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# Review Article

## From Contact to Reaction: The hypersensitivity side of orthodontic practice – A Clinical Overview

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***ABSTRACT:***
Hypersensitivity in orthodontics is a frequently reported concern among patients undergoing fixed or removable appliance therapy. It can develop as a response to thermal, tactile, or chemical stimuli during various stages of treatment. This condition is often linked to factors such as enamel demineralization, dentin exposure, and periodontal changes resulting from orthodontic force application. While typically temporary, hypersensitivity can affect a patient's comfort, compliance, and overall treatment experience. Identifying underlying causes and applying appropriate preventive and management strategies are essential for maintaining oral health and improving patient outcomes during orthodontic care.
**Keywords:** Hypersensitivity in orthodontics, Nickel hypersensitivity, Orthodontic materials, Allergic reactions.

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## INTRODUCTION

Hypersensitivity reactions are adverse immune responses to specific antigens, leading to tissue damage and clinical symptoms. In orthodontics and dentofacial orthopaedics, such reactions can be triggered by materials used in appliances, including metals like nickel and chromium, as well as latex products. Understanding these reactions is crucial for clinicians to prevent and manage adverse effects in patients undergoing orthodontic treatment. Nickel, commonly found in stainless steel brackets and wires, is a significant allergen in orthodontic materials. Studies have shown that nickel can provoke a late-phase, type IV hypersensitivity reaction, characterized by signs such as gingival overgrowth, angular cheilitis, and labial desquamation in the oral cavity.[1]

The prevalence of nickel hypersensitivity in orthodontic patients varies but is significant, particularly among those with a prior history of nickel allergy from jewellery or other exposures.[2] Therefore, a thorough patient history and appropriate diagnostic testing are essential for identifying hypersensitivity reactions and ensuring safe orthodontic treatment.

Proper understanding of these immune responses is crucial for safe and effective patient care.[3]

### History of Hypersensitivity

Hypersensitivity reactions in the context of orthodontics and dentofacial orthopaedics have been recognized and studied since the increased use of synthetic and metallic materials in dental appliances during the mid-20th century. Early reports in the 1960s and 70s documented cases of oral mucosal irritation and contact dermatitis linked to nickel-containing orthodontic devices, especially in patients with a pre-existing history of metal allergies. As stainless-steel alloys became the standard material for brackets and wires, the incidence of hypersensitivity reactions gained attention due to the high nickel content.

The development of latex gloves and elastics in orthodontic practice also introduced new allergens, leading to cases of immediate-type (Type I)

hypersensitivity reactions, such as urticaria and anaphylaxis in sensitized individuals. By the 1990s, awareness of material biocompatibility had significantly increased, prompting manufacturers to produce nickel-free brackets, latex-free elastics, and alternative bonding agents to reduce allergic risks.

Today, with greater understanding of immunological mechanisms and improved diagnostic tools like patch testing, clinicians are better equipped to identify and manage hypersensitivity in orthodontic patients. The historical evolution of this awareness underscores the importance of individualized treatment planning and material selection in orthodontic and dentofacial orthopaedic care.

Diagram of the original idea of allergy developed by von pirquet. When the individual contacts with an antigen (germs, pollens, foods, ect.), a change in reactivity occurs. This change- "allergy" according to von Pirquet can induce a protective or harmful response: the protective one renders the individual immune to the antigen, that is, he does not manifest any symptom or signs after exposure to the antigen and the harmful one causes signs and symptoms after such a contact the first response is known as "immunity" and the second as "hypersensitivity". Both are the ends of the same physiological process and can overlap.

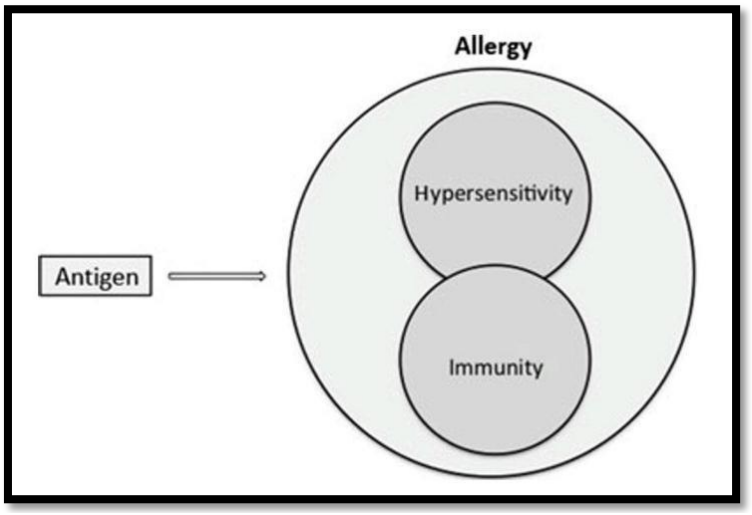


**Figure- 1: History of the idea of allergy**

**Table 1: Timeline of Hypersensitivity in Orthodontics**

| Time Period | Event / Development | Significance | Reference |
|---|---|---|---|
| 1960s–1970s | Initial reports of allergic reactions to metals like nickel in orthodontic appliances | Recognized oral lesions and dermatitis in patients using stainless steel brackets and wires | Kerosuo et al., 1996[4] |
| 1980s | Increased use of latex gloves and elastics in dental and ortho practice | Emergence of immediate-type (Type I) hypersensitivity, including anaphylactic reactions | Binkley et al., 1999[5] |
| 1990s | Growing awareness about material-related allergies in dentistry | Introduction of nickel-free brackets and latex-free materials | Binkley et al., 1999[5] |
| 2000s–Present | Emphasis on biocompatibility and patient-centered material selection | Use of patch testing, patient history screening, and hypoallergenic alternatives | Malkoc et al., 2010[6] Golz et al., 2015[7] |

