Petrified Ears and Imaging Studies

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Abstract

Petrified ears are an uncommonly reported condition characterized by stony hard calcified auricular cartilage of one or both ears without visible changes in the appearance of the ear. This condition is associated with various metabolic, endocrine and systemic diseases. In addition, other local causes like injury, trauma, actinic damage, insect bites, frostbite, radiation therapy, chondritis and perichondritis may also play a role. We present such a rare case in a 61-year-old chronic diabetic male with ischemic heart disease.

Keywords: Petrified ears; Calcification; Diabetes; Microangiopathy

Abbreviations: PE: Petrified Ear; PTCA: Percutaneous Transluminal Coronary Angioplasty; CT: Computed Tomography

Introduction

Petrified ear (PE), whether due to calcification or ossification is a rare diagnosis. It is usually asymptomatic and characterized by development of stony hard auricular cartilage of one or both ears without visible changes in the appearance of the ear. A variety of exogenous environmental exposures and endocrinopathies have been associated with the development of this entity, although the exact aetiology remains unknown [1].

Case Report

A 61-year-old male presented with rigid, inflexible ears, with pain on pressure and no hearing loss of 5 years duration. Detailed clinical history revealed that he was a non-smoker and has been suffering from diabetes and hypertension for last 10 years. He underwent percutaneous transluminal coronary angioplasty (PTCA) with stent for inferior wall myocardial infarction 2 months ago. There was no history of wrestling, boxing, headset use, frostbite, trauma, infection to the ears. On physical examination, helix and anti-helix of both ears was stony hard, with slight tenderness and could not be folded (Figure 1-2). The earlobes were normal and easily mobile. Patient

Figure 1: Helix and anti-helix of ear is stony hard with normal earlobes.

Figure 2: Inflexible ear.
general condition was good and vitals were: pulse rate: 82 beats per minute; blood pressure: 130/90mmHg; respiratory rate: 18cycles per minute. There was no regional lymphadenopathy. Complete ear, nose and throat examination including audiometry revealed no abnormalities. Systemic examination was normal. The following investigations were carried out, haemoglobin:14.6gm/dl; total count: 6500cells/cumm; differential count: P: 80%; L:15%; E:4%; M:1%; ESR: 13mm/hr; liver function test: normal; blood urea:40mg/dl; serum creatinine:0.9mg/dl; fasting blood sugar:104mg/dl; HbA1c:7; serum calcium:10.5mg/dl; serum phosphorus:4.5mg/dl; TSH:3.31uIU/ml; T3:1.68mmol/L; T4:94.08mmol/L. A complete metabolic panel, lipid profile, parathyroid hormone, serum morning cortisol, adrenocorticotropic hormone and vitamin D were within normal range. Radiograph of the chest was normal. X-ray of the right ear AP and lateral view showed mildly bulky right external ear. A non-contrast temporal bone computed tomography (CT) scan demonstrated dense ossification of the external auricular cartilage (Figure 3a-3b & 4a-4b). Ossification was not seen in other areas. Paranasal sinuses were normal. Based on the clinical and radiological findings a diagnosis of petrified ears was made.

Discussion

Petrified ears is characterized by development of partial or total rigidity either due to calcification or ossification of the auricle without any visible external change. Bochdalek first reported a case of PE in 1866, while Wassmund first reported the X-ray findings of this condition in 1899. Approximately, 140 cases with calcification or ossification of elastic cartilage have been reported till date by radiological and histological examination [1]. Most patients are asymptomatic and bilateral involvement is more common. It occurs more commonly in men than in women. Rarely, associated with hearing loss [2,3].

Elastic cartilage is a component of the auricle, external ear canal, nose and epiglottis which usually does not have a tendency for calcification or ossification [2]. Petrification of auricular cartilage is attributed to dystrophic calcification or metastatic calcification and ectopic ossification. Petrification is caused more often by calcification than ossification. Dystrophic calcification is seen in damaged tissue after local injuries, mechanical trauma, actinic damage, insect bites, frostbite, radiation therapy, chondritis and perichondritis. Metastatic calcification occurs in normal soft tissue due to presence of high serum calcium-phosphorus levels secondary to hypercalcemia, milk-alkali syndrome, vitamin D intoxication,
hyperparathyroidism, sarcoidosis. Adrenal insufficiency is the most common aetiology of metastatic calcification. Other systemic diseases that have been associated with auricular calcification include diabetes, hypertension, hypothyroidism, hyperthyroidism, hypopituitarism, hyperparathyroidism and many other conditions [3,4]. Ectopic ossification involves new bone formation in tissue that normally does not ossify. Severe hypothermia has been considered as the most common cause of auricular ossificans. Rapid cooling has been suggested to produce vascular thrombosis, occlusion and ischemia which can induce lamellar bone proliferation [2,3]. Recently, one interesting case of PE as a complication of Bluetooth headset use has been reported [5].

Laboratory evaluation of these cases is mandatory to detect underlying metabolic, endocrine disorders or other systemic diseases. Imaging studies help to demonstrate calcification. Simple X-ray may not demonstrate the calcification in all cases. Jessie et al. [3] & Clarke et al. [6] reported that lateral skull and Caldwell X-ray views are best to visualize calcification. Temporal bone CT will definitely demonstrate auricular calcification and the common CT findings are track-like or trabecular calcification of the cartilage of the pinnæ.

PE may be the presenting symptom in some cases and help the physician to detect underlying endocrinopathies like diabetes, addison’s and other systemic diseases. Thus, PE may act as cutaneous marker for systemic disease. As PE is asymptomatic, no specific therapy is necessary. Evaluating and correcting the underlying metabolic, endocrine or systemic condition is essential. Patients who are symptomatic show improvement with wedge resection of affected cartilage or conchal reduction surgery [1].

In our patient there were no triggering factors for PE except diabetes. We speculate, diabetic micro-angiopathy may be the causative factor for development of PE. Microangiopathic process may induce local damage or cartilage necrosis with subsequent calcification, probably because tissue damage allows increased alkalinity, with increased intracellular calcium fluxes [7,8].

References