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External auditory canal cholesteatoma and benign necrotising otitis externa: clinical study of 95 cases in the Capital Region of Denmark

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Main Article

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Abstract

Objective. To identify epidemiological and pathophysiological factors, and treatment strategies, in external auditory canal cholesteatoma and benign necrotising otitis externa.

Methods. A retrospective case study was conducted of patients suffering from external auditory canal cholesteatoma and benign necrotising otitis externa admitted to tertiary hospitals, in the Capital Region of Denmark, over a five-year period.

Results. Eighty-three patients (95 ears) with external auditory canal cholesteatoma or benign necrotising otitis externa were identified. A minimum incidence rate of 0.97 per 100 000 inhabitants per year was demonstrated. Sixty-eight per cent of cases had a history of smoking. Most lesions (74 per cent) were localised in the floor of the ear canal. Treatment time was 3.2 months for patients who had surgery and 6.0 months for those who received conservative treatment.

Conclusion. It is suggested that external auditory canal cholesteatoma and benign necrotising otitis externa are in fact the same disease, and therefore the diagnosis of external auditory canal cholesteatoma should be changed to benign necrotising otitis externa. Microangiopathy has a leading role in the aetiology. Surgery should be conducted in most cases.

Introduction

External auditory canal cholesteatoma is a rare otological disease with an estimated incidence of 0.19–0.30 cases per year per 100 000 inhabitants in Central and Northern Europe, and 1 per 1000 new otological patients in private practices.[1]

External auditory canal cholesteatoma is often divided into two categories: primary and secondary. Primary external auditory canal cholesteatoma is idiopathic. It is characterised by: localised bone erosion filled with concentric layers of keratinised epithelial lamellae within a cyst-like sac; and a matrix with an inner layer consisting of keratinised and stratified squamous epithelium, and an outer layer of connective tissue.[2] Secondary external auditory canal cholesteatoma has the same pathophysiology, but the aetiology is different. It has a clear cause, resulting from surgery, trauma, irradiation, inflammation or years of bisphosphonate treatment. Whether primary or secondary, the treatment is the same.

Benign necrotising otitis externa is described as a condition with an ulcer in the floor of the external auditory canal, with small granulations at the edges, and underlying bony necrosis with sequestrum.[3] According to the sparse literature on benign necrotising otitis externa, it is a rare disease (it is not even mentioned in the *Scott-Brown's Otorhinolaryngology* textbooks). It differs only from external auditory canal cholesteatoma in that it has no cholesteatoma matrix in the bottom of the defect. Benign necrotising otitis externa has no code in the International Classification of Diseases, 10th revision ('ICD-10').[4] Both external auditory canal cholesteatoma and benign necrotising otitis externa are treated the same way.

Different differential diagnoses for external auditory canal cholesteatoma and benign necrotising otitis externa are important to consider, such as auditory canal carcinoma, malignant necrotising external otitis and Langerhans' cell histiocytosis of the petrosal bone.[5] External auditory canal cholesteatoma has often been associated with other diseases of the external ear canal, especially keratosis obturans. In 1980, Piepergerdes *et al.* classified keratosis obturans as a separate disease, with different clinical and pathological characteristics.[6] Keratosis obturans appears with presenting symptoms such as acute severe pain and conductive hearing loss, often occurring bilaterally.

Several hypotheses on the pathogenesis of external auditory canal cholesteatoma have been suggested. It has been hypothesised that external auditory canal cholesteatoma develops from a reactive process due to an underlying osteitis of the external auditory canal. Mechanical factors (hearing aid, cotton swabs) as well as smoking, resulting in tissue ischaemia, have been suggested as part of the aetiology.[1] Makino and Amatsu suggested abnormal migratory capacity of the ear canal epithelium.[7] However, in 2008, Bonding and Ravn

conducted a study on the migratory capacity of the epithelium in the external auditory canal, and did not find significant differential migration between normal ears and ears with external auditory canal cholesteatoma, except on crust-covered surfaces.[8]

This study focused on the epidemiology of external auditory canal cholesteatoma and benign necrotising otitis externa together. As benign necrotising otitis externa is a little known diagnosis in Denmark and, furthermore, has no International Classification of Diseases 10th revision code, we believe that patients with this disease are often referred to ENT departments under the diagnosis of external auditory canal cholesteatoma. Our clinical experience with these patients over more than 20 years has led us to believe that benign necrotising otitis externa and external auditory canal cholesteatoma are in fact the same disease. Hence, regardless of whether there is cholesteatoma matrix or not, we diagnose it as external auditory canal cholesteatoma with the International Classification of Diseases 10th revision code H60.4.