**Classification of Hypersensitivity[3]**

The classification of hypersensitivity reactions was initially proposed by Gell and Coombs, but has since been refined with advancements in immunology. The classification is essential for understanding the immune mechanisms that underlie allergic reactions, and is useful for diagnosing and managing clinical conditions. Hypersensitivity reactions are categorized based on the immune response and the type of cells or molecules involved.

| Type | Name | Immune Mechanism | Onset duration | Examples |
|---|---|---|---|---|
| I | Immediate (IgE-mediated) | IgE antibodies → mast cells/basophils → histamine, cytokines | Seconds to minutes | Anaphylaxis, allergic rhinitis, asthma, urticaria |

| | | | | |
|---|---|---|---|---|
| IIa | Cytotoxic (Antibody-mediated) | IgG/IgM antibodies target cells → complement activation/cell lysis | Minutes to hours | Autoimmune hemolytic anemia, Goodpasture's syndrome |
| IIb | Antibody-mediated cell stimulation | Antibodies bind to cell surface receptors, altering cell function | Variable | Graves' disease, chronic idiopathic urticaria |
| III | Immune Complex-mediated | Antigen-antibody complexes → complement activation → inflammation | Hours to days | Serum sickness, systemic lupus erythematosus, post-streptococcal glomerulonephritis |
| IVa | Delayed-type (Th1-mediated) | CD4+ Th1 cells activate macrophages, leading to tissue damage | 48–72 hours | Tuberculin skin test, contact dermatitis |
| IVb | Th2-mediated | CD4+ Th2 cells recruit eosinophils via IL-4, IL-5, and IL-13 | Variable | Chronic asthma, drug-induced eosinophilic reactions |
| IVc | Cytotoxic T-cell-mediated | CD8+ cytotoxic T cells induce apoptosis of target cells | Variable | Stevens-Johnson syndrome, toxic epidermal necrolysis |
| IVd | T-cell and neutrophil-mediated | T cells recruit neutrophils through IL-8 and GM-CSF, leading to inflammation | Variable | Acute generalized exanthematous pustulosis (AGEP) |

**Immune Response During Hypersensitivity**

Hypersensitivity reactions refer to exaggerated or inappropriate immune responses that cause tissue damage upon contact with an otherwise harmless antigen. Each type of hypersensitivity—classified as Type I through Type IV—is mediated by distinct immunologic pathways involving specific immune cells and molecules.[8]

Type I (Immediate) Hypersensitivity – IgE-Mediated

Type I hypersensitivity is mediated by IgE antibodies, which are produced in response to initial exposure to allergens like pollen, food, or animal dander. Antigen-presenting cells (APCs) such as dendritic cells present the allergen to CD4+ Th2 cells, promoting class switching in B cells to secrete IgE. These IgE antibodies bind to FcεRI receptors on mast cells and basophils, sensitizing them. Upon re-exposure to the allergen, cross-linking of IgE leads to mast cell degranulation and the release of histamine, leukotrienes, and prostaglandins, causing symptoms such as urticaria, rhinitis, bronchospasm, or anaphylaxis.[9,10]

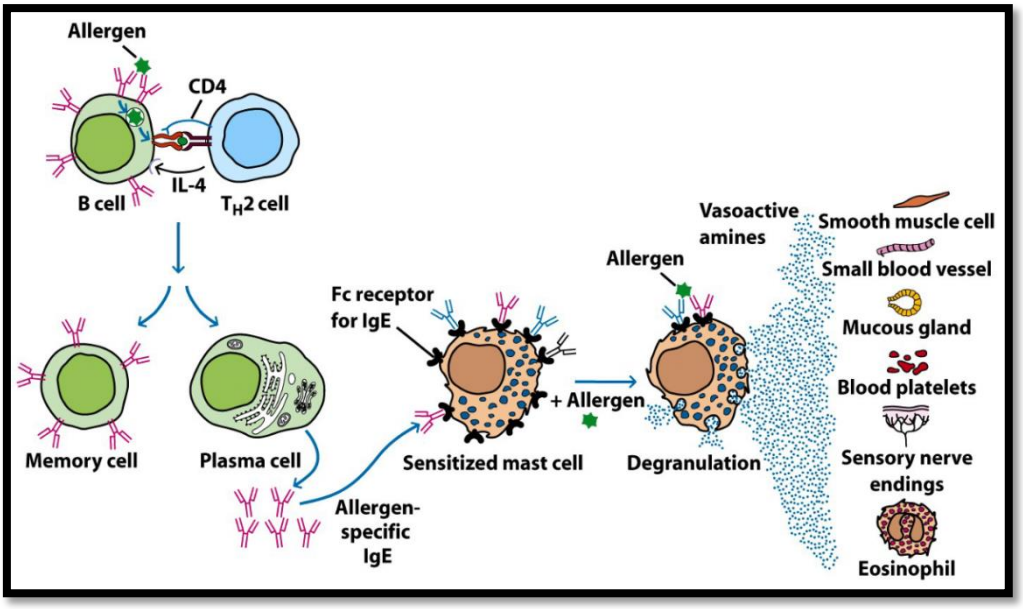


**Figure- 2 Diagrammatic representation of type I hypersensitivity reactions**

**Diagrammatic representation of type I hypersensitivity reactions**

Type II (Cytotoxic) Hypersensitivity – IgG/IgM-Mediated

In Type II hypersensitivity, IgG or IgM antibodies bind to antigens on host cell surfaces or extracellular matrix components. This can lead to complement activation, phagocytosis, or antibody-dependent cellular cytotoxicity (ADCC). Immune cells like macrophages and NK cells are recruited to destroy the opsonized target cells. Diseases such as autoimmune hemolytic anemia, Graves' disease, and Goodpasture's syndrome are classic examples of this mechanism.[3,10]

