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Original Article

Otogenic Intracranial Abscesses, Our Experience Over the Last Four Decades

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OBJECTIVE: To evaluate the predisposing factors for otogenic intracranial abscesses, assess their changes over time, and analyze how they differ from those due to other causes.

MATERIALS and METHODS: The medical records of all patients treated for otogenic intracranial abscesses, between 1970 and 2012 at a tertiary referral center, were retrospectively analyzed. The analysis included patient demographics, clinical characteristics, causative pathogens, treatments, outcomes, and comparisons of otogenic and non-otogenic intracranial abscesses.

RESULTS: Of all intracranial abscesses, 11% (n=18) were otogenic. In the 1970s, otogenic infections were a common predisposing factor for intracranial abscess; but within our study period, the incidence of otogenic intracranial abscesses decreased. Most (94%) otogenic cases were due to chronic suppurative otitis media and 78% were associated with cholesteatoma. Most patients (94%) had ear symptoms. The most common presenting symptoms were discharge from the infected ear (50%), headache (39%), neurological symptoms (28%), and fever (17%). The most common pathogens belonged to Streptococcus spp. (33%), Gram-negative enteric bacteria (22%), and Bacteroides spp. (11%). Neurosurgery was performed on all patients, 69% of which were prior to a later ear surgery. Surgery of the affected ear was performed on 14 patients (78%). A favorable recovery was typical (78%); however, one patient died.

CONCLUSION: Otogenic intracranial abscesses were most commonly due to a chronic ear infection with cholesteatoma. Ear symptoms and Gram-negative enteric bacteria were more common among patients with otogenic than non-otogenic intracranial abscesses.

KEYWORDS: Intracranial abscess, otitis, otogenic, complication

INTRODUCTION

A rare, but serious, complication of otitis media (OM) is intracranial abscess (IA) formation. Otogenic IAs usually develop from the contiguous spreading of a middle ear infection into the temporal bone and intracranial space either through the eroded bone of the tegmen tympani or of Trautmann's triangle, or via retrograde thrombophlebitis [1, 2]. In the pre-antibiotic era, intracranial complications of OM were common and often resulted in death [3].

Studies show that a common cause of IA formation is an otogenic infection [4]. Intracranial abscess formation may follow acute otitis media (AOM), but is more commonly associated with chronic suppurative otitis media (CSOM) [2, 5-7]. The development of more effective treatments for AOM and its complications has resulted in fewer chronic ear problems, at least in the developed countries [3, 5, 8-12]. The incidence of otogenic IAs has been decreasing [13-15].

The aim of this study was to evaluate the predisposing factors for otogenic intracranial abscesses, to assess changes in them over time, and to analyze how otogenic intracranial abscesses differ from those due to other causes.

MATERIALS and METHODS

Our group initially retrospectively analyzed the medical records of all patients treated for IAs at our institution from 1970 to 2012 (n=200) [15]. Our institution is a tertiary referral center and provides healthcare services to a population of 1.5 million; all operations for IAs are performed in the Department of Neurosurgery. Patient data from 1970 to 1989 came from patient journals saved on microfilm, and data from 1990 to 2012 came from the hospital's electronic database. International Classification of Diseases (ICD) version 9, code 324 was

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used for cases from 1990 to 1995, and ICD version 10, codes G00-G06, and G08, for cases from 1996 to 2012. Only intracranial, intra- and extra-axial abscesses were included. The methods and analysis of the entire study sample (n=166 IAs) have previously been published [15].

This study focuses on the 18 cases (11%) from that original sample that had a predisposing otogenic infection (otogenic IA). For these cases, we evaluated the underlying causes, clinical characteristics, causative pathogens, diagnostics, treatments, and outcomes of otogenic IA. Favorable recovery was defined as recovery from this infection without severe neurological deficits or health problems, and the ability to return to independent life. Additionally, comparisons of this smaller otogenic IA data are made to the data on non-otogenic IAs (n=148) [15].

