Recurrent Inflammatory Chondritis Following Ear Piercings

Sarah Strandjord 1, Sara Akhtar 2, Blanca E. Gonzalez 2, Sangeeta Krishna 2, Andrew S. Zeft 2

Abstract

Auricular chondritis is an uncommon complication of ear trauma, including ear piercing, that carries a risk of permanent deformity. Auricular chondritis in young adults is typically infectious and without recurrence. Other rare cases of chondritis, which are more common in older adults, are inflammatory and occur as part of an autoimmune disorder called relapsing polychondritis (RP). We present the case of an 18-year-old girl with two separate episodes of auricular chondritis in each ear following cartilage piercing that responded to prolonged steroid treatment.

Key words: Auricular chondritis, perichondritis, relapsing polychondritis, ear piercing

Introduction

Auricular chondritis, inflammation of the cartilage of the ear, is a rare but serious complication of ear trauma that can lead to permanent deformity [1,2]. Rapid identification of the cause of inflammation and appropriate treatment are essential to prevent residual damage. The etiology of auricular chondritis is predominantly infectious but may also be immunological [3]. Non-infectious chondritis usually occurs as part of a rare autoimmune disorder called relapsing polychondritis (RP) [4]. RP typically presents in older adults as inflammation of cartilaginous tissue throughout the body, particularly the ears, nose, joints, and respiratory tract. Here we report a case of recurrent auricular chondritis in a young adult following multiple cartilage piercings.

Case Report

An 18-year-old girl presented to the emergency room with a five-week history of progressive swelling and tenderness of the right ear following piercing of her tragus. These symptoms first became apparent five days following the piercing, prompting her to seek medical care. Despite removal of the piercing and four weeks of oral and intravenous antibiotic therapy targeting Pseudomonas aeruginosa, methicillin-resistant Staphylococcus aureus, and anaerobes, she had minimal clinical improvement. Her past medical history was significant for a similar reaction to piercing of her left helix three years prior, which gradually resolved over two months following piercing removal, incision and drainage, and a one month course of intravenous antibiotics.
Physical examination revealed a well-appearing, afebrile patient with pronounced edema, erythema, and tenderness of the right pinna, sparing the tragus and the earlobe (Figure 1a,b). The patient had mild subjective hearing loss, presumably from swelling of the external ear canal, and no aural discharge. Laboratory results revealed a normal white blood cell count and C-reactive protein, mild anemia (Hbg 10.9 g/dL), and elevated erythrocyte sedimentation rate (66 mm/hour).

Otolaryngology, Infectious Disease, and Rheumatology services were consulted during her hospital stay. She was started on broad-spectrum intravenous antibiotics along with non-steroidal anti-inflammatory (ketorolac 30 mg) and opioid (hydromorphone 0.5 mg) analgesics for pain. Her ear exam remained unchanged on antibiotics and the patient reported better pain relief with anti-inflammatory, as compared to opioid, therapy. A CT scan was performed for a possible retained foreign body due to her lack of improvement with prolonged antibiotics. Results showed extensive soft tissue inflammation involving the right auricle without abscess or foreign body. Antibiotics were subsequently discontinued and an autoimmune inflammatory process was considered. Complete physical examination, including ophthalmologic evaluation, was unremarkable for other sites of inflammation. We elected not to perform a tissue biopsy on the ear in order to prevent further incitation of the inflammatory response or subsequent infection [2,5]. Given a presumptive diagnosis of inflammatory chondritis, the patient was started on a daily intravenous pulse of methylprednisolone (1 gram) with a significant response in swelling over a two-day period. She was discharged on a three-month prednisone taper starting at 1
mg/kg/day along with oxycodone for pain. After nine weeks of tapering prednisone (current dose of 15 mg/day), her ear was no longer tender with only a mild erythema (Figure 2) and, by 16 weeks, was within normal limits.

**Discussion**

Auricular chondritis usually begins with localized redness, warmth, and swelling that, if left untreated, progresses to involve the entire cartilage of the ear. This inflammatory process eventually results in disfiguring necrosis, often referred to as “cauliflower ear.” The most common etiology of auricular chondritis is infectious, with Pseudomonas aeruginosa responsible for over two-thirds of cases followed by Staphylococcus aureus [3]. Recurrent episodes of infectious auricular chondritis are uncommon and usually occur in patients with comorbid conditions that predispose them to infection, such as diabetes mellitus [6,7]. Our patient is unique in that she experienced nearly identical episodes of inflammatory chondritis in each ear following cartilage piercing. While her initial episode resolved without immune-modulating therapy, the second episode required a prolonged course of glucocorticoid therapy. We suspect that the pathogenesis of the second episode involved an adaptive memory immune response from the first trauma.

Non-infectious auricular chondritis typically occurs as part of a rare autoimmune disorder called relapsing polychondritis (RP). RP is a disease characterized by recurrent inflammation of cartilaginous tissue in various sites of the body, including the ears, nose, and laryngobronchial tree [4]. The diagnostic criteria for RP, first proposed by McAdam et al. in 1976, require three or more of the following: (1) bilateral auricular chondritis, (2) nonerosive, seronegative inflammatory polyarthritis, (3) nasal chondritis, (4) ocular inflammation (conjunctivitis, keratitis, scleritis/episcleritis, uveitis), (5) respiratory tract chondritis (laryngeal and/or tracheal cartilages), and (6) cochlear and/or vestibular dysfunction (neurosensorial hearing loss, tinnitus, and/or vertigo) [8]. Since then, several modifications have been recommended, including diagnosis on the basis of one or more of the above findings along with positive histologic confirmation or chondritis at two or more anatomic locations with response to steroids [9].

The etiology of RP remains unknown, but genetic susceptibility and immunologic abnormalities appear to play a prominent role in disease pathogenesis [10]. Associations with the HLA-DR4 serotype [10-12], for example, have been identified as well as numerous associations with autoimmune diseases, particularly systemic vasculitis, rheumatoid arthritis, and systemic lupus erythematosus [13,14]. In addition to genetic and immunologic predisposing factors, many cases of RP are preceded by some inciting event, such as an infection, drug exposure, or trauma [2,15]. Puncture wounds, for instance, have been hypothesized to trigger RP by exposing cartilage matrix protein antigens to immune surveillance, leading to an autoimmune response [2]. Case reports have identified cartilage ear piercing as the initiating insult leading to RP in individual patients [15,16]. Classically, RP is a condition that occurs during mid-adulthood, although RP has been identified in children and young adults [4,17].

At the time of presentation, our patient did not meet the traditional diagnostic criteria for RP and, since we opted not to perform a tissue biopsy, it is unclear whether she met the modified diagnostic criteria for RP at that time. Her minimal improvement on antibiotics and her subsequent recovery on steroids, however, support the diagnosis of a primary non-infectious inflammatory chondritis. These two episodes of auricular chondritis may be the initial presentation of RP, which will eventually progress to involve other anatomic sites, or they may be the manifestation of a milder form of...
non-infectious chondritis. Regardless of the final diagnosis, our report supports expanding the differential for trauma-induced chondritis in young adults to include rheumatological as well as infectious etiologies. It also adds to the existing literature on recurrent trauma-induced chondritis, in this case from multiple cartilaginous locations occurring at separate time points. A course of steroids is justified in patients with chondritis that is refractory to antibiotic therapy, even without a clear diagnosis of RP, in order to prevent residual damage and deformity.

**Competing interests:** The authors declared no competing interest.

**Funding:** None.

**Provenance and peer review:** Not commissioned; externally peer reviewed.

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