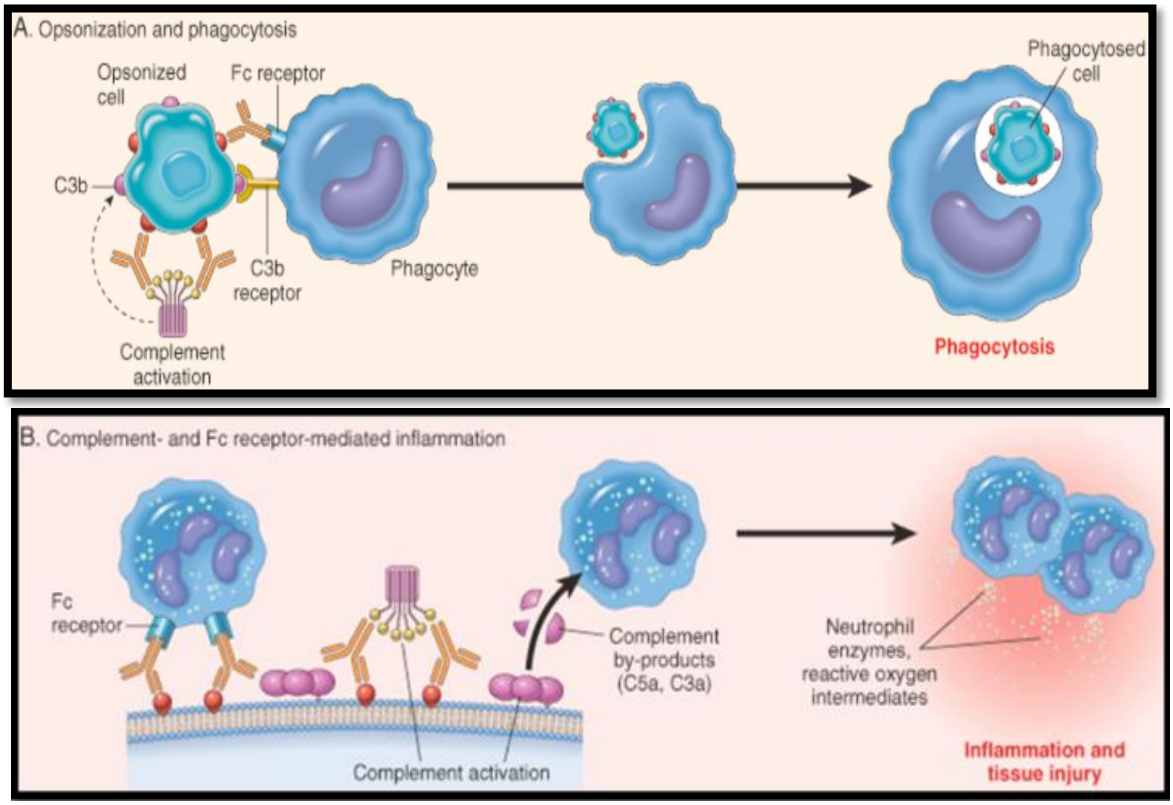


**Figure – 2: Type II Hypersensitivity reactions**

**Type III (Immune Complex-Mediated) Hypersensitivity**

Type III hypersensitivity is driven by the formation and deposition of immune complexes composed of antigens and IgG or IgM antibodies—in tissues like the kidneys, joints, and blood vessels. These immune complexes activate the classical complement pathway, resulting in inflammation and tissue damage through the recruitment of neutrophils and the release of destructive enzymes and reactive oxygen species. Clinical manifestations include systemic lupus erythematosus (SLE), serum sickness,and post-streptococcal glomerulonephritis.[11,8]

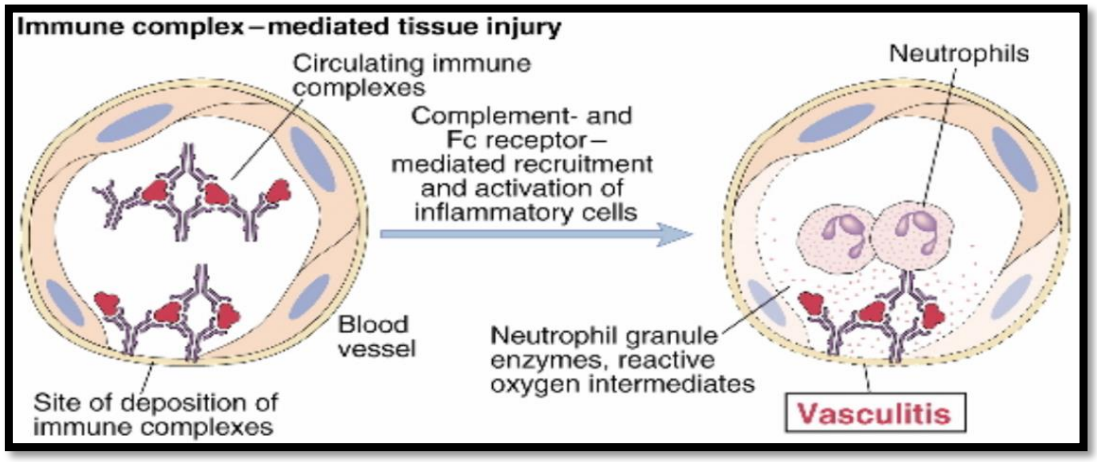


**Figure – 3: Type III Hypersensitivity reactions**

**Type IV (Delayed-Type) Hypersensitivity – T Cell-Mediated**

Type IV hypersensitivity is mediated by T lymphocytes, particularly CD4+ Th1 or Th17 cells and CD8+ cytotoxic T lymphocytes. Upon re-exposure to the antigen, memory T cells become reactivated and release cytokines like interferon-gamma (IFN-γ) and tumor necrosis factor-alpha (TNF-α), which recruit and activate macrophages, leading to tissue injury. This reaction usually appears 24–72 hours after antigen exposure and is termed delayed-type hypersensitivity (DTH). Examples include contact dermatitis (e.g., due to nickel or latex), tuberculin skin test, and chronic transplant rejection (Marrack et al., 2010; Abbas et al., 2022)[12,10].