Permission for this retrospective study was obtained from the Helsinki University Hospital, the local Ethics Committee approved the study protocol.

Statistical Analysis

Statistical analysis was performed (Statistical Package for the Social Sciences Statistics for Windows, Version 22.0, IBM Corp. Armonk, NY, USA, 2013). Chi-square test and Fisher's exact test, where appropriate, determined the significance between categorical variables; p-values of <0.05 were considered statistically significant. The Mann-Whitney U test evaluated the equality of medians between continuous variables.

RESULTS

Age, Demographics, and Medical History

Of the 18 otogenic IA patients, 16 (89%) were male. The mean age was 44 years (median 41, range: 7–74); two patients were children [15] (aged 7 and 16 years, both in 1972). The incidence of otogenic IAs peaked in 1970–1979 (n=10, 56%) (Figure 1) [15]. Most patients (61%) had more than one simultaneous underlying medical condition such as upper respiratory tract infection (n=3), dental infections (n=2), and prior scarlet fever (n=2). Three patients had a history of ear or head trauma unrelated to the IA. Ear surgery had been performed on three patients (Table 1). Chronic suppurative otitis media was common (18 patients, 94%) and 14 patients also had cholesteatoma. The predisposing ear infection was on the right side in 12 cases (67%) and the rest were on the left side. The ear infection and IA were ipsilateral in 17 cases (94%). In a child with Fallot tetralogy and a simultaneous dental infection, the ear infection and IA were contralateral. Seven patients (39%) had received antibiotic treatment for this infection prior to hospitalization. Ten patients were treated at other (smaller) hospitals initially and the rest were seen in our hospital as their first point of contact.

Symptoms

The mean duration of any symptom, prior to hospitalization, was 17 days. Ear symptoms were present in 17 patients (94%); these included discharge from the infected ear (n=9), and infection of the external ear canal (EAC) (n=2). Headache was a common first symptom (n=7, 39%), three patients had fever. Some patients presented with neurological symptoms (n=5) such as convulsions (n=2) and altered consciousness (n=1); however, none had paresis. All patients had some neurological symptom during the hospital treatment period; these included drowsiness (n=13, 72%), mental confusion (n=9, 50%), hemiparesis (n=6, 33%), dysphasia (n=5, 28%), unconscious-

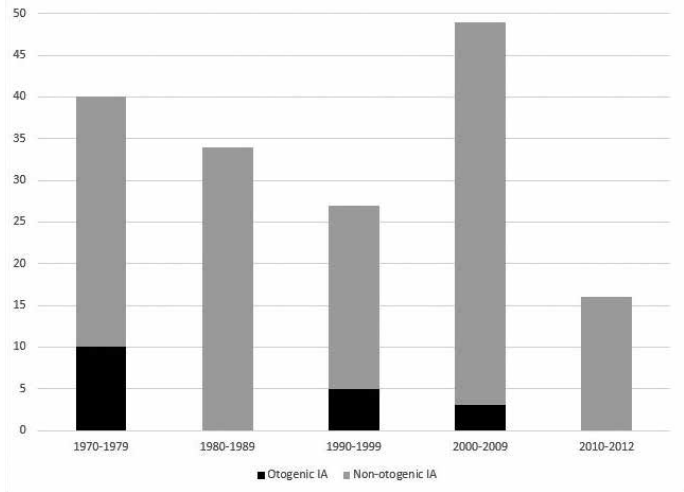

Figure 1. Otogenic and non-otogenic intracranial abscesses (IA) over time.
Y-axis: number of cases, X-axis: period of time

