Hypersensitivity to Dental Cast Metals: A Clinical Study

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Abstract: Concerns over allergic reactions to dental restorative and orthodontic materials have increased in the past few years. The incidence appears to be quite low, but increases with increased quantity and duration of exposure to a causative antigen. In most instances intraoral allergies result in a type IV, delayed contact response that may manifest in various ways including lichenoid reactions, burning mouth/burning lip syndromes, cheilitis and lip swelling, oral granulomatous reactions, gingival hyperplasia, non-specific erythema and edema, ulceration and gingivitis or even periodontitis. In this paper, hypersensitivity reactions to dental metals are reviewed and allergy to dental cast metal alloys is studied in a group of patients presenting with oral features sometimes associated with contact hypersensitivity reactions. Of 438 patients evaluated, 18 (4.5%) were determined to have mucosal lesions confirmed by clinical appearance, contact with causative restorations, patch testing and biopsy.

Keywords: Hypersensitivity, dental metallic alloys, contact reactions, patch testing, biopsy, allergy related periodontitis.

BACKGROUND

Concerns over biocompatibility and hypersensitivity response to dental restorative materials and orthodontic appliances have increased markedly world-wide in the past few years [1-9]. It is not clear whether this interest is due to an increased frequency in adverse events regarding dental materials or due to an increased awareness of and recognition of oral hypersensitivity reactions. At present, the true incidence of hypersensitivity response to dental materials is unknown. When one considers the millions of dental restorations placed annually and the number of published reports it appears the incidence is quite low. Several studies have attempted to determine the incidence in various populations but geographic population variations, differences in inclusion and exclusion criteria, differences in study methodology and differences in assessment of results make it virtually impossible to determine the true incidence [1, 2, 4, 5, 7-12]. However, hypersensitivity reactions to dental materials clearly increase with increased exposure and the worldwide increase in body piercing, tattooing and the wearing of jewelry in both sexes potentially enhances the likelihood of exposure to metallic and non-metallic materials used in dental restorations and orthodontic appliances [13,14].

Type I anaphylactoid reactions to dental materials have been reported usually manifesting as eczema. However, in the vast majority of incidences oral hypersensitivity reactions are a type IV delayed contact dermatitis/stomatitis most often affecting those oral sites in direct contact with the allergen [13].

The oral cavity is less likely to manifest a contact allergic reaction than skin. This may be due to the flushing and buffering effect of saliva, to the increased vascularity of oral mucosa compared to skin and possibly to the lower number of Langerhans cells and t lymphocytes found in mucosa [3]. It has been estimated that expression of a contact reaction in the oral cavity requires 5-12 times the antigen exposure than required on skin [15].

Hypersensitivity reactions to dental materials require the release of allergenic antigens from the material. Release of metal ions occurs due to corrosion, the enzymatic activity of saliva, a more acidic oral pH, and other factors. These factors have less effect on non-metallic dental restorative materials. However materials such as epoxy, acrylates and resins have been found to contain more than 40 known allergens and to cause hypersensitivity reactions in addition to chemical irritation (Fig. 1). These allergens are released in large amounts during the early phase of the curing process. Complete polymerization may take days to weeks after insertion and contact reactions may be noted. However, the reaction may be self limiting because allergen release from the material diminishes over time as final complete polymerization takes place [16].

There is no single oral presentation of hypersensitivity reactions and a variety of clinical signs and symptoms have been reported. It is often difficult to validate these signs and symptoms as indicative of an allergic reaction, thereby complicating the diagnosis of the reaction. The literature describes contact lichenoid reactions resulting in tissue changes basically identical to the idiopathic form of oral lichen planus [2, 3, 17] (Fig. 2). Other reports associate oral hypersensitivity with burning mouth/burning lip syndromes [16,18] cheilitis and lip swelling [13,19], oral granulomatous reactions [20,21], gingival hyperplasia [22], non-specific erythema and edema, ulceration and gingivitis or even periodontitis [23]. In most instances, however, contact reactions are believed to occur more extensively and perhaps exclusively in tissue that is in direct contact with the suspected allergen [13].
Fig. (1). Hypersensitivity reaction to non-metallic temporary crowns. (a) gingival erythema. (b) labial erythema and ulceration.