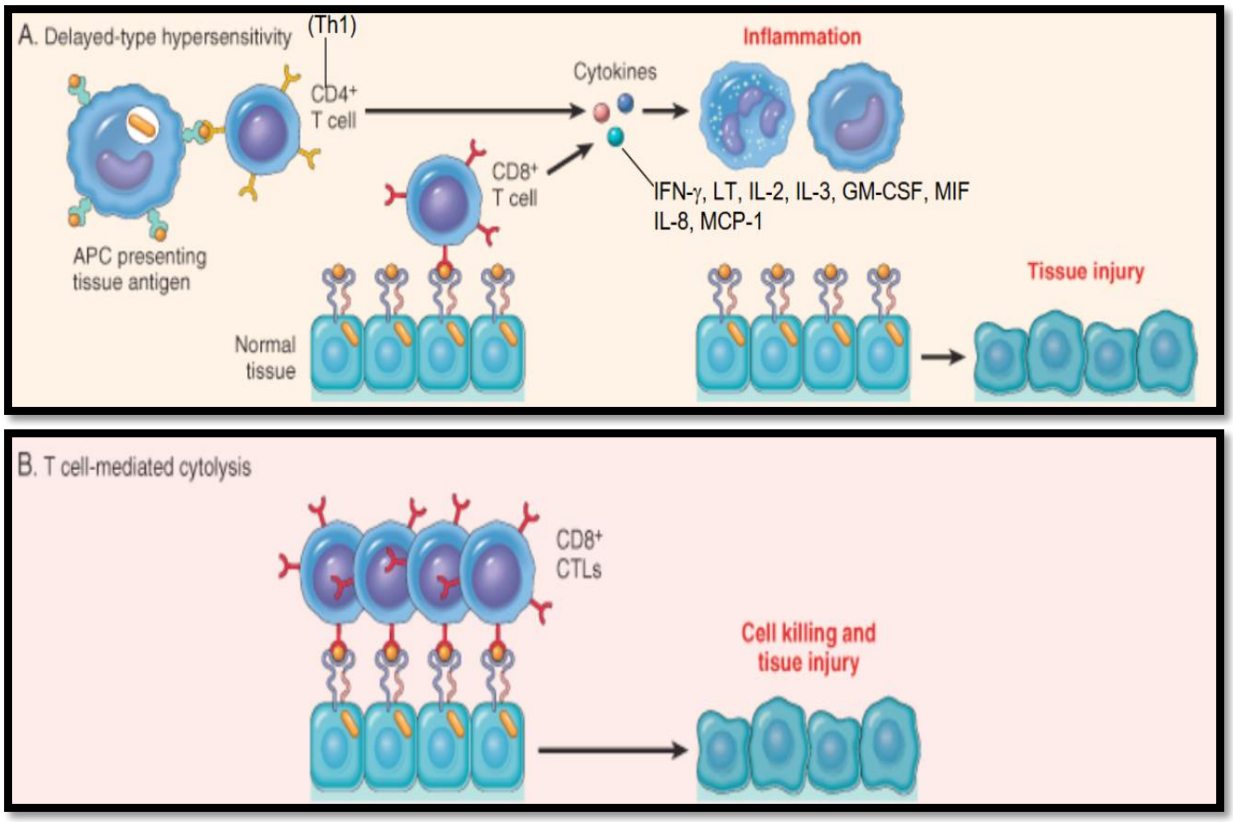


**Figure- 4: Immune-Complex Reactions**

**Importance of Examination for Allergy/Hypersensitivity**

A thorough history and diagnostic work-up—such as **patch testing**, **serum-specific IgE assays**, and **skin prick tests**—are essential for identifying hypersensitivities before initiating orthodontic or dental treatment, particularly in patients with suspected metal or latex allergies.[13,14,15]

The prevalence of metal allergy is high in the general population, and it is estimated that up to 17% of women and 3% of men are allergic to nickel.[14]

**Mechanisms of FcεRI-Mediated Mast Cell Activation in Anaphylaxis**

**FcεRI binds IgE on mast cells:**

The high-affinity IgE receptor, FcεRI, is predominantly expressed on the surface of mast cells and basophils. This receptor binds to the Fc region of immunoglobulin E (IgE) antibodies, sensitizing the cells to specific allergens.[16]

**Allergen re-exposure leads to cross-linking:**

Upon subsequent exposure to the same allergen, the allergen cross-links the bound IgE on the surface of sensitized mast cells. This cross-linking triggers the activation of FcεRI, leading to a cascade of intracellular signaling events, including activation of protein kinases and phospholipase enzymes.[17]

**Mast cell degranulation and mediator release:**

Activation of FcεRI results in mast cell degranulation, a process where preformed mediators such as histamine, proteases, and chemokines are rapidly released from cytoplasmic granules. Additionally, activated mast cells synthesize and secrete newly formed mediators, including prostaglandins, leukotrienes, and cytokines.[18]

**Clinical manifestations of anaphylaxis:**

The release of these mediators leads to the characteristic symptoms of anaphylaxis, such as vasodilation, increased vascular permeability, smooth muscle contraction, and mucous secretion. Clinically,

this manifests as hypotension, bronchoconstriction, urticaria, angioedema, and, in severe cases, anaphylactic shock.[9]

Nickel Allergy in Orthodontics[19,20,21,22]

Nickel allergy is a delayed-type (Type IV) hypersensitivity reaction triggered by exposure to nickel-containing orthodontic appliances such as brackets, wires, or bands. It results from the release of nickel ions that come in contact with the oral mucosa, causing a localized or systemic allergic reaction.