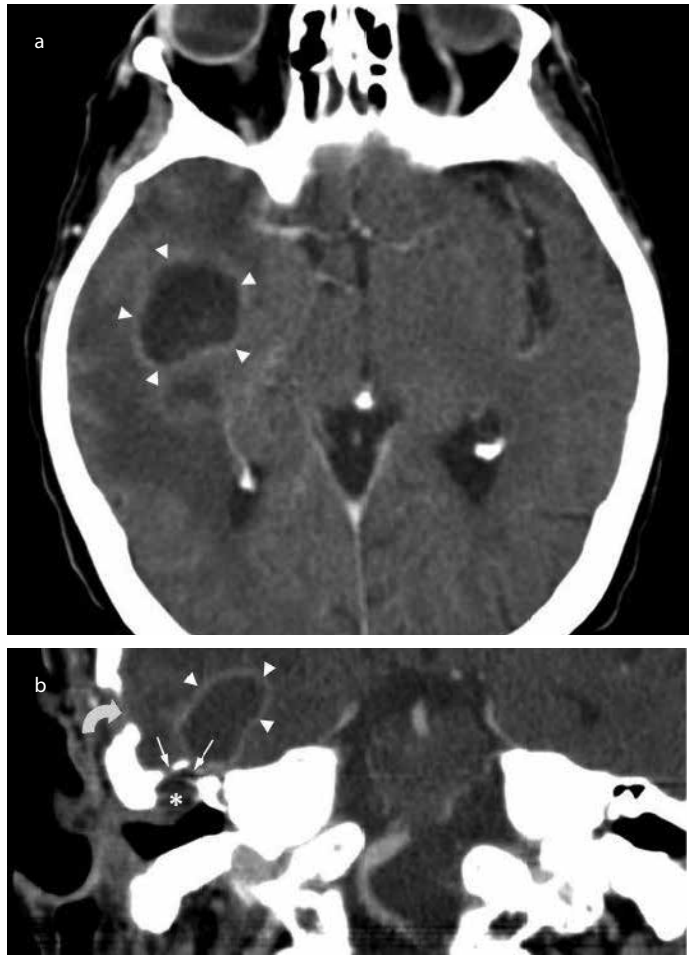

Figure 2. a, b. Axial contrast-enhanced CT image of the brain (a) and coronal contrast-enhanced CT image of the temporal bone (b) showing an otogenic intracranial abscess of the right temporal lobe. The abscess has an enhanced wall (arrowheads) and is surrounded by marked vasogenic edema obliterating the cortical sulci and the temporal horn of the right lateral ventricle. The underlying cholesteatoma (asterisk) has caused bony erosions (arrows) toward the middle cranial fossa. Note that the temporal bone CT (b) was performed after initial punction of the abscess (post-trepanation defect marked with curved arrow), which alone, without curing the underlying COM with cholesteatoma, has been insufficient as a surgical treatment.
CT: computed tomography

Table 1. Medical history, type of otitis, and symptoms of the patients with otogenic intracranial abscesses