Materials and methods

A retrospective study was conducted of the records of patients diagnosed with external auditory canal cholesteatoma (International Classification of Diseases 10th revision code H60.4). As stated above, in this study we defined external auditory canal cholesteatoma as a condition with bony erosions or cavitation in the external auditory canal, with or without cholesteatoma matrix.

The patients were referred from private otologists. The diagnosis was then verified and malignancy was ruled out at ENT departments in hospitals in the Capital Region in Denmark (i.e. Rigshospitalet, Gentofte Hospital and Hillerød Hospital). The patient records were collected over a five-year period from 1 January 2009 to 31 December 2013.

The research focused on: patients' symptoms, age and gender, risk factors, co-morbidities, and disease treatment. The patients were divided into primary or secondary cases.

The patients underwent either conservative treatment or surgery. Conservative treatment consisted of cleansing, topical antibiotics, anti-inflammatory ointment on gauze and curettage. The indications for surgery were: more extensive disease and failure of initial conservative treatment. Surgical treatment mostly consisted of: ear canal surgery, with excision of abnormal skin, and drilling and saucerising to remove necrotic bone; and grafting of the defects with fascia or perichondria. This was followed by packing with gauze for two to three weeks. In extensive cases, mastoid surgery with a canal wall down procedure was performed, with/or without obliteration.

Results

During the 5-year period, 153 patients with suspected external auditory canal cholesteatoma were referred by private otologists to Rigshospitalet, Gentofte Hospital and Hillerød Hospital. Of the referred patients, we identified 83 patients diagnosed with external auditory canal cholesteatoma. The remaining 70 patients had different diagnoses, such as cyst-like processes, cerumen impaction or middle-ear cholesteatoma. Eleven patients had bilateral external auditory canal cholesteatoma, and one patient had two separate external auditory canal cholesteatomas on the same ear, three years apart. In total, we found 95 ears (52 left, 43 right) with external auditory canal cholesteatoma in the 5-year period. Eight ears had advanced external auditory canal cholesteatoma, in which adjacent structures were involved. The causes, extension and presenting symptoms of these cases are depicted in Table I.

TABLE I ADVANCED CASES OF EXTERNAL AUDITORY CANAL CHOLESTEATOMA*

Characteristic	Cases (*n* (%))
Primary cases	4 (50)
Secondary cases	4 (50)
– Post-traumatic	1 (25)
– Post-operative	2 (50)
– Irradiation	1 (25)
Structures involved	
– Mastoid	3 (38)
– Middle ear	1 (12)
– Antrum	1 (12)
– Atticus	1 (12)
– Temporomandibular joint	2 (25)
– Tympanic membrane	4 (50)
Presenting symptoms	
– Otalgia	2 (25)
– Otorrhoea	5 (63)
– Hearing loss	5 (63)
– Feeling of occlusion	2 (25)
– Vertigo	1 (12)
– Tinnitus	1 (12)

*Total $n = 8$

We identified 69 primary and 26 secondary cases of external auditory canal cholesteatoma. According to their causes, radiation was the most common secondary cause, with 15 cases (58 per cent). This was followed by the causes of: trauma (microtrauma associated with excessive use of cotton swabs or wooden sticks used to scratch the external auditory canal), in six cases (23 per cent); surgery, in four cases (15 per cent); and bisphosphonate treatment (for 12 years), in two bilateral cases (8 per cent). In one case, both surgery and radiation therapy were considered the causes of secondary external auditory canal cholesteatoma.

The distributions of age, gender and presenting symptoms of the external auditory canal cholesteatoma cases are shown in Table II.

The localisation of external auditory canal cholesteatoma for all cases is shown in Figure 1. Some ears had more than one area affected simultaneously. The primary cases involved the canal floor in 72 per cent of cases, the anterior wall in 52 per cent, the posterior wall in 20 per cent and the canal roof in 4 per cent. The secondary cases had a similar spread of location: 79 per cent in the canal floor, 54 per cent in the anterior wall, 46 per cent in the posterior wall and 4 per cent in the canal roof.

In 68 per cent of cases, the patients were smokers or former smokers. Only 25 per cent were non-smokers. Smoking status was unknown in 8 per cent of cases.