**Table 2: Clinical Features, Signs and Symptoms**

| Category | Details |
|---|---|
| Onset | Usually within a few days to weeks after appliance placement. |
| Oral Mucosa Reaction | • Erythema (redness)<br>• Edema (swelling)<br>• Burning sensation |
| Extraoral Signs | • Perioral dermatitis<br>• Facial eczema (in severe cases) |
| Gingival Symptoms | • Gingivitis-like symptoms localized around brackets and bands |
| Other Symptoms | • Itching<br>• Pain<br>• Oral ulcers in contact areas |
| Common Sites | Buccal mucosa, inner lips, and gingiva in contact with metal appliances |
| Severe Reaction | • Lichenoid lesions<br>• Chronic inflammation<br>• Hyperplastic tissue |

**Diagnosis & Management**

| Aspect | Details |
|---|---|
| Diagnosis | • Clinical history<br>• Patch testing (Dermatologist)<br>• Elimination and rechallenge method |
| Management | • Use of nickel-free appliances (e.g., titanium brackets, Teflon-coated wires)<br>• Topical corticosteroids for symptom relief<br>• Close monitoring |

**Figure-5: Typical arrangement of patch test units in adults**

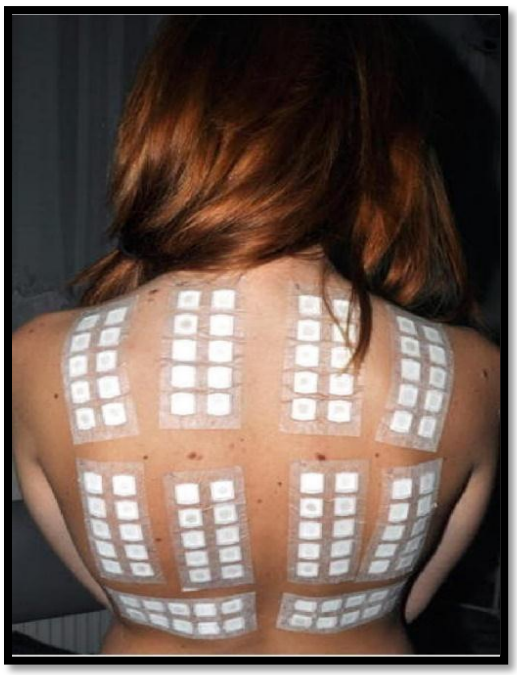

**Figure-6: Transverse placement of test units may prove more practical in small children**

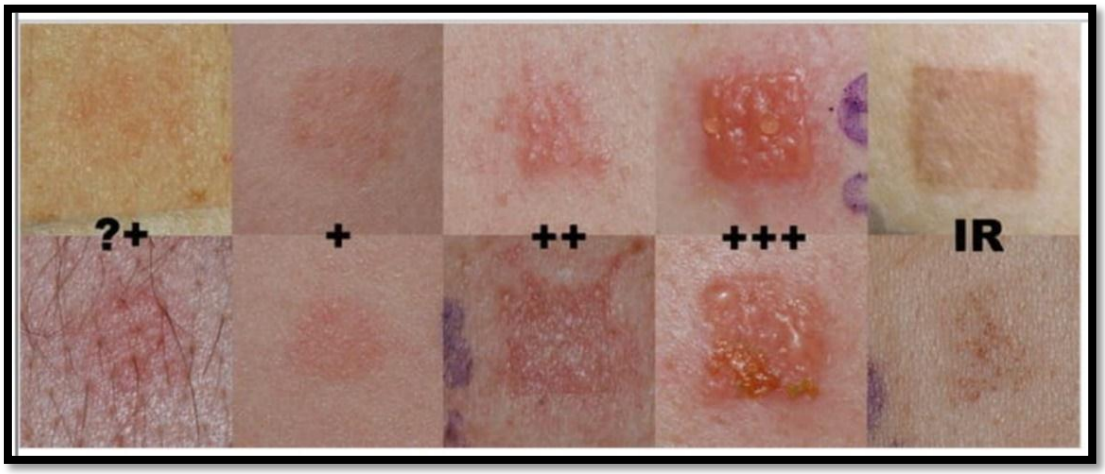


**Figure-7: The practical CODEX system for assigning relevance to positive allergic reactions**

**Table 3: Patch Test Notation Descriptions and Interpretations**

| Notation | Description | Interpretation and Comments |
|---|---|---|
| - (Negative) | No visible reaction in the tested area | All haptens with negative test results should be listed in the result form |
| ?+ | Faint, non-palpable erythema | Doubtful reaction |
| + | Palpable erythema; moderate oedema or infiltrate; papules not present or scarce; vesicles not present | Weak reaction |
| ++ | Strong infiltrate; numerous papules; vesicles present | Strong reaction |
| +++ | Coalescing vesicles, pseudo-bullae, or ulceration | Extreme reaction |
| IR (Irritant Reaction) | Limited to the exposed area; lack of infiltrate (oedema may be present); common reaction with homogeneous erythema without infiltration; poral reaction with papular or punctate | This type of reaction may cause relevant problems upon interpretation |

| | | |
|---|---|---|
| | erythema; sometimes haemorrhagic or pustular reaction; with one or numerous pustules; possibly other efflorescences beyond papules or vesicles | |
| AQ2 | With one or numerous pustules; possibly efflorescences other than papules and vesicles | Needs clarification; possibly related to IR-type reactions |
| NT (Not Tested) | Test substance not tested | Must be clearly marked if unavailable or skipped even if listed in the standard form |

**Preventive strategies**[30]

1. Avoidance of nickel

The only way to prevent recurrence is avoiding skin contact with metallic items that release nickel. It has been documented that this strategy results in a statistically significant decrease in the frequency of hand eczema in nickel-sensitive individuals. Although metallic items that could not be avoided are often covered with enamel, dye or adhesive this procedure may carry the risk of inducing sensitization to these compounds.