Single metals and metal alloys in dental restorations include a wide variety of substances. Some may be classified as noble metals (gold, platinum, palladium, iridium ruthenium, rhodium) while others are considered base metals (silver, copper, zinc, indium, tin, gallium chromium, cobalt, molybdenum, aluminum, iron, beryllium, titanium, nickel, vanadium, niobium, zirconium). Note that silver is a noble element but the American Dental Association (ADA) has classified it as a base metal because of its relatively high ionic release and reactivity in the oral cavity [24]. ADA metal alloy specifications identify:

- High noble alloys- noble metal ≥60wt% plus gold ≤40 wt%
- Noble alloys- Noble content ≥25 wt %
- Base alloys- Noble content <25 wt%

High noble alloys are considered to be more resistant to corrosion and release of metal ions or metallic microparticles. Noble alloys are less resistant and base alloys are most likely to release metallic salts. However, studies have confirmed that all metals are capable of releasing metallic ions in the oral environment and the majority of ions reside in soft or hard tissue that directly contacts the restoration or appliance [25]. The likelihood of a contact hypersensitivity reaction to a specific metal diminishes

Fig. (2). Contact lichenoid reaction to dental amalgam. Lesions had been present for 13 years. (a) Tongue lesion. (b) Buccal view. (c) Resolution of tongue lesion 3 weeks after replacement of amalgam with a non-metallic restoration. (d) Resolution of buccal lesion.
proportional to the quantity of a potential allergen found in the material. Thus trace elements such as molybdenum, beryllium, gallium, rhodium, and iridium rarely have been identified as causative of a oral allergic reaction [24]. Conversely, hypersensitivity reactions to nickel, mercury, gold, palladium, cobalt and platinum are more common [12]. Palladium is cross reactive with nickel although a mono-reaction to palladium is rare [2, 26].

Titanium is considered by some to be non-allergenic [27, 28] but reactions have been reported and titanium ions are found in large quantity in contacting tissues and often throughout the body. A hypersensitivity reaction to titanium may have devastating results since the material is used in implanted devices such as pacemakers, automatic implanted cardioversion devices (AICDs), joint replacements and dental implants [29, 30] (Fig. 3).

Hypersensitivity reaction to nickel containing dental alloys has been a matter of special concern in orthodontics because orthodontic appliances (brackets, bands, wires, etc.) may contain nickel. Stainless steel is composed of approximately 8% nickel while nickel-titanium alloys contain from 50%-70% nickel [15, 23, 31]. Despite this, some authorities have reported that nickel ionic release from orthodontic appliances is low and does not constitute a risk for inducing hypersensitivity [4, 11, 23]. Others, however, have offered evidence that clinical manifestations of nickel contact stomatitis or gingival hyperplasia do occur, possibly due to metal corrosion resulting from oral environmental factors such as warm oral temperatures, salivary enzymatic components, electrochemical (galvanic) currents, microbial composition, trauma, wear etc [5, 22, 31-34] (Fig. 4).

A previous history of allergy to metals or other substances is a possible predictive factor in oral contact hypersensitivity but not all patients who manifest a positive epicutaneous patch test reaction to a metal will experience an oral reaction to that metal even if found in dental restorations or appliances. On some occasions patients with a positive patch test reaction for a dental metal will never experience a contact allergic reaction while others may remain free of signs and symptoms for years then develop contact reactions if the oral environment changes, as in the onset of xerostomia or if additional restorations are place that contain the same allergen. Consequently there appears to be little value in performing patch tests prior to placement of a particular metallic or non-metallic material in most patients. However, patients who are atopic and inclined to allergies and those who have previously demonstrated an allergenic reaction to jewelry, tattooing, body piercing, etc., may benefit from patch testing before initiation of dental treatment in an effort to identify materials likely to induce an adverse reaction.

Patch testing is considered by many to be the “gold standard” for contact allergy testing although its use continues to be controversial because it may yield false negative or false positive results. For example a weak (macular erythematous) response to a particular metallic salt may represent an irritant effect rather than a true hypersensitivity reaction. At present it is not possible to predictably differentiate between these two outcomes. Patch testing for some materials such as titanium may not be totally accurate and positive results may be found only in 50% of less of patients believed to be experiencing a contact hypersensitivity reaction. Consequently patch testing may primarily be of value only if a clinically relevant lesion is present. It may also be important to obtain biopsy confirmation of the nature of the lesion. In many instances it may be possible to confirm the diagnosis only by combining patch testing with biopsy and examination of the clinical lesion [16, 24, 25, 35, 36].

Fig. (3). Allergic reaction to commercially pure titanium implants. Implants had been in place for 4 months. (a) Clinical view. (b) Radiographic view. Patient proved to have secondary osteomyelitis and large sinus perforation.
The process of patch testing is not difficult for a trained individual so long as one adheres to a precisely defined method. Several agencies have offered standardized guidelines for performing the test and the tester should be familiar with the guidelines and compliant with them. Interpretation of results is subjective and consequently should probably only be undertaken by an allergist, dermatologist or other individuals well trained in the diagnostic process.

METHODS AND MATERIALS

Sequential patients presenting to the Stomatology Center, Baylor College of Dentistry were screened to identify those who had one or more cast dental restorations and signs or symptoms suggestive of a diagnosis of one or more conditions documented by studies as being sometimes associated with a hypersensitivity reaction to dental cast metal restorations or appliances.