The patients were treated with surgery in 41 cases and conservative therapy in 48 cases. Six cases were lost to follow up before treatment. The conservative treatment consisted of: cleansing (in 100 per cent of cases); topical antibiotics and anti-inflammatory ointment (in 79 per cent), including two

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TABLE II TYPE, GENDER, AGE AND PRESENTING SYMPTOMS OF EXTERNAL AUDITORY CANAL CHOLESTEATOMA CASES

Characteristic	All cases	Primary cases	Secondary cases
Ears diagnosed with EACC (*n* (%))	95 (100)	69 (73)	26 (27)
Gender (*n* (%))			
– Male	46 (48)	37 (54)	9 (35)
– Female	49 (52)	32 (46)	17 (65)
Age at diagnosis (mean ± SD; years)	58.0 ± 17.9	59.4 ± 16.4	53.6 ± 21.3
Presenting symptoms (*n* (%))*			
– Hearing loss	24 (25)	15 (22)	9 (39)
– Otalgia	22 (23)	16 (23)	6 (26)
– Asymptomatic	21 (22)	17 (25)	4 (17)
– Prurigo	15 (16)	13 (19)	2 (9)
– Otorrhoea	14 (15)	10 (14)	4 (17)

*Presenting symptoms not described in six patients. EACC = external auditory canal cholesteatoma; SD = standard deviation

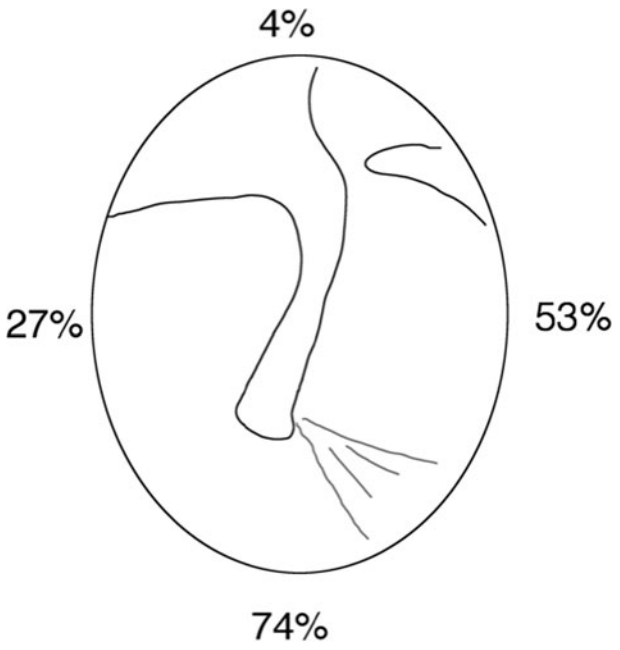

Fig. 1 Illustration of the right ear canal, with localisation of external auditory canal cholesteatoma shown in percentages.

TABLE III OUTCOME AT DISCHARGE AND SMOKING STATUS

Parameter	Cases (*n* (%))
Total discharged from out-patient clinic	78 (82)
– Discharged to private otologist	40 (42)
– Discharged without further treatment	38 (40)
Outcome at discharge from out-patient clinic	
– Complete skin cover of EAC	51 (65)
– No pain	66 (85)
– Self-cleansing ear	49 (63)
Smoking	
– Smoker at diagnosis	58 (61)
– Non-smoker	24 (25)
– Former smoker	6 (6.5)
– Unknown	7 (7.5)

EAC = external auditory canal

to three weeks of gauze packing in the external auditory canal; and curettage (in 38 per cent).

Overall, 78 cases were discharged from the out-patient clinic. Of these, 27 had minor skin defects of no significance, and could be treated regularly at a private otologist. Seventeen cases were lost to follow up during the period of treatment and observation. For details regarding smoking status and outcome at discharge from the out-patient clinic, see Table III.

The mean time (± standard deviation (SD)) from diagnosis to the end of therapy for patients treated conservatively was 6.0 ± 0.4 months. The mean time from surgery to the end of therapy was 3.2 ± 0.8 months.

Discussion

Eighty-three patients with 95 external auditory canal cholesteatomas were identified during the 5-year study period. Given the average population size between 2009 and 2013 of 1 703 970 inhabitants in the Capital Region,[9] the incidence rate is 0.97 per year per 100 000 inhabitants. General otorhinolaryngology practitioners may have treated early or mild cases of external auditory canal cholesteatoma conservatively, without referral to a tertiary centre. These patients are not included in the study. In addition, we do not know how many asymptomatic cases remain undiagnosed. These biases clearly influence the incidence rate, resulting in a false lower rate. Therefore, 0.97 per 100 000 per year is the minimum incidence rate for the Capital Region of Denmark. The population has been relatively constant during the period; hence, the estimated incidence rate seems reliable.

