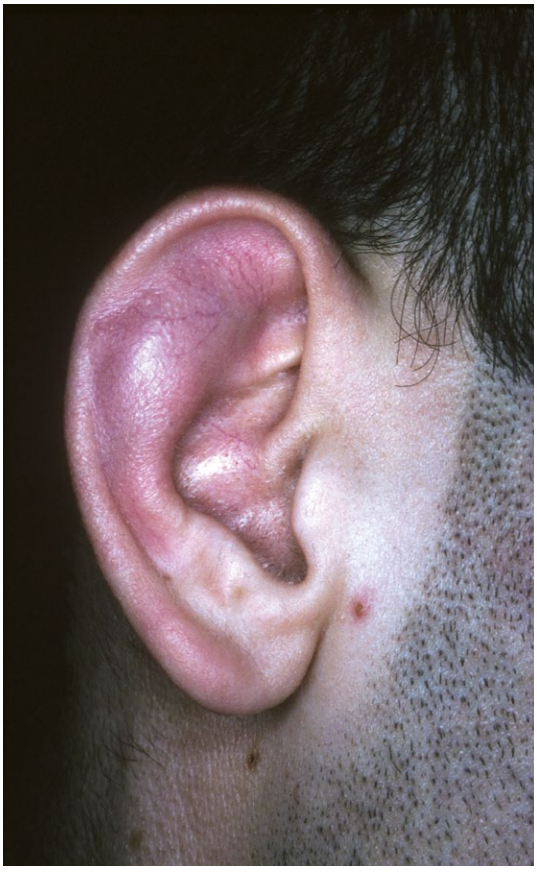
What Is Your Diagnosis?



A 41-year-old man presented with slightly tender swelling on the ear helix of several years' duration. The patient was otherwise healthy and his family history was noncontributory. There were no other abnormal associated skin findings. He did not recall any history of trauma to the pinna.

PLEASE TURN TO PAGE 20 FOR DISCUSSION

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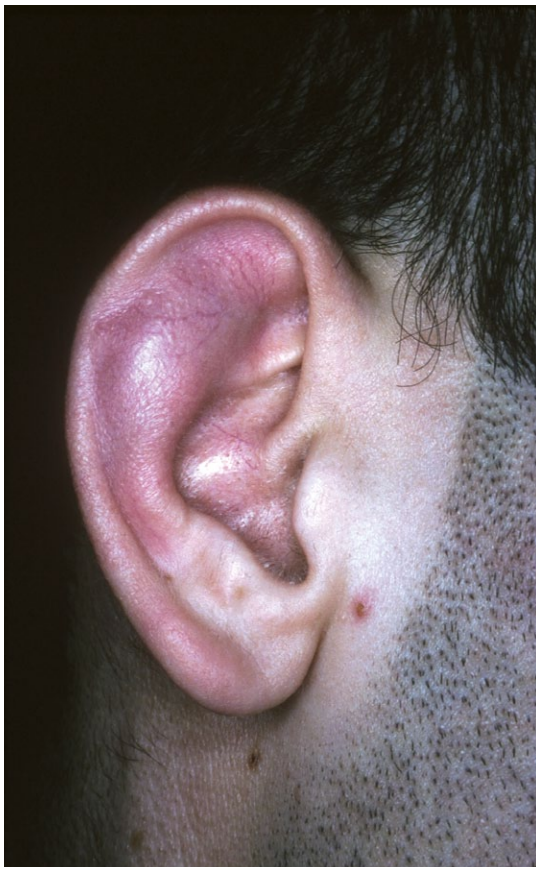
The Diagnosis: Pseudocyst of the Auricle



Pseudocyst of the auricle (PCA) is a benign condition characterized by noninflammatory, painless, cystic enlargement of the anterior and lateral aspects of the auricle. It most often presents unilaterally in healthy adult males. The most common areas of the pinna affected by PCA are the scaphoid and triangular fossae.[1] Patients develop painless fluctuant swelling of the auricle over 4 to 12 weeks. Diagnosis often is made on clinical grounds.

Pseudocyst of the auricle was recognized in 1846[1] and was first described in the English language literature by Engel[2] in 1966. In 1990, Cohen and Grossman[3] conducted a world literature review and reported that PCA most frequently occurred in Chinese and white patients. However, cases also have been reported in other ethnicities (ie, Japanese, French, Indian, Malaysian, black, and Puerto Rican patients). Most patients were males who developed PCA between 16 and 73 years of age. Most of the patients developed PCA between 30 and 39 years of age. Overall, a majority of patients had unilateral involvement of the auricle in the right ear.[3]