The following patients were selected:

Oral lichen planus 178
Burning mouth syndrome, burning lips, localized burning 125
Allergic stomatitis/mucositis 54
Unexplained leukoplakia 37
Restoration related atypical gingivitis/aggressive periodontitis 44

Total Patients evaluated 438

With the exception of patients with burning mouth syndrome and those with periodontitis, biopsies were performed to confirm the tentative clinical diagnosis.

Those with biopsy confirmed diseases or abnormalities located in tissues that were in direct contact with cast dental restorations or appliances were patch tested for hypersensitivity to cast metals commonly found in cast dental alloys (Table 1).

Table 1. Dental Metals Patch Test Materials Stomatology Center, Baylor College of Dentistry

| 1. Ammoniated mercury 1% in petrolatum |
| 2. Sodium thiosulfatoaurate 0.25% in petrolatum |
| 3. Postassium dicyanoaurate 0.002% in water |
| 4. Palladium chloride 1% in petrolatum |
| 5. Ammonium tetrachloroplatinate 0.25% in water |
| 6. Amalgam 5% in petrolatum |
| 7. Cobalt chloride hexahydrate 1% in petrolatum |
| 8. Nickel sulphate hexahydrate 5% in petrolatum |
| 9. Tin chloride 0.5% in petrolatum |
| 10. Potassium dichromate 0.5% in petrolatum |
| 11. White petrolatum 100% (control) |
| 12. Copper sulfate 1% in water |
| 13. Silver nitrate 1% in water |
| 14. Copper Oxide 5.0% in petrolatum |
| 15. Mercury ammoniumchloride 0.1% in petrolatum |
| 16. Mercuric chloride 0.1% in petrolatum |
| 17. Mercury 0.5% in petrolatum |
| 18. Zinc chloride 2.5% in petrolatum |
| 19. Goldsodiumthiosulfate 0.5% in petrolatum |

Fig. (4). Hyperplastic gingivitis induced by a stainless-steel orthodontic appliance. Appliance had been removed a few hours prior to photograph. Patient was patch test positive to nickel. (a) Facial view. (b) Palatal view. (c) Facial view showing remission 5 months following appliance removal. (d) Palatal view also showing remission.
Patch testing was performed as recommended by the North American Contact Dermatitis Group guidelines using test materials obtained from Dormer Laboratories, Rexford Ontario and Trolab via Omniderm Pharma Canada Inc, Quebec. Metallic salts were applied to each patient’s back using Finn chambers mounted on Scanpor hypoallergenic tape (Allerderm Labs Inc, Petaluma CA). Patients were instructed not to wash the test area for two days. At 48 hours the materials were removed and the back was examined for evidence of a positive reaction. The patient was evaluated again after 72 hours for a delayed reaction.

Grading was performed using the diagnostic criteria recommended by the North American Contact Dermatitis Group as follows:
1. Negative/doubtful- 0
2. Weak positive- macular erythema (graded as +)
3. Strong positive- edematous erythematous or vesicular (graded as ++)
4. Extreme- Spreading, bullous, ulcerative (graded as +++)

Gradings 2-4 were identified as positive findings.

RESULTS

The following diseases were confirmed as probably related to hypersensitivity reactions:
Localized atypical gingivitis/localized aggressive Periodontitis (Figs. 5-8) - 9
Contact localized lichenoid reaction (Fig. 9) -5
Allergic localized mucositis/glossitis (Fig. 10) -3
Burning mouth (localized) -1

Total -18 (4.8%)

DISCUSSION

It has been suggested that numerous oral signs and symptoms occur as a result of hypersensitivity reactions. These include burning lips, burning mouth syndrome, localized burning, cheilitis, angular cheilitis, erythema, desquamation, ulceration, contact lichenoid reaction, glossitis, generalized stomatitis, xerostomia, altered taste sensation and others. Eczema and systemic manifestations have also been described. However the concept for this study was to only evaluate those oral conditions that have been repeatedly confirmed or suggested by the majority of studies on the topic. It has been our experience in the Stomatology Center, Baylor College of Dentistry that hypersensitivity reactions to dental hygiene products, flavoring (especially cinnamic aldehyde), preservatives and foods are almost universally involved when generalized lesions develop such as stomatitis, generalized oral lichen planus, lip edema, perioral inflammation, and generalized gingival erythema or glossitis. We were unable to find convincing evidence in the scientific literature to associate xerostomia, altered taste sensation, and random systemic symptoms with a hypersensitivity reaction to cast dental restorations. Consequently we elected to confine our evaluation to lesions that were in direct contact with the suspected cast dental restorations.
Fig. (6). Hyperplastic gingivitis induced by hypersensitivity to gold, chromium and cobalt. Patient was 23 years old and crowns were placed for esthetic reasons. (a) Maxillary Facial View. (b) Mandibular Facial View. (c) Maxillary radiograph showing excessive loss of alveolar height. (d) Mandibular radiograph also showing evidence of periodontal destruction. (e) Facial view 6 months after removal of crowns containing nickel, scaling and root planning and placement of temporary crowns.

