

Mysterious Recurrent Urticarial Rash and a Life Altering Metal

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Introduction

Systemically reactivated allergic contact dermatitis (ACD) occurs in sensitized individuals after exposure to the antigen by direct skin or systemic contact.

Case Presentation

A 34-year-old woman with a history of peripheral artery disease and transient ischemic attack, presented with generalized itchy hives for one week. She denied exposure to any potentially new allergens. She had tried topical antihistamines, emollients, and steroids without much relief. Prior to the onset of her symptoms, she had a left carotid artery stent placement, and was started on apixaban and carvedilol. Her vital signs were within normal limits. Physical exam revealed erythematous patches with hives on the palms. The patient was started on methylprednisolone and hydroxyzine for symptomatic treatment.

In subsequent office appointments, there was concern that her rash might be due to apixaban, which was switched to rivaroxaban. However, her urticarial rash continued to worsen and became generalized despite adherence to medications. Subsequently, she became hypotensive with a blood pressure of 89/62 mmHg. She was stabilized with intravenous fluids and underwent an extensive allergy testing, which concluded that she was allergic to nickel. Her left carotid stent was a nitinol stent, a nickel-titanium alloy. Her carotid artery stent was removed. Within a week her generalized urticarial rash resolved.



Figure 1: Images of the urticarial rash a) on the bilateral thighs with excoriations, b) on the right wrist with signs of dryness and lichenification, and c) on the anterior left knee with well-defined borders of erythema. *Provided by patient.*

Discussion

ACD has a prevalence of about 20.1% in the general population, the most commonly identified metallic allergen affecting individual is nickel. Nickel is commonly found in watches, earrings, belt buckles, and bracelets. Typical acute presentation includes erythema, edema, vesicles, bullae, and scaling in the affected areas of skin, such as ears, wrists, and infrapubic region. If left untreated, lesions can progress to a chronic phase consisting of hyperkeratosis, dryness, fissuring, and lichenification.

Differential diagnosis should include more life-threatening allergic reactions, and systemically reactivated ACD as in our patient; hence anaphylaxis should be ruled out immediately upon presentation. In addition to contact allergens and fungal infections, systemic inflammatory conditions, such as systemic lupus erythematosus, psoriasis, and dermatomyositis should also be considered due to the increased risk of ACD in this subgroup.

Management of nickel allergy primarily consists of avoiding exposure to nickel-based compounds and removal of the device or piercing. Conservative measures like moisturizers and emollients can be used, however if these are ineffective, the first line pharmacotherapy is topical or systemic corticosteroids. The overall prognosis for nickel-induced contact allergy is excellent, and in the case of our patient, she has experienced complete resolution of her urticarial rash upon removal of the nickel-based stent.

References

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Conclusion

Our case highlights a rather atypical presentation of systemic urticarial rash and anaphylaxis secondary to a nickel-based nitinol carotid stent, which required high index of suspicion and early management by removal of the offending nickel-based agent, ultimately leading to a favorable outcome.