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# Incus and stapes necrosis associated with diabetes mellitus

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**Abstract**
Chronic otitis media is often associated with ossicular defects, the most frequent being necrosis of the long process of incus. Except for infection and cholesteatoma; trauma and local pressure by chorda tympani are uncommon causes leading to incus erosion. In the literature, no case of incus necrosis has been reported associated with type II diabetes mellitus (DM). A patient is presented in this report with incus and stapes suprastructure necrosis and associated type II DM who was admitted to the out-patient clinic with complaints of conductive hearing loss.

**Key words: Incus; Stapes; Necrosis; Diabetes Mellitus**

## Introduction

Chronic otitis media is most often associated with ossicular defects and the most frequent of these is necrosis of the long process of the incus.[1] The long and lenticular processes of the incus are the segments most vulnerable to necrosis and the malleus and footplate are the most resistant.

Infection and cholesteatoma are the commonest causes of ossicular damage but trauma and local pressure by the chorda tympani may also cause incus erosion. In the literature no cases of incus necrosis have been reported associated with diabetes mellitus. A patient with incus necrosis associated with type II diabetes mellitus is presented in this report.

## Case report

A 43-year-old male patient was admitted with a rapid onset of hearing loss. Symptoms of hearing loss had been present for one year and he had a history of type II diabetes mellitus dating back 10 years. The patient was under the care of the endocrinology department and his blood glucose had been regulated with diet and oral anti-diabetic agents. He has taken no other medication. There was no history of head trauma, middle-ear infection or previous aural surgery. Otoscopic and otomicroscopic examination was normal but the pure tone audiogram, which was normal on the left side, showed an average bone conduction level of 20 decibels (dB) with an air conduction level of 90 dB on the right. The tympanogram on the right suggested an ossicle dislocation or defect (type AD) but was normal on the left side. The ipsilateral acoustic reflex on the left ear was positive and the contralateral was negative. Mastoid pneumatization was normal bilaterally in Schüller graphy.

The middle ear was explored in January 2003 under general anesthesia by elevation of a tympanomeatal flap. The malleus and footplate were intact but the lenticular process and suprastructure of the stapes were lacking (Figure 1). No additional pathology was found. The remaining incus was removed and, after sculpturing, was inserted between the malleus and the footplate of the stapes. The position was maintained by Gelfoam.

In the post-operative period the air conduction level of the right ear had returned to an average of 23 dB.

## Discussion

Ossicular erosion can result from inflammation, cholesteatoma and trauma, and incus erosion has also been reported following pressure from the chorda tympani nerve.[1] It has also been shown that the lenticular and long processes may undergo progressive symmetric resorption with advancing age due to increasing osteoclastic activity.[2]

Alberti [3] first detailed the arterial supply of the lenticular and long processes of the incus and most believe that the predilection of the long process of incus to undergo resorption is attributable to its irregular and tenuous blood supply. However, Thomsen *et al*.[4] suggested that both the long process of the incus and the stapes suprastructure were at risk simply because of their delicate structure, and Lannigan *et al*.[5] showed that the vascular anatomy of the long process of incus is similar to that of the short process and body and concluded that necrosis in the long process could not be related to vascular insufficiency. They also demonstrated that the lenticular and long processes possess a greater degree of osteoclastic pitting of the bone surface than is found elsewhere on the incus or in the rest of the ossicular chain and suggested that osteoclastic activity may account for the predilection of this portion of the ossicular chain to undergo resorption.[2] Local control of osteoclastic ossicle resorption has not yet been described, but it is known that the mediators responsible for bone resorption are collagenase, osteoclastic activating factor, prostaglandins and lipopolysaccharide as well as increased lysosomal activity.[1] However, the situation is not clear. Imauchi *et al*.[6] reported an acquired atrophy of the long process and proposed several pathologies

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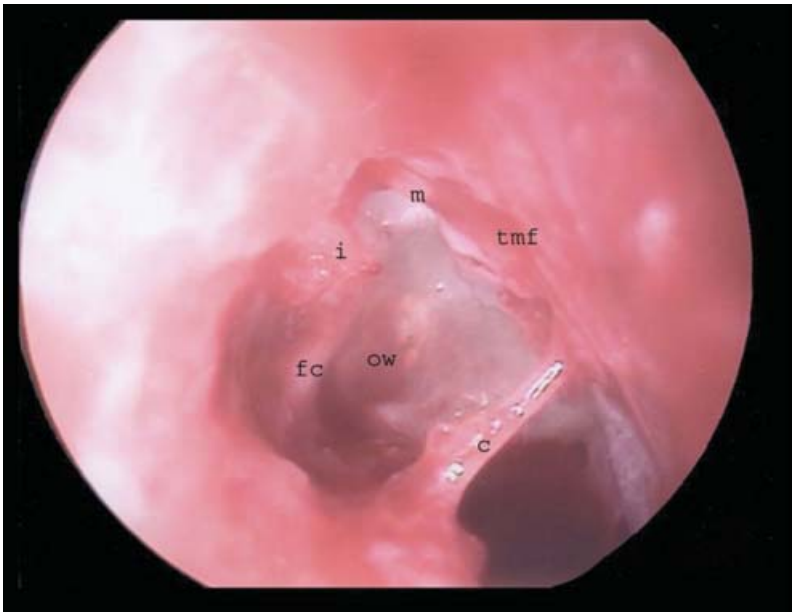


FIG. 1
The view of the middle ear after the elevation of the tympanomeatal flap. m = malleus; i = incus; tmf = tympanomeatal flap; fc = facial canal; ow = oval window

including an inherited impairment of the vascular supply and there have been experimental studies which have shown that acute otitis media may alter modelling in bone tissue surrounding the middle-ear cavity – including the bony external auditory canal and the ossicles.[7,8]

While there is no consensus as to the pathophysiology of many cases of ossicular discontinuity, necrosis has not been previously reported in association with systemic causes or without tympanic membrane or middle-ear pathology. Diabetes mellitus causes incremental increases in prostaglandin levels by activating the enzyme cyclooxygenase, and prostaglandins may result in bone erosion by inducing osteoclastic activity.[9] Alternatively, basal membrane thickening in the arterial supply is common in diabetes and this may result in an insufficient local blood flow.[10] While our case had no overt history of acute otitis media it is also possible that he had a low grade infection which his diabetes exacerbated.

On balance, since no local cause has been determined for necrosis in our patient, we believe that the necrosis of the incus and head of the stapes must be attributable to the type II diabetes mellitus consequent upon an impaired vascular supply and subsequent increased osteoclast activity and incus long process dissolution with absorption of the stapes head. Such erosion is likely to have been relatively acute since, if necrosis occurs over a long period of time, the resultant defect is usually filled with fibrous tissue and conduction may be preserved. In our patient hearing loss developed in one year and no fibrous tissue was seen intra-operatively.

In conclusion we believe that our patient's diabetes mellitus caused marked osteoclastic activity with ossicular necrosis – particularly in the proximal section of the long process of incus. Such a pattern of progressive hearing loss has not, we believe, been reported previously.

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