2. Antiperspirants

The use of antiperspirants in order to decrease sweating can sometimes prevent nickel ACD, as sweating induces releaseof nickel ions from metallic items.

3. Smoking

Heavy smoking is a risk factor for nickel allergy, as the metal is found in tobacco with an average content of 1 to3 mg per cigarette.

**Symptomatic treatments**[30]

Since pruritus is an important complaint sedating oral antihistamines might be indicated. Topical antihistamines on the other hand must be discouraged, as they are possible sensitizers. Oral doxepin (10–25 mg at night in adults) can be considered if other oral antihistamines are not helpful. Acute exudative or bullous lesions can be treated with cool antiseptics compresses, three times a day, and topical steroids. Topical or oral antibiotics must be prescribed in case of secondary bacterial infection. Emollients in creams are useful to relief itching and dry skin.

**Steroids -** Topical steroids are very useful and represent the first-line treatment. Potency should be chosen according to the body sites, as low potency steroids are recommended for face andflexural areas and high potency agents might be used for othersites as palms and soles. Oral steroids act as immunosuppressive agents and might be indicated for short-term treatment of severe dermatitis. In adults' prednisone in a single morning dose of 40 to 60 mg can be prescribed and tapered over 2 to 3 weeks, as symptoms resolve.

**Calcineurin inhibitors -** Calcineurin inhibitors are currently approved for the treatment of atopic dermatitis but not ACD. Advantages over topical corticosteroids include that they do not cause cutaneous atrophy or glaucoma or cataracts when applied near the eye. Pimecrolimus cream might be used for the face and tacrolimus 0.1% ointment can be used for ACD of Allergy Caused by Nickel.

**Psoralen plus UV-A –** Some patients with chronic ACD can benefit for PUVA. Kalimo treated with PUVA 5 female patients with long- standing hand dermatitis with complete resolution after 1 year. However, sensitivity of blood lymphocytes to nickel after treatment was approximately the same or increased which provides no evidence to indicate that systemic, nickel- specific suppressive immune regulative mechanisms would have been activated by the treatment.

**Disulfiram –** It has been shown that disulfiram can chelate nickel, interfere with its absorption and metabolism and then improve nickel contact dermatitis, particularly pompholyx.85 this agent can be considered only in nickel-sensitized patients with severe hand involvement refractory to all other treatments as it can cause severe side effects including liver toxicity. It is also important to inform the patients that they cannot drink alcohol during treatment. Adult dose is 500 mg PO qd and 125 to 500 mg PO qd for maintenance.

**Binding agents and barrier creams –** It is known that some topical and oral substances can chemically bind nickel and prevent nickel ACD. These substances promote chelation of nickel, and thus prevent its antigenic properties; they are usually used in combination with others treatments, such as topical steroids. Barrier creams act as an ―invisible glovel, protecting the skin from environmental allergens.

The most utilized binding agent is ethylene diamine tetra-acetic acid (ETDA), which can be included at a 15% concentration in a cream in association with topical steroids. Memon showed that a cream containing 15% ETDA and 1% hydrocortisone was able to reduce the allergic reactions to patch tests with 20 pence coins (16% NI, 84% Cu) in 10 of 26 nickelsensitive subjects challenged for 2 days. Another binding agent is 5-chloro-7-iodoquinolin-8-ol (clioquinol), which was able of prevent allergic reaction in 2 days, in a cream containing clioquinol 3% and hydrocortisone 1%. Clioquinol is commercially available in association with hydrocortisone or methasone.

**Low-nickel diet -** Food is important source of nickel and daily ingestion depends both on the type of food and on the production environment. Foods with high nickel content include whole- grain flour, oats, soybeans, legumes, shellfish, nuts, licorice and chocolate. The efficacy of prescribing a diet is still controversial as the daily oral uptake from food is much lower than the doses utilized to produce symptoms in experimental studies. However, it has been shown that some patients might benefit from a nickel free or a low nickel diet. Dietary restriction must be prescribed according to **Veien's** guidelines. Patients should be followed for 1 to 2 months to evaluate outcome before deciding if dietary restrictions should be maintained or not.

**Alitretinoin (9-cis retinoic acid) -** This oral retinoid has been described as a promising new option in treatment of chronic, severe, and refractory hand dermatitis. This substance is a pan-agonist that binds to retinoic acid receptors A (RAR) and X (RXR), acting as anti-inflammatory and immunomodulator.

Chromium Allergy in Orthodontics[19,20,21,23]

Chromium allergy is a delayed-type (Type IV) hypersensitivity reaction caused by exposure to chromium-containing orthodontic alloys, such as stainless steel. Chromium ions released from these materials can cause allergic contact dermatitis and oral mucosal reactions, particularly in sensitized individuals.