Case	Decade	Prior ear surgery, latency	Type of otitis	Cholesteatoma	Ear symptoms, duration	Acute symptoms began	Other related diagnosis	Diagnosis of ear infection	IA diagnosed	First treatment of IA
1	1970	None	COM	Yes	Prior ear trauma. Discharge, years.	Jan 31	Feb 1: meningitis	Feb 3	Feb 3	Feb 3
2 *	1970	None	NA	No	Otitis, duration unknown.	Oct 20	-	Oct 25	Oct 25	Oct 26
3 *	1970	None	COM	Yes	Discharge, prolonged.	Aug 8	Aug 11: meningitis	Aug 11	Aug 14	Aug 15
4	1970	None	COM	Yes	Chronic ear problems, 6 years. Hearing problems.	Jun 1	Jun 26: meningitis	Jun 1	Jul 11	Jul 12
5	1970	None	COM	Yes	Discharge, duration unknown.	Aug 25	Sep 4: meningitis	Oct 8	NA	Oct 17
6 †	1970	None	COM	NA	Discharge, years.	Jun 23	-	Jul 14 Obduction post mortem	Jul 7	Jul 7
7	1970	None	COM	Yes	Discharge, years.	Mar 21	Mar 28: meningitis	Mar 21	Apr 9	Apr 10
8	1970	Radical mastoid operation, 4 years. Skin graft to mastoid cavity, 3 years.	COM	No	Discharge, years.	Jan 22	-	Jan 22	Jan 24	Jan 26
9	1970	None	COM	Yes	Discharge, years. Deaf ears.	May 15	Jun 25: meningitis	Jun 14	Jul 2	Jul 3
10	1970	None	COM	Yes	Mild chronic otitis, duration unknown.	Oct 20: mild, Nov 10: severe	-	Oct 20	Nov 25	Nov 25
11	1990	None	COM	Yes	Mild chronic, 10 years.	Jan 21	Jan 31: hydrocephalus, ventriculostomy Feb: shunt	Jan 29	Feb 20	Feb 21
12	1990	None	COM	Yes	Discharge, years. Hearing problem, TM perforation.	Oct 18	Oct 18: meningitis	Oct 20	Oct 22	Nov 25
13	1990	None	COM	Yes	Missing information regarding long term symptoms.	Feb 15	Feb 15: intratemporal lesion	Feb 15	Feb 15	Mar 4
14	1990	None	COM	Yes	Discharge, prolonged.	Nov 20	-	Nov 20	Jan 3	Jan 5
15	1990	None	COM	Yes	Traumatic TM perforation in childhood. Missing information regarding long term symptoms.	Sep 1: mild, Sep 13: severe	-	Sep 1	Sep 13	Sep 13
16	2000	Stapedoplasty l.a, 20 years	COM	Yes	Recent diagnosis of external otitis.	Aug 15	-	Sep 7	Sep 3	Sep 4
17	2000	None	COM	NA	Ear symptoms, duration unknown.	Jan 10	Jan 13: temporal lesion, considered a glioma	Jan 16	Jan 29	Jan 29
18	2000	Myringoplasty l.sin, 30 years ago	COM	Yes	Mild chronic, duration unknown.	Nov 7: AOM Nov 13: headache, Nov 15: convulsion	-	Nov 7	Nov 16	Nov 16

Missing information: NA; child: *; COM: chronic otitis media; AOM: acute otitis media; TM: tympanic membrane; IA: intracranial abscess; died due to abscess: †

ness (n=4, 22%), convulsions (n=2), vertigo (n=2), and facial nerve paresis (n=1). Seven patients (39%) had concomitant meningitis (Table 1). Other symptoms or conditions during hospital treatment included headache and/or fever (n=11, 61%), nuchal rigidity (n=6, 33%), and papillary stasis (n=5, 28%).

Microbiology, Laboratory Tests, and Radiological Examinations

The mean preoperative leukocyte count was 13 (median 12, range 7–24), and the mean preoperative C-reactive protein (CRP) was 87 (median 85, range <5–158). Imaging was performed on all patients including computer tomography (CT) (n=10), angiography (n=8),

Table 2. Abscess location, imaging, and surgery of patients with otogenic intracranial abscesses

