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HEAD AND NECK

Comparison of MR imaging findings in paediatric and adult patients with acute mastoiditis and incidental intramastoid bright signal on T2-weighted images

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Abstract

Objectives To compare MR imaging features in patients with incidental mastoid T2-hyperintensity with those of clinical acute mastoiditis, to ascertain characteristic differences between them.

Methods MR images of 35 adult and paediatric patients with clinical acute mastoiditis and 34 consecutive age-matched controls without relevant middle ear pathology and with incidental T2-hyperintensity that covered≥50 % of the mastoid were retrospectively analysed with regard to signal, diffusion, and enhancement characteristics, and presence of complications.

Results Incidental mastoid T2-hyperintensity that covered≥50 % of the mastoid volume was found in 4.6 % of reviewed MR scans (n=2341), and associated significantly ($p<0.05$) less with the involvement of the tympanic cavity (38 % vs. 74 %) and mastoid antrum (56 % vs. 80 %), hypointense-to-CSF signal intensity on T2 FSE (6 % vs. 86 %), intramastoid diffusion restriction (0 % vs. 62 %), intense intramastoid enhancement (0 % vs. 51 %), periosteal enhancement (3 % vs. 69 %), perimastoid dural enhancement 3 % vs. 43 %), bone destruction (0 % vs 49 %), intratemporal abscess or cholesteatoma (0 % vs. 24 %), labyrinth involvement (0 % vs. 14 %), and extracranial abscesses (0 % vs. 20 %).

Conclusion Hypointense-to-CSF signal intensity on T2WI, restricted diffusion, intense intramastoid enhancement among other MR imaging characteristics favoured an acute mastoiditis diagnosis over clinically non-relevant incidental mastoid pathology.

Key Points

- *Intramastoid T2-hyperintensity alone is not a reliable sign for acute mastoiditis.*
- *In acute mastoiditis, intramastoid T2-weighted signal intensity is usually hypointense to CSF.*
- *Diffusion restriction and intense intramastoid enhancement are absent in incidental mastoid effusion.*
- *An ADC value≥1.72× 10^{-3} mm^2/s contradicts the AM diagnosis.*

Keywords Mastoiditis · Otitis media · Middle ear · Temporal bone · Magnetic resonance imaging

Abbreviations

AM	acute mastoiditis
SI	signal intensity
ROC	receiver operating characteristic
AUC	area under the curve
SNHL	sensorineural hearing loss
CSF	cerebrospinal fluid

Introduction

Acute mastoiditis (AM) is a feared complication of otitis media in which purulent infection involves the mastoid air cells.

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Infection may cause osteolysis of the bony septa or cortical bone (coalescent mastoiditis), which can lead to intracranial and extracranial complications [1].

AM most commonly affects children, and in this age group, it typically has a distinct clinical picture (classical AM) [2]. In adults, the so-called latent AM prevails, which has a slow course and less pronounced symptoms [3, 4].

The diagnosis of AM is made clinically. Imaging is mainly useful for detecting complications, but may be requested to confirm the suspicion of latent AM, rule out other disease entities that may mimic AM, or characterize the severity of mastoid involvement in patients that have responded unsatisfactorily to conservative treatment, to evaluate the need for surgery [5, 6].

A sensitive sign of severe mastoid infection in computed tomography (CT) is osteolysis. Unfortunately, evidence for clinically relevant magnetic resonance (MR) imaging signs of mastoid infection is scarce. Evaluation of bone involvement is difficult to determine by MRI, and other signs should be considered.

The most striking imaging sign of middle ear and mastoid inflammation by MRI is the bright signal on T2-weighted images that reflects a middle ear effusion and mucosal oedema. Unfortunately, this is also a fairly common incidental finding in head MRI scans [7–9]. Intramastoid T2 hyperintensity alone is unreliable for making the diagnosis of mastoiditis because it yields many wrong positive diagnoses in radiological imaging, which have lead to unnecessary consultations and treatments [10, 11]. For example, Polat et al. conducted a study involving 406 patients with such incidental radiological diagnoses of mastoiditis, but otological infectious disease was clinically verified in only 17 % of their 275 patients with known otological status, and none of them had clinical mastoiditis [11].

In our previous work, we have shown that several features of AM, revealed by MR imaging characteristics, such as decreased signal intensity (SI) on T2WI, and intramastoid diffusion restriction and enhancement, were present in most of AM patients [12]. Similar results were obtained by Platzek et al. [13].

The purpose of this study was to compare the MR imaging features of patients with clinical AM and those with incidental mastoid T2-hyperintensity to ascertain any characteristic differences.

Methods

Formal consent was not required for this type of retrospective study.

Patients

Consecutive patients with a clinical discharge diagnosis of AM (ICD-10 code H70.0) recorded in the hospital database from January 2003 to December 2013 were identified, and those with available MR scans of the head or temporal bone (n=38) were enrolled into the study. Recurrent disease after previous mastoidectomy (n=1), secondary inflammation due to an underlying tumour (n=1), and underlying middle-ear sarcoidosis (n=1) were excluded. The final AM group comprised 35 patients (13 males and 22 females) of age range 2–81 years, (mean, 31.5 years). Paediatric patients (2–16 years of age) numbered 11 (31.4 %). Mastoidectomy was performed for 24 patients (68.6 %).

The control group comprised 34 patients (16 male and 18 female) of age range 2–82 years (mean, 37.3 years) with incidental T2-hyperintensity that covered≥50 % of the mastoid, but without AM or relevant middle ear pathology. The MR scans of the control group were found by screening 2341 consecutive MR scans of head or temporal bone for hyperintense signal covering≥50 % of the mastoid volume on T2-weighted images by the first author of this paper. We separately evaluated 1498 MR scans for any evidence of intramastoid T2-hyperintensity. Inclusion and exclusion criteria for the control patients are given in the flowchart in Fig. 1, and their otological and infectious statuses are given in Table 1.

MR imaging protocol

MR scans were performed at a 1.5 T Avanto unit (Siemens, Erlangen, Germany) with a 12-channel head and neck coil in 28 AM patients and 24 controls, at a 1.5 T Sonata unit (Siemens, Erlangen, Germany) with a 8-channel head and neck coil in five AM patients, and at a 1.5 T Achieva unit (Philips Healthcare, Best, Netherlands) with an 8-channel head coil in two AM patients and ten controls. Imaging characteristics were estimated from axial and coronal T2 FSE and axial T1 spin-echo images, axial EPI-DWI with b factors of 0 and 1000 s/mm^2, and an ADC map with 3-mm section thickness, and high-resolution T2-weighted CISS images with 0.7-mm section thickness. Contrast enhancement was estimated from T1-weighted MPRAGE images with 1-mm section thickness and / or T1-weighted spin-echo images with 3-mm section thickness after intravenous administration of 0.1 mmol per kg of body weight gadoterate meglumine (Dotarem; Guerbet, Aulnay-sous-Bois, France).

Image interpretation

The MR images were independently analysed from a PACS by two board-certified head-and-neck radiologists with 7 and 4 years of experience, respectively, in reading temporal bone