**Table 4: Clinical Features, Signs and Symptoms**

| Category | Details |
|---|---|
| Onset | Typically appears days to weeks after contact with chromium-containing orthodontic appliances. |
| Oral Mucosa Reaction | • Redness (erythema)<br>• Swelling (edema)<br>• Burning or tingling sensation |
| Extraoral Signs | • Perioral dermatitis<br>• Contact eczema |
| Gingival Symptoms | • Localized gingival inflammation near bands, brackets, or wires |
| Other Symptoms | • Oral discomfort<br>• Tingling<br>• Mild ulceration |
| Common Sites | Inner cheeks, lips, and gingiva exposed to orthodontic components |
| Severe Reaction | • Persistent contact dermatitis<br>• Lichenoid reactions<br>• Painful ulcerations |

**Diagnosis & Management**

| Aspect | Details |
|---|---|
| Diagnosis | - History of allergic reactions to metal<br>- Patch testing for chromium sensitivity<br>- Clinical correlation |
| Management | - Use of chromium-free appliances (e.g., titanium-based, Teflon coated (Tooth-colored epoxy resin) wires, Optifelx archwires, Fiber reinforced composite archwires, Beta III Titanium, CNA Beta –Titanium and TMA wires (Toms, 1988) –<br>- Use of metal ligatures instead of orings or elastics, use of self-ligating brackets<br>- Patients with more intense reactions should be treated with antihistamines, anesthetics.<br>- Symptomatic relief with topical corticosteroids<br>- Monitoring and substitution of appliances if needed |

**Latex Allergy in Orthodontics[19,20,24,25]**

Latex allergy is an immediate-type (Type I) hypersensitivity reaction caused by exposure to natural rubber latex, commonly found in orthodontic products such as elastic bands, gloves, separators, and some ligatures. It can lead to serious allergic responses, particularly in sensitized individuals, ranging from localized itching to anaphylaxis.

**Table 5: Clinical Features, Signs and Symptoms**

| Category | Details |
|---|---|
| Onset | Usually immediate (within minutes) but can be delayed up to a few hours. |
| Oral Mucosa Reaction | • Swelling<br>• Itching |

| | |
|---|---|
| | • Burning sensation<br>• Ulceration |
| Extraoral Signs | • Perioral itching or swelling<br>• Rash or urticaria (hives)<br>• Eczema |
| Gingival Symptoms | • Inflamed gingiva near elastic contacts<br>• Localized discomfort |
| Other Symptoms | • Sneezing<br>• Rhinitis<br>• Watery eyes<br>• Difficulty breathing in severe cases |
| Common Sites | Lips, oral mucosa, gingiva in contact with latex-containing items |
| Severe Reaction | • Anaphylaxis<br>• Bronchospasm<br>• Respiratory distress<br>• Cardiovascular collapse |