Case	Location of brain abscess	Imaging	Imaging findings	Ear operation due to abscess	Operative findings	Neurosurgery before ear surgery
1	Temporal	A+B	Temporal expansion	Feb 3: RMO and temporal abscess canalization	Antral bone defect, dural exposure, dura covered by thick pus. Cholesteatoma.	No (simultaneous)
2 *	Parietal	A	Parietal expansion	None	-	No
3 *	Temporal	A	Temporal expansion, large	Aug: RMO and evacuation of sigmoid sinus thrombosis	Exposed tempora dura, cholesteatoma and pus in sinusdura-angle, thrombosis and pus in sigmoid sinus.	No
4	Temporal	A+B	Temporal expansion	Jun 6: RMO and facial nerve nudation	Jun 6: Attic cholesteatoma, necrosis at the roof of EAC. Jun 17: Craniotomy: 2 abscesses in temporal lobe, fistula to EAC.	No
5	Temporal	A+B	Temporal expansion, large	Oct 9: RMO	Temporal dura and sigmoid sinus exposed, fistula to horizontal semicircular canal. Antral and mastoid cholesteatoma, erosion of the EAC.	No
6†	Intracerebellar	A	Hydrocephalus	None	Obduction post mortem: Intracranial spreading of middle ear infection. Continuation of cerebellar abscess under tentorium to the middle ear.	No
7	Temporal	A	Suspicion of temporal expansion	Mar 28: RMO	Attic and supralabyrinthine cholesteatoma, antral abscess formation. Middle fossa dura exposed, pus discharge through tegmen.	No
8	Temporal	A	Temporal abscess	Nov 8: Revision of mastoid operation	The first mastoid operation 4 years earlier: temporal dura exposed in the antral roof. 8 Nov: Granulation, bony defect in tympanic tegmen and antrum. Fistula from tegmen tympani into the intracranial space and EAC.	Yes
9	Temporal	CT	Temporal intracranial abscess	Jun 30: RMO	Cholesteatoma and bone erosion of the EAC, polyps and granulation tissue. Bone erosion, temporal dura exposed; purulent effusion and prolapse of brain tissue through the dural defect.	No
10	Occipital	CT	Occipital abscess	Nov 27: RMO	Cholesteatoma in antral aditus. Abscess formation through the mastoid cavity over and into the EAC. Erosion of bony EAC. Antral dura exposed and abnormally thick.	Yes
11	Intracerebellar and occipital	CT	Hydrocephalus, bilateral occipital abscesses (sic!), connection to the right mastoid cavity.	May 15: RMO	Antral and attic cholesteatoma spreading into Trautmans triangle. Thick granulation tissue on top of the sigmoid sinus. Posterior fossa dura abnormally thick with granulations.	Yes
12	Temporal	CT	COM, tegmen tympani defect, cholesteatoma, destruction of auditory ossicles. Temporal intracranial abscess, subdural empyema. ±	Oct 13: Tympanomastoidectomy	Large mastoid cholesteatoma, exposed dura covered by infectious tissue.	No
13	Temporal	CT	Brain CT: large temporal expansion (tumor or abscess)	Mar 10: RMO	Antral cholesteatoma. Erosion of tympanic tegmen with granulations and irritation of exposed dura.	Yes
14	Temporal	CT	Non-aerated left ear, hypodense lesion in left temporal lobe. No enhancing abscess. ±	Jan 10: RMO	Antral cholesteatoma with bone erosion and exposed dura in the middle fossa.	Yes
15	Temporal	CT	COM, EAC, and antral cholesteatoma, two fistulas into intracranial space (middle- and posterior fossa). Temporal abscess, impaired CSF flow. §	Oct 6: RMO	Bone erosion in tympanic tegmen with cholesteatoma spreading between bone and dura.	Yes
16	Temporal	CT±MRI	COM, EAC cholesteatoma, fistulation into middle fossa, two fistulas; one through the roof of the EAC and one through tegmen. Temporal abscess. §	None	-	Yes
17	Temporal	CT±MRI	COM, no bone destruction. Temporal lesion, tumor suspicion. §	None	-	Yes
18	Temporal	CT±MRI	Cholesteatoma above EAC, fistula into the middle fossa. §	Nov 17: RMO	Large mastoid cholesteatoma fills the anterior epitympanum and continues to the horizontal semicircular canal. Erosion of the bony EAC. Defect in tympanic tegmen with granulation of the dura.	Yes

