Systemic Nickel Allergy after Internal Fixation of a Bunionectomy

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ABSTRACT

Allergic reactions to implanted metals have been estimated to occur in 1% to 5% of orthopedic cases. Stainless steel screws, which contain 14% nickel, are commonly used for internal fixation in an array of podiatric procedures. We present a rare case of a systemic allergic reaction to nickel secondary to stainless steel screw fixation in a bunionectomy procedure.

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An allergic contact dermatitis to stainless steel implants is an infrequent and poorly understood occurrence. Stainless steel screws are commonly used for internal fixation in an array of podiatric procedures. These screws normally contain 14% nickel, 20% chromium, and 27% cobalt (1). Of these components, nickel is the most likely to cause an immune response. Nickel is the most common human metal allergen, representing 10% of all contact dermatitis cases worldwide (1). A greater prevalence has been seen in women. This has been attributed to the greater exposure to nickel-containing metals used in custom jewelry, makeup, belt buckles, zippers, perfumes, and garments (1,2).

Four types of hypersensitivity reactions can occur. Allergic responses have mainly been attributed to type I and type IV reactions. Type I reactions occur within minutes, and the responses range from a dermatitis to deadly anaphylactic shock. Certain allergens, such as food or pollen, are mediated through this response (3,4)

Type IV reactions have a delayed response. Although these reactions can lead to debilitating consequences, they are rarely deadly. Contact dermatitis to nickel is an example of a type IV delayed hypersensitivity reaction and occurs in multiple phases (2–5). The first phase is known as the sensitization phase. It begins with skin exposure to a metal containing nickel. Keratinocytes have a high affinity for nickel. This metal can be retained in the epidermis for an extended period through reversible covalent bonds with skin proteins. Metal ions are known as incomplete antigens or haptens. These haptens alone are too small to trigger an immune response. Once bonds have been formed with endogenous skin peptides, these haptens form complete immunoantigens (1–4). Antigen presenting cells in the skin, known as Langerhans cells or dermal dendritic cells, will engulf the hapten–protein complex and migrate to local lymph nodes, where they will come in contact with T cells. The expression of processed hapten–protein complexes on dendritic cell surfaces will lead to the formation of dendritic cell–T-cell complexes. These complexes result in the activation and formation of memory T cells. Some of these T cells will migrate to the original contact site and could begin a pathophysiologic response. Other cells will circulate as memory cells and respond to the appearance of additional nickel–protein complexes (4). On re-exposure to the antigen, the elicitation phase will be initiated. Such an exposure can be external or internal, such as in screw implantation (4). When the metal ion concentration exceeds a certain threshold, which will vary on an individual basis, memory T cells will enter the skin and stimulate an inflammatory process. Symptoms of contact dermatitis can include erythema, edema, scaling, and a pruritic reaction of the skin (6). The present report describes a rare podiatric case of a type IV delayed hypersensitivity reaction secondary to stainless steel screw fixation.

Case Report

A healthy 56-year-old female had undergone a left chevron bunionectomy that had been fixed with 2 stainless steel screws. Almost immediately postoperatively, within 1 to 2 days, she developed a sensation of warmth around the incision area without any color or other significant visible changes. The feeling never subsided, and she attributed it to the normal healing process and did not report it to the surgeon. At her 6-week postoperative visit, all was normal, and she was...
actively ambulating in sneakers, just as she had been since the second postoperative week. Approximately 2 days after the 6-week visit, she developed a rash about her foot, emanating from the surgical site and extending only proximally (Fig. 1). Initially, the rash was a localized erythematous patch at the incision area. She telephoned a dermatologist. She was seen with consideration for an allergic reaction and prescribed prednisone. She had not yet telephoned the surgeon. In the next 2 days, the rash began to spread systemically about her entire body, including the legs, abdomen, back, and arms and was bilateral (Figs. 1 and 2). The rash consisted of erythematous patches and plaques. No blistering or other systemic reaction was present. At this point, the surgeon was telephoned. Immediately, arrangements were made to have the screws removed. She returned to the dermatologist just before screw removal, and a patch test for nickel was done, with negative results. The screws were removed on postoperative day 56 from the initial surgery. The steroids were discontinued immediately after screw removal. She had taken prednisone for a total of 8 days. Within 48 hours of screw removal, the rash had begun to subside and had fully resolved at the 6-week postoperative visit. A second patch test was done, with results positive for nickel allergy. She had no scarring or other residual effects from the rash.

Discussion

Cutaneous hypersensitivity reactions to implanted metals have been estimated to occur in 1% to 5% of orthopedic cases (5). Early cases of metal hypersensitivity were first reported in the orthopedic data in the 1960s (2). Since then, efforts have been made by manufacturers to produce devices with anticorrosive properties. In modern implants, a small percentage of ions could still leak out when exposed to biologic fluids (2). Also, in patients who have been presensitized to nickel, a minuscule amount of nickel ions could be sufficient to trigger a systemic response. In a study by Disegi et al (7), subjects with known nickel allergies were exposed to a solution containing 5%, 1%, 0.01%, and 0.001% of NiSO₄·6H₂O in petroleum jelly. Of 50 patients, 44% reacted to concentrations ranging from 0.001% to 0.01% (7). Because the specific threshold to nickel varies on an individual basis, it will be difficult to predict which patients could develop a hypersensitivity reaction. The reference standard to diagnose metal sensitivity is the patch test. This test consists of exposing the patient to suspected allergens under controlled conditions (5,8). It is not practical to perform a patch test on every surgical candidate. This test is warranted by a history of nickel allergy (5,8). However, patients could be unaware of such an allergy. In our case, the patient was able to recall a minor reaction to metal earrings retrospectively after her reaction had occurred. It is important for the surgeon to be aware that the patch test can have false-positive or false-negative results (8). In our case, the patient had initially had a negative patch test result owing to the use of concurrent prednisone. Three months after surgical removal of the screws, the patient was retested, and her results were positive for a nickel allergy.

Oral anti-inflammatory and antihistamines downregulate the immune response. This can lead to some relief of metal allergy symptoms, but use of these drugs will not lead to a cure. If the internal fixation remains in the body, the T cells will continue to clone, leading to a continuous response. The definitive treatment of a suspected allergy to internal fixation is removal of hardware, when possible (5).

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References