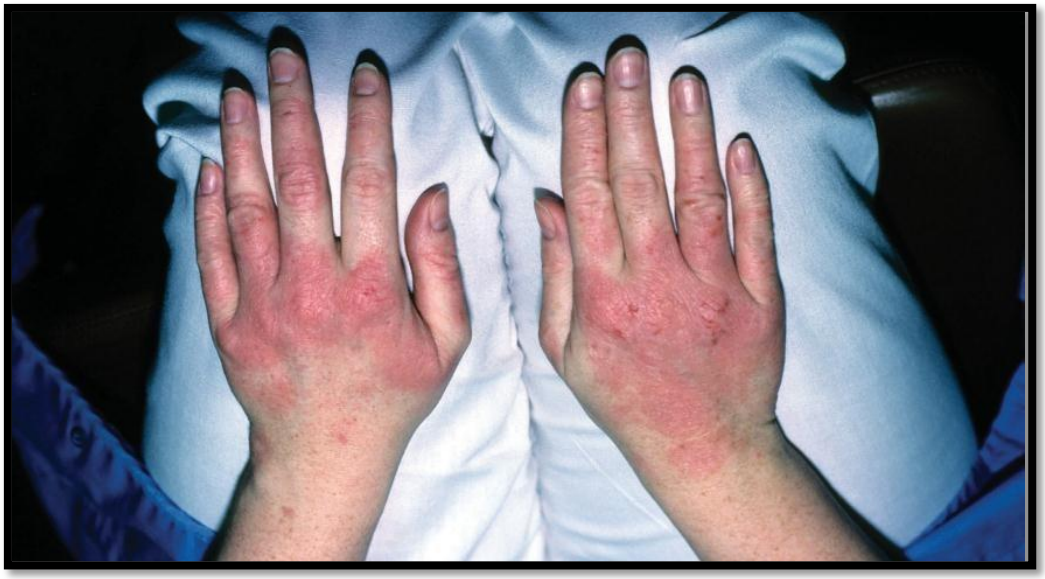

**Figure- 8: Contact Dermatitis (Hand Eczema) due to Latex Glove-induced Type IV Hypersensitivity Reaction**

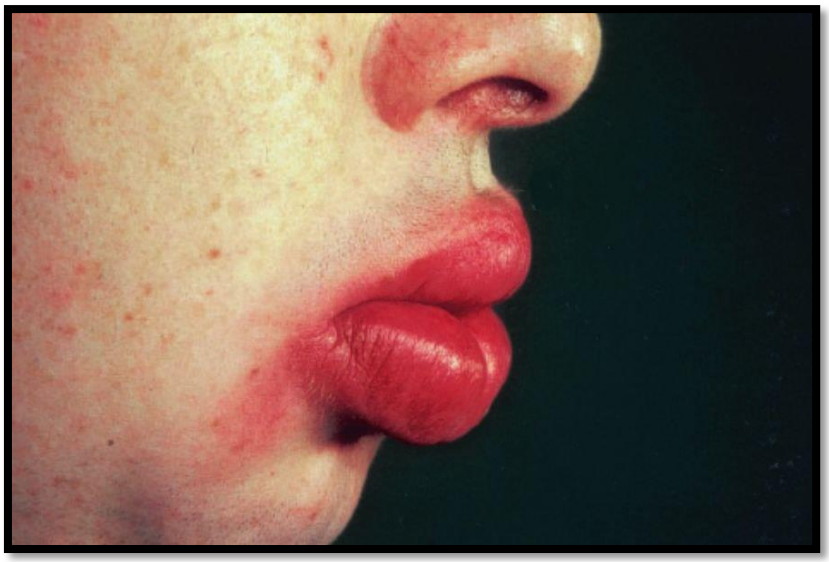

**Figure- 9: Angioedema of the Lips and perioral region Due to Type I Hypersensitivity Reaction from Latex Exposure.**

**Table 6: Diagnosis & Management**

| Aspect | Details |
|---|---|
| Diagnosis | - Detailed allergy history<br>- Skin prick test or blood test for latex-specific IgE, Intradermal test<br>- Observation of clinical symptoms |
| Management | - Avoidance of latex-containing materials<br>- Use of non-latex alternatives (e.g., silicone, vinyl, nitrile)<br>- Emergency preparedness for anaphylaxis (e.g., epinephrine auto-injector) |

**Acrylic Allergy In Orthodontics[20,26,27]**

Acrylic allergy is a hypersensitivity reaction to methyl methacrylate (MMA) or residual monomers found in acrylic resins commonly used in orthodontic appliances. It most frequently occurs due to incomplete polymerization of the material or repeated contact with unpolymerized acrylic dust or vapors.

**Table 7: Clinical Features, Diagnosis, and Management**

| Aspect | Details |
|---|---|
| Allergen | Methyl methacrylate (MMA), other acrylic monomers used in cold-cure and heat-cure acrylic resins |
| Common Sources | Orthodontic retainers, habit-breaking appliances, bite blocks, and other removable appliances |
| Type of Reaction | Type IV Delayed Hypersensitivity (cell-mediated) |
| Signs & Symptoms | - Oral mucosal burning, redness, swelling<br>- Vesicles or ulcers at contact points<br>- Contact dermatitis (skin) |
| Diagnosis | - Clinical history and symptom correlation<br>- Patch test for methyl methacrylate allergy |
| Management | - Remove appliance and replace with hypoallergenic material<br>- Cover the prosthesis with light polymerized methyl methacrylate, with ultraviolet polymerized urethane acrylate, with ultraviolet polymerized methacrylate, - use a polycarbonate prosthesis, vulcanite, or titanium associated to ceramic brackets.<br>- Instead of methacrylate resin Clear aligner can be used as a retainer to avoid the allergic reactions<br>- Use completely polymerized acrylic<br>- Topical corticosteroids (if needed) |
| Prevention | - Use proper curing protocols<br>- Avoid monomer exposure |
| Special Consideration | Dental professionals are also at risk of developing hand dermatitis due to repeated handling of uncured acrylic |

**Resin and Adhesive Allergy in Orthodontics[20,24,25]**

Resin and adhesive allergy in orthodontics is a hypersensitivity reaction caused by exposure to epoxy resins, Bisphenol A glycidyl methacrylate (Bis-GMA), urethane dimethacrylate (UDMA), TEGDMA, HEMA, and other acrylate-based components used in bonding agents and composites. Most reactions are Type IV delayed hypersensitivity reactions, often triggered by handling uncured materials or inadequate polymerization during clinical procedures.

**Table 8: Clinical Features, Diagnosis, and Management**

| Aspect | Details |
|---|---|
| Allergen | Epoxy resins, Bis-GMA, UDMA, TEGDMA, HEMA |
| Common Sources | Composite adhesives, bonding agents, sealants, and light-cured resin materials |
| Type of Reaction | Type IV Delayed Hypersensitivity Reaction |
| Mode of Exposure | Direct contact with uncured/partially cured resin during bonding procedures |
| Predisposing Factors | - Incomplete curing of resins<br>- Handling uncured adhesives<br>- History of allergic dermatitis |
| Signs & Symptoms | - Oral mucosal burning, redness, itching<br>- Vesicles or ulcers at contact sites<br>- Dermatitis on hands of clinicians |

| | |
|---|---|
| | - Eye irritation (rare) |
| Diagnosis | - Clinical correlation with exposure<br>- Patch test for acrylate resin sensitivity |
| Management | - Use fully cured, BPA-free or biocompatible resins<br>- No-touch bonding techniques<br>- Personal protective equipment (gloves, eyewear)<br>- Topical corticosteroids if needed |
| Prevention | - Use proper polymerization protocols<br>- Substitute with non-allergenic materials if required |

## CONCLUSION

Allergic reactions are host immune responses to endogenous or exogenous antigens, which can result in local and systemic problems. Among the main allergens are the dental materials used in orthodontics, which faces some challenges with regard to biocompatibility with oral tissues is possible to conclude that reactions such as erythema, edema, papules, blisters and periodontal changes, for example gingival inflammation, are frequent manifestations of contact with orthodontic materials in patients allergic to nickel or, in some cases, chromium. A stronger body of research is needed to clarify the relationship between metal allergy and reactions to implanted metal devices. Once a clear understanding of this relationship is defined, if it exists, appropriate guidelines can be drafted in the attempt to clarify management of or completely avoid allergic reactions to metal implants.

- Reactions to metal orthopaedic implants do occur, though rarely, even in those with metal allergy.
- Routine pre-implant testing in asymptomatic individuals is not indicated.
- Listen to patients concerns about metal allergy if the concern arises.
- Patch testing is probably the best pre- and post-implant screening test.
- Post-implantation testing is controversial and even positive LTT or patch test does not definitively diagnose morbidity from a metal allergy. Complete recovery following revision with an immunologically inert device is diagnostic.

Therefore, prior knowledge of the dentist is essential for the correct management and treatment of these adverse reactions. Safe and effective practice depends on identifying patients with allergy along with knowledge of materials that can potentially cause them.

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