Nevertheless, our incidence rate is three times higher than that previously suggested by Dubach *et al.*[1] Dubach *et al.* found incidence rates between 0.19 (as reported in Tos[3]) and 0.3 (as cited in Owen *et al.*[10]) per 100 000 inhabitants per year in Switzerland, and Denmark and Greenland, respectively. Owen *et al.*[10] did not include cases with hyperplasia and periostitis, only the Naim external auditory canal cholesteatoma classification stages of III and IV,[11] which may explain the disparity between their incidence rate and that reported in this study. This study included all stages of the disease, which

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might explain why the incidence rate is higher than previously suggested.

Age, gender and side

The mean age (± SD) at the time of diagnosis was 58.0 ± 17.9 years (range, 9–91 years). This is in line with the findings of earlier studies. Owen *et al.*[10] reported a mean age of 57 years in primary cases, and Vrabec and Chaljub[12] demonstrated a mean age of 61 years for spontaneous external auditory canal cholesteatoma cases. The wide age range, from 9 to 91 years, shows that external auditory canal cholesteatoma can be found in all ages.

Of the 95 external auditory canal cholesteatomas, 46 were in males and 49 were in females, showing an almost equal distribution between the genders.

The side of affliction showed a 52:43 left-to-right ratio, including 11 bilateral cases (11 per cent). Previously, both right-sided and left-sided predominance had been suggested;[10] however, this study demonstrates no predominant side of affliction.

Primary or secondary

Contrary to the findings of Dubach *et al.*,[1] the present study showed that primary ($n = 69$) was more common than secondary ($n = 26$) external auditory canal cholesteatoma. Lin found almost the same primary-to-secondary rate, with a 34:11 primary-to-secondary ratio.[13]

Location and aetiological factors

As shown in Figure 1, the canal floor of the external auditory canal was the most common location of external auditory canal cholesteatoma.

According to Dubach and Häusler, primary external auditory canal cholesteatoma only occurs in the inferior part of the external auditory canal; otherwise, there is a secondary reason or it is a complicated form of the disease.[5] The canal floor is the most common location, but we found that both primary and secondary external auditory canal cholesteatoma can also be located in the anterior and posterior part of the external auditory canal. The only difference between primary and secondary cases is the posterior wall, where we found it more often in the secondary cases. The canal roof is rarely affected.

Naim *et al.* suggested that hypoxia is pivotal for the establishment of external auditory canal cholesteatoma, and they detected a strong expression of angiogenic factors such as vascular endothelial growth factor in the external auditory canal cholesteatoma matrix.[11] We found that 68 per cent of the cases were smokers or former smokers; microangiopathy caused by smoking might therefore be an important pathogenic factor in the development of external auditory canal cholesteatoma. The canal roof is the best vascularised part of the external auditory canal. The inferior part of the external auditory canal is vulnerable to angiopathy, because of the poor blood supply to the area. Radiation therapy can cause thrombotic microangiopathy;[14] consequently, this can affect the vascularisation of the external auditory canal. This indicates that the auditory canal floor could be predisposed to develop external auditory canal cholesteatoma as a consequence of its poorer blood supply and because of microangiopathy caused by smoking or irradiation. Irradiation was the suspected cause in 58 per cent of the secondary cases. This supports the hypothesis that microangiopathy plays a role in the pathogenesis of external auditory canal cholesteatoma. The impetus of this should not be underestimated.

Trauma is the second most common cause of external auditory canal cholesteatoma, at 23 per cent. Jahnke and Lieberum have previously described it as a potential risk factor.[15] There is an apparent correlation between microtrauma, microangiopathy (caused by smoking, for example) and the development of external auditory canal cholesteatoma. In this study, 63 per cent of the primary and 54 per cent of the secondary cases were smokers. According to The Danish Cancer Society,[16] approximately 26 per cent of Danish people aged 50–59 years are smokers, showing a much higher rate of smokers in the case population. Smoking was not considered a secondary cause to developing external auditory canal cholesteatoma in this study. The correlation between smoking and external auditory canal cholesteatoma has to be investigated further, but the epidemiological data found in this study indicate an apparent association.

Presenting symptoms

Almost all patients had crust-covered lesions in the external auditory canal, and the majority had bone erosion as well. Several patients had more than one presenting symptom. The main presenting symptom, as showed in Table II, was hearing loss (25 per cent), followed by otalgia (23 per cent). Surprisingly, we found almost as many asymptomatic cases (22 per cent) as those presenting with otalgia. The hearing loss is most probably due to crust and debris in the external auditory canal. Prior studies showed that the most common symptoms are otalgia, hearing loss and otorrhoea.[1] In this study, we found that only 15 per cent of cases presented with otorrhoea. Dubach *et al.*,[1] Owen *et al.*,[10] Shin *et al.*[17] and Naim *et al.*[11] also described otalgia as a major symptom. These results clearly show that otalgia should be considered a major presenting symptom in the diagnosis of external auditory canal cholesteatoma.