Patients with PCA develop painless fluctuant swelling of the auricle over 4 to 12 weeks and often are otherwise healthy and asymptomatic. Pseudocyst of the auricle is called *pseudocyst* rather than *cyst* because lesions lack a true epithelial lining. There are no epidermal findings because the pseudocyst is contained in the cartilage. The cartilage deteriorates and becomes filled with "viscous, yellow-tan fluid, resembling 'olive oil,'" leading to the pseudocyst forming.[4] Diagnosis often is made on clinical grounds, but further aspiration and histologic examination can be performed.[4] Histologic examination shows sparing of the epidermis and dermis overlying the pseudocyst. The dermis around the pseudocyst may have a lymphocytic infiltrate with fibrosis and granulation tissue.[1]

The cause of PCA is unknown, but there are 2 main theories of its pathogenesis. The first hypothesis indicates that PCA is caused by repetitive or acute trauma. Insult to the auricle results in

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expulsion of lysosomal enzymes that damage and disrupt the cartilage.[2,5] This hypothesis was supported by Ichioka et al[6] who reported an increase in levels of lactate dehydrogenase isoenzmes LD_4 and LD_5 in pseudocyst fluid as compared to serum; LD_4 and LDH_5 are found predominantly in human cartilage.[6] Examples of trauma prior to PCA formation include striking the ear,[7] wearing a helmet,[6] sleeping on a hard pillow,[8] or carrying heavy items on the shoulders.[8] Ng et al[9] also reported several cases of PCA in patients with chronic atopic dermatitis. They suggest that pruritus secondary to atopic dermatitis could cause repetitive trauma to the auricle, leading to PCA.[9]

The second hypothesis suggests that during embryogenesis, an abnormality in the development of the first and second brachial arches that develop into the auricle results in excess tissue planes that can allow for pseudocyst formation.[5] This hypothesis was supported by data from Zhu and Wang[10] who histologically examined 42 auricles with PCA. They found 12 patients with connective tissue, blood vessels, and lymphatics within the cartilage. Four of those auricles had vessels and lymphatics connected outside the cartilage. Moreover, 22 patients had excess tissue planes in the cartilage where PCA could develop.[10]

Other diagnoses to consider are relapsing polychondritis, chondrodermatitis nodularis chronica helicis, othematoma, and subperichondrial abscess.[11] Relapsing polychondritis can be clinically ruled out because the auricle would be erythematous and tender and other cartilage would invariably be involved.[11] Histologic examination would reveal a lack of cystic changes, signs of acute inflammation, and antibody deposition.[4]

Chondrodermatitis nodularis chronica helicis also can be clinically ruled out because only the superior helix would be involved. Furthermore, the auricle would have a tender nodule with ulceration and would lack cystic changes. Frequently the patient would have a history of acute trauma.[4,11]

Othematoma can be clinically ruled out because the auricle would be tender with erythema due to hematoma formation. Patients also would have a history of trauma.[11]

Subperichondrial abscess can be clinically ruled out because the patient would have tender, erythematous, fluctuant swelling or the auricle would contain pus. The patient also could have a history of trauma (ie, ear piercing).[11,12]

Treatment of PCA can include oral steroids, steroid injection, application of auricular pressure and simple aspiration, minocycline injection, surgical intervention, or curettage followed by fibrin glue.[13-17] Failure to treat can result in permanent deformity of the auricle that resembles a cauliflower ear.[13] Job and Raman[14] reported 4 cases in which oral steroids (high-dose oral corticosteroids) were used as a noninvasive treatment of PCA. They hypothesized that the steroids helped decrease the formation of cartilage fibrosis and granulation tissue and helped decrease edema.[14]

Intralesional steroids for PCA also have been used. Glamb and Kim[13] reported a case of bilateral auricle involvement in which betamethasone phosphate was injected into one the pseudocysts. The long-term result was anatomical deformity of both auricles. Although they could not be certain the steroids were the cause, they do not support this treatment.[13]

Another therapy is auricular pressure and simple aspiration. Salgado et al[15] reported success using this minimally invasive treatment to yield ideal cosmetic results. This method prevents side effects of steroids, such as pigment changes in the skin and tissue atrophy.[15]

Another treatment method is intralesional injection with minocycline. Oyama et al[16] reported 2 cases of refractory PCA in which both patients responded to intralesional injection with minocycline. Although the exact mechanism is unknown, this treatment could have been successful because minocycline induced fibrosis and prevented cytokine inflammation in the auricle.[16]

If medical therapy fails, surgical intervention also is an option. Harder and Zachary[8] reported success by using a deroofing technique whereby the skin over the pseudocyst was dissected, allowing for excision and drainage of the lesion. Another surgical option reported by Tuncer et al[17] involved curettage followed by application of fibrin glue between the layers of cartilage to increase adherence. In their case report, the patient had failed 2 needle aspirations and an attempt at incision and drainage followed by pressure. At the 6-month follow-up, the patient had no PCA reoccurrence with ideal cosmetic results.[17]

Treatment options were reviewed with our patient, but he declined all of them and was lost to follow-up.

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