. Contact lenses and lasik were also discussed as strategies to help avoid nickel exposure. The option of referral for dermatological patch testing was presented within a discussion of ACD. Topical 1% hydrocortisone cream was prescribed to be used twice daily for one week. The patient was appreciative of the diagnosis and information. She greatly anticipated the cosmetic improvement of a clear complexion, and declined patch testing. She did not return as scheduled and was lost to follow-up.

3. ACD Treatment

Preventing sensitization to nickel is the best strategy to prevent nickel ACD. Barrier creams and lotions and use of topical chelation therapy with ethylenediaminetetraacetic acid (EDTA) may further reduce the degree of exposure (Anderson, & Aaseth, 2016). Once sensitized to nickel, application of cold, moist compresses helps relieve edema and erythema in more severe or acute presentations, which can take from days to months to fully resolve. Oral antihistamines, including diphenhydramine, can help reduce the itch of ACD. Topical hydrocortisone cream applied locally on a short-term basis, reduces the immunologic inflammation. Tacrolimus 0.1% ointment, a calcineurin inhibitor blocking transcription of inflammatory mRNA in lymphocytes, has also

demonstrated efficacy in reducing ACD (Mose, et al., 2018). Acute occurrences may be treated with topical corticosteroids or calcineurin inhibitors. Some recalcitrant cases have been treated with immunosuppressants: methotrexate, cyclosporine or azathioprine to control inflammation (Martin, Rustemeyer, & Thyssen, 2018). A recent option for a subgroup of patients, where first- and second-line therapies have failed is use of dupilumab, an immunobiologic (Jacob, Sung, & Mahler, 2019). Preventing exposure and sensitization through lifestyle choices is the best and only definitive strategy for nickel ACD.

4. Conclusion

Nickel ACD is highly prevalent, more so in women, and in countries without nickel regulations. The prevalence of nickel ACD is increasing around the world (Rietschel et al., 2015), as is the prevalence of myopia (Holden et al., 2016), suggesting ever more people will be wearing eyeglasses. Knowledge and awareness of ACD, history taking and observation of the patient, facilitates recognizing the signs and symptoms of nickel ACD, particularly from eyeglasses. Earlier patient education, diagnosis and treatment allows for more prompt care and relief for the allergic population. Preventing exposure and sensitization to nickel will help mitigate the increasing prevalence of nickel ACD. Optometrists and opticians should be aware of ICD and nickel ACD signs and symptoms from eyeglass frames as well as sensitization from jewelry and piercings. A limitation of this report is not having DMG testing available at the time of examination to confirm presence of nickel in the spectacle frames. Allergen patch testing would have provided confirmation of ACD but at increased cost and inconvenience to the patient, which was declined. In this case, removing the suspect frame from the patient exhibiting classic nickel ACD from eyeglass frames was a clinically appropriate treatment option.

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