Owen *et al.* suggested that the extension of external auditory canal cholesteatoma (advanced external auditory canal cholesteatoma) is related to the experience of otalgia.[10] Darr and Linstrom found that otalgia and hearing loss were the most common symptoms in advanced cases.[18] We identified eight advanced cases; of these, only two presented with otalgia, five had otorrhoea and five had hearing loss. Both of the cases with otalgia also had otorrhoea. This makes hearing loss another one of the main symptoms in advanced cases. Altogether, both hearing loss and otalgia should be considered the main presenting symptoms in the diagnosis of external auditory canal cholesteatoma.

Treatment

Treatment depends on the severity of symptoms and extension of the disease. During the 5-year study period, 41 cases had surgery and 48 cases were treated conservatively. All patients with advanced external auditory canal cholesteatoma underwent surgery.

Each case was specifically considered in terms of compliance, extension, symptoms and co-morbidities related to either surgery or conservative treatment. We found that 16 out of 21 asymptomatic cases (76 per cent) could be conservatively treated, and 15 out of 22 cases suffering with otalgia (68 per cent) needed surgery.

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At the time of analysis, 78 cases (82 per cent) had been discharged from the out-patient clinic; the rest were lost to follow up. They were either completely healed ($n = 38$) or referred to a private otolaryngologist ($n = 40$) for cleaning every three to six months. These latter patients had a residual cavity with or without full skin coverage in the external auditory canal, and, as these defects where of minor significance, they could be treated by a private otologist.

Of the patients discharged from the out-patient clinic, 46 per cent had undergone surgery and 54 per cent had been conservatively treated. The conservatively treated patients needed an average of 6 months' treatment, and the patients who needed surgery were healed an average of 3.2 months after surgery. The patients who underwent surgery typically had a more severe form of external auditory canal cholesteatoma and were often treated conservatively for a period before surgery was considered. These results show that surgery is a good treatment for this disease, with quicker and more complete healing, which should be carried out with no delay in most cases referred to hospitals. Nevertheless, it is important that the treatment is adapted according to the needs of patients, compliance and extension of the disease.

Diagnosis and pathogenesis

As stated in the introduction, we believe that benign necrotising otitis externa and external auditory canal cholesteatoma are in fact the same disease, which presents itself at different stages; that is, benign necrotising otitis externa in the beginning and external auditory canal cholesteatoma at the end. We suggest that the pathogenesis is as follows. It starts with necrosis of the bone and overlying skin in the external auditory canal. This is often not noted by the patient, and in many cases fresh skin grows under the sequestra, thus forming a skin-covered cavity. If the cavity stays clean, the condition has limited itself. If on the other hand debris starts to accumulate, infection might supervene. Immunological cells will be responsible for further bone resorption, resulting in what could look like a cholesteatoma.

Hence, we believe that the disease is a continuous process which might be diagnosed and treated at different stages of disease development. Therefore, we propose that the disease is in future called benign necrotising otitis externa and not external auditory canal cholesteatoma, since the latter is only the end product of the pathogenesis of the disease. We have been informed (personal communication) that British otologists are advocating the same change.

- This study indicates that external auditory canal cholesteatoma and benign necrotising otitis externa are the same disease at different stages
- It is suggested that external auditory canal cholesteatoma is henceforth known as benign necrotising otitis externa
- The incidence rate was 0.97 per year per 100 000 inhabitants
- Microangiopathy has a leading role in the pathogenesis of external auditory canal cholesteatoma
- The best treatment depends on disease extension, but surgery should be considered at an early stage
- Healing time for patients who underwent surgery was almost twice as fast as for those conservatively treated

Conclusion

External auditory canal cholesteatoma and benign necrotising otitis externa are rare otological diseases. We demonstrated a minimum incidence rate in the Capital Region of Denmark of 0.97 per year per 100 000 inhabitants, which is three times higher than that previously suggested by others. The presenting symptoms are hearing loss and otalgia. Smoking and irradiation are risk factors for developing external auditory canal cholesteatoma. Results from this study suggest that microangiopathy has a leading role in the pathogenesis of external auditory canal cholesteatoma. Treatment should be adapted to the patient's individual needs according to symptom severity, extension, co-morbidity and compliance. Healing time for the patients who underwent surgery was almost twice as fast as for those who were conservatively treated, indicating that surgery is a good treatment choice for this disease, and should be conducted in most patients referred to hospitals. We propose that the disease is in future called benign necrotising otitis externa.

Competing interests. None declared

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