Child: *; brain and ear: ±; Imaging re-evaluated by radiologist in 2015: §; COM: chronic otitis media; CT: computed tomography; MRI: magnetic resonance imaging; A: angiography; B: brain scintigraphy; EAC: external auditory canal; RMO: Radical mastoid operation; died due to abscess: †

brain scintigraphy (n=3), and magnetic resonance imaging (MRI) (n=2). Computer tomography of the ear was performed on three patients (Figure 2, Table 2). Most abscess were singular (n=17, 94%), though in one case, three abscesses were found. Usually abscesses were located temporally (n=14, 78%) [15]. A sample of pus was taken from all patients for microbiological culturing. Most cultures were mono-bacterial (n=10, 56%) while five (28%) were poly-bacterial. The pathogens have been reported previously [15]; the most common pathogens belonged to *Streptococcus* spp. (n=6, 33%) and Gram-negative enteric bacteria (n=4, 22%) [15]. Bacteria belonging to *Bacteroides* spp were found in two cases [15]. *Fusobacterium* spp, *Haemophilus* spp, and *Peptostreptococcus* were found in one case each [15]. Of the three patients with negative bacterial cultures, [15] two had received prior antimicrobial treatment.

Treatment

Preoperative antibiotic treatment was administered to 15 patients (83%) and preoperative steroids to six patients (33%). Surgery of the IA was performed on all patients including punction (n=14), craniotomy (n=7), extirpation (n=4), and ventriculostomy (n=2). The number of neurosurgical operations ranged from one to four (mean of two). The first approach was punction for 14 patients (78%), craniotomy on three (17%), and ventriculostomy on one patient. Neurosurgical reoperation was necessary for 12 (67%) patients; these procedures included punction (n=6), craniotomy (n=5), and ventriculostomy (n=1). The mean time interval between the first and last neurosurgery was 16 days (range 5–34 days). Surgical treatment for the affected ear was also necessary for 14 patients (78%): 13 radical mastoid operations and one mastoid revision operation (Table 2). The mean total amount of collected pus was 52 mL (range 13–164 mL, missing n=4). Irrigation of the abscess cavity was performed with saline only on nine patients (50%), and with antimicrobials on six patients (33%). All patients received postoperative intravenous antibiotic treatment. Of these, the most common were metronidazole (n=8), third-generation cephalosporins (n=8), penicillin (n=7), ampicillin (n=5), and tetracycline (n=4). The mean duration of postoperative intravenous antibiotic treatment was 23 days (range 3–60 days, missing n=5).

Outcome of the Patients

Of the 17 patients with recovery data, a favorable recovery was seen in 14 patients (78%), two patients failed to recover well (one died soon after treatment of the IA due to cardiac problems), and one patient died due to the IA (in 1976, this patient also had simultaneous pneumonia). Although most patients had favorable recoveries, some had remaining health problems including epilepsy (n=3), hemiparesis (n=2), dizziness (n=2), hemianopia (n=2), psychological alteration (n=1), and hydrocephalus (n=1).

Comparison of Otogenic and Non-Otogenic Intracranial Abscesses

Of all IAs in the study, 11% were otogenic [15]. The proportion of otogenic IAs was largest in 1970–1979 (of 40 IAs, 10 were otogenic, p=0.002) (Figure 1) [15]. There were no significant differences when comparing patient age or sex among otogenic and non-otogenic IAs. Prior ear surgery was more common in patients with otogenic IAs than non-otogenic (22% vs. 1%, p=0.001, respectively). Concurrent meningitis was more common in patients with otogenic IAs (39% vs. 7%, p=0.001, respectively). There were no significant differences when comparing duration of prior symptoms, but most otogenic IA patients had ear symptoms (94%) while none of the non-otogenic cases did. Paresis as a first symptom was, however, was not seen in patients with otogenic IAs, but was present in 19% of non-otogenic cases. Regarding symptoms over the course of treatment, patients with otogenic IAs had more drowsiness (72% vs. 39%, p=0.010, respectively). There were no significant differences between IA types when comparing the laboratory parameters. Angiography was more common in patients with otogenic IAs (44% vs. 20%, p=0.034, respectively), while CT (56% vs. 81%, p=0.029, respectively) and MRI (11% vs. 40%, p=0.019, respectively) were both less common in patients with otogenic IAs. Gram-negative enteric bacteria species were more common in patients with otogenic IAs (22% vs. 3%, p=0.005, respectively) [15]. Temporal abscesses were more common in patients with otogenic IAs (78% vs. 12%, p<0.001, respectively) [15]. There were no significant differences regarding the method of operative treatment for the IA. However, those with otogenic IAs, needed multiple neurosurgeries (67% vs. 35%, p=0.018, respectively) more often. The need for more than one neurosurgery was more common in the beginning of our study period than in the latter (58% in 1970–1989 vs. 33% in 1990–2012, p<0.001). No significant differences emerged regarding the outcomes of patients with otogenic and non-otogenic IAs.