Fig. (7). Atypical gingivitis and possible periodontitis in 21 year old female following placement of noble alloy crowns containing palladium and nickel. (a) Clinical view. Note localized gingivitis involving all metal containing crowns but not involving central incisors with porcelain jacket crowns. (b) Radiograph confirming presence of metal alloy crowns.
Fig. (8). Severe aggressive periodontitis in a 28 year old female who was allergic to nickel. Patient had full mouth reconstruction with exception of mandibular anterior teeth. (a) Facial view one year after placement of porcelain fused to alloy metal crowns. (b) Palatal view of same patient. (c) Radiograph of maxillary right anterior immediately prior to initiation of restorative treatment. (d) Radiographic view of maxillary left anterior prior to restorative treatment. (e) Right bitewing radiographic view prior to restorative treatment. (f) Left bitewing radiographic view prior to restorative treatment. (g) Radiograph of maxillary right anterior exactly one year following restorative treatment. (h) Radiograph of maxillary left anterior exactly one year following restorative treatment. (i) Radiograph of mandibular right posterior one year following restorative treatment. (j) Bitewing of mandibular left posterior one year following restorative treatment. (k) Lingual view of mandibular anteriors one year following treatment. This was the only unrestored area in entire dention. (l) Radiograph of the mandibular anterior teeth one year following restorative treatment. Note height of alveolar bone.
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By confining our selection process to localized lesions that contacted the suspected metallic restoration we believe that we could better assure that the diseases or disorders studied did represent a true hypersensitivity reaction and we felt that biopsy confirmation of the clinically visible lesions added to the validity of this conclusion. Complaints of burning discomfort are fairly common in individuals experiencing an oral contact hypersensitivity reaction and we in our study only one patient described burning of tissues that contacted a suspected dental restoration without visible signs of inflammation. This patient complained of localized burning of the gingiva, buccal mucosa and tongue in areas that contacted the restoration and we felt that this was a true symptom of a mild hypersensitivity reaction. This thought was supported by the finding of only a weak (+) reaction to gold salts on patch testing. Although it has been noted that a weak patch test results cannot distinguish between a hypersensitivity reaction and an irritant effect, we felt that the excellent marginal integrity, contour and surface smoothness of the restoration made it unlikely that this was an irritant reaction.

Contact lichenoid reactions to dental restorations (Fig. 10) have frequently been identified in the literature although they seem to be more often associated with dental amalgam restorations than cast restorations. Among the cast metal alloys, nickel appears most likely to elicit a lichenoid response as was the case with 4 of the 5 patients who presented with localized lichenoid lesions. Several authorities have suggested that lichen planus rarely if ever occurs as only a single isolated lesion indicating that such a finding should raise suspicion of a localized hypersensitive reaction.

Fig. (9). Contact lichenoid reaction to hypersensitivity reaction to metallic alloy. Patient was allergic to cobalt.

Fig. (10). Non-specific mucositis left buccal mucosa contacting “high noble” gold cast restoration. Patient had been treated for several years for idiopathic localized oral lichen planus.

Nine of the eighteen documented hypersensitivity reactions (50%) manifested as atypical gingivitis and gingival recession and occasionally with advancing periodontitis. There are many possible reasons for this association. Impingement on the biologic width of the gingiva, overhanging or deficient margins, improper contours, traumatogenic occlusion, open or excessively tight contacts, each may play a role in this phenomenon and no doubt are major etiologic factors in localized restoration related periodontal diseases. However, hypersensitivity reactions to cast metal alloys may also play an important role and may represent the most likely etiologic factor when localized rapidly destructive periodontitis is evident on restored teeth.

CONCLUSIONS

1. The incidence of hypersensitivity reactions to cast metal dental restorations and appliances may be increasing due to increased exposure of the public to jewelry, body piercing and tattooing.

2. Hypersensitivity reactions to dental cast metal alloys may induce localized oral lesions in tissues contacting the identified restoration. These reactions include contact lichenoid reactions, mucositis and glossitis, atypical gingivitis and rapidly progressing periodontitis. Complaints of burning in tissues contacting a metal to which the patient is hypersensitive may be representative of a weak hypersensitivity reaction.

3. Patch testing is a valid and valuable diagnostic tool in detecting many cases of hypersensitivity to dental materials but identification of a metals hypersensitivity does not necessarily mean that an individual will experience oral signs or symptoms of the allergy.

4. Diagnosis of an oral hypersensitivity reaction often requires patch testing, recognition of clinically significant lesions and lesional biopsy.
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