DISCUSSION

In agreement with previous studies, most patients presenting with otogenic IAs were middle-aged men [1]. Children were a clear minority [5], only presenting at the beginning of the study period. In the 1970s, otogenic infections were a common predisposing factor for intracranial abscess formation and 25% of all IAs were otogenic [15]. Otogenic infections became less common as a predisposing condition for IAs within our study period, also in accordance with previous data [14]. Otogenic IAs were completely absent from 1980 to 1989 and during the last twelve-year period, only 5% of all abscesses were otogenic [15]. Our institution treated 99 patients with an otogenic brain abscess from 1930–1960 and only three cases from 1961 to 1969 [14].

In a recent Danish study, five of seven otogenic brain abscesses were due to AOM [16]. In our otogenic subsample, almost all IAs were associated with CSOM, particularly CSOM with cholesteatoma. The incidences of cholesteatoma, and its operative treatment, have decreased over time in our country [17]. The annual risk of an adult with active CSOM developing an IA is 1:10000 [18], and the lifetime expectancy of a 30-year-old patient with active CSOM developing an abscess is 1:200 [18]. The treatment of OM has changed over time. In the 1970s, paracentesis of the tympanic membrane was recommended for all patients with middle ear secretions and hearing problems [19]. However, later guidelines no longer recommend paracentesis for uncomplicated cases of OM [20, 21]. These changes in the treatment of cholesteatoma, CSOM, and AOM, have occurred within our study period, and may explain the changes in otogenic IA incidence over time. The absence of otogenic IAs from 1980 to 1989 is surprising. Within this time period, patients with OM were actively treated, [19] which may have reduced the number of complications. It is also possible that some patients are missing from this sample; for example, if an otogenic epidural abscess caused only mild symptoms and the patient required only ear surgery, resulting in simultaneous resolution of the undiagnosed epidural abscess, the case would not have been seen in the neurosurgery department.

Prior ear surgery was, surprisingly, uncommon in our subjects, but was more common in otogenic than non-otogenic IAs. Ear symptoms were common in our otogenic IA patients; discharge from the infected ear was seen in half of the patients and some patients also had signs of infection of the EAC. Paresis, while seen in non-otogenic cases, was not seen in otogenic IAs.

Leukocyte count and CRP values were only moderately elevated in otogenic cases, possibly due to the infection being localized. The most common pathogens belonged to *Streptococcus* spp. and Gram-negative enteric bacteria, the latter were more common in patients with otogenic IAs than non-otogenic [15]. The bacteriology of CSOM is known to differ from that of AOM [10, 22]. In CSOM, with or without cholesteatoma, prolonged infection caused by typical pathogens may lead to osteitis, bone erosion, and may cause the infection to spread further [1, 23].

Many patients in our material had CSOM, often associated with cholesteatoma, and wide areas of bone erosion. However, obliteration of the mastoid cavity was not common in this group of patients. Bony erosions were typically seen in the cranio-temporal part of the EAC and toward the middle fossa. Along these bony erosions, dura was exposed, infected, and often covered by granulations; infection was also often seen in the mastoid and the aditus to mastoid antrum. Thus, management of CSOM to avoid prolonged infections and their complications is important.

Concomitant meningitis was more common in patients with otogenic than non-otogenic IAs. Otogenic IAs usually develop via the contiguous spreading of a middle ear infection through eroded bone, and through the meninges into the intracranial space [1, 2, 15, 24]. In our material, because of contiguous spreading of the infection, the underlying otitis and IA were usually ipsilateral, and most otogenic IAs were singular and temporally located.

Many of our patients presented with severe neurological conditions, and were initially examined due to these conditions, with the underlying otological condition being diagnosed later. We also noticed that the diagnosis of the underlying ear infection was delayed in some cases (Table 1). These diagnostic patterns were also seen in prior literature [16]. If an intracranial complication of otitis is suspected, imaging should be performed [25]. Possible underlying otogenic infection should be suspected, ruled out, and treated in all IAs, especially if situated in typical locations of otogenic origin.

Imaging was performed on all of our patients, but the imaging technique varied over time (Table 2). Because most otogenic IAs occurred in the beginning of the study period, when CT and MRI were not available, angiography was more common in these patients. Advancements in imaging techniques have had a positive effect on the prognosis of patients with IAs [26]. Brain CT is widely available nowadays and is often used in the initial diagnostics for neurological conditions; nowadays, it may also be used for patients with otogenic IAs [27]. When otogenic infection is suspected, a CT scan of the ear is often performed because CT is efficient in evaluating bony structures [28, 29]. Additionally, MRI has proved valuable for locating otitic intracranial or extracranial complications [28].

Surgical treatment of the IA was performed on all patients with punction being the most common procedure. Most patients required multiple surgeries; the need for multiple neurosurgical procedures was more common in the beginning of the study period when most otogenic IAs occurred and diagnostic tools were not as advanced. All patients received postoperative antimicrobial treatment, often metronidazole and third-generation cephalosporins, penicillin, or ampicillin.

Surgical treatment of the infected ear, usually radical mastoidectomy, was performed in 78% of the cases. Typically, neurosurgery was performed before ear surgery. The departments of neurosurgery and otorhinolaryngology are located in different buildings of our hospital with critically ill patients usually being taken to the neurosurgery department first. The duration of the neurosurgical procedure is often short; however, simultaneous ear surgery would lead to longer procedures that these critically ill patients would not always be able to tolerate.

A favorable recovery was seen in most patients. Only one patient, in 1976, died due to the otogenic IA. No significant differences emerged in the outcome of patients with otogenic compared to non-otogenic IAs.

CONCLUSION

Although CSOM and cholesteatoma have become rare in developed countries [17], they still exist and complications are possible. The modern, efficient, treatment of OM, fortunately, seems to have resulted in less otogenic IAs. However, this serious complication must not be forgotten. With increasing immigration, the number of patients with CSOM may rise in developed countries. The typical otogenic IA patient, according to our findings, had a chronic ear infection, cholesteatoma, and prolonged secretions of the ear. Chronic infection results in bone erosion and exposure of the dura, which allows bacterial infection to easily spread through the meninges into the intracranial space, causing an abscess, often in the temporal lobe.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Helsinki University Hospital.

Informed Consent: Permission for this retrospective study was obtained from the Helsinki University Hospital.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - G.B., J.J.; Design - G.B., J.J., K.L.; Supervision - A.A., A.M., G.B., J.J.; Resources - A.A., G.B., J.J.; Materials - A.A., G.B., J.J.; Data Collection and/or Processing - A.L.H, L.L., K.L., R.S.; Analysis and/or Interpretation - A.A., G.B., J.J., A.L.H., L.L., K.L., A.M., R.S.; Literature Search - A.A., A.L.H., J.J.; Writing Manuscript - A.A., A.L.H., J.J.; Critical Review - G.B., L.L., K.L., A.M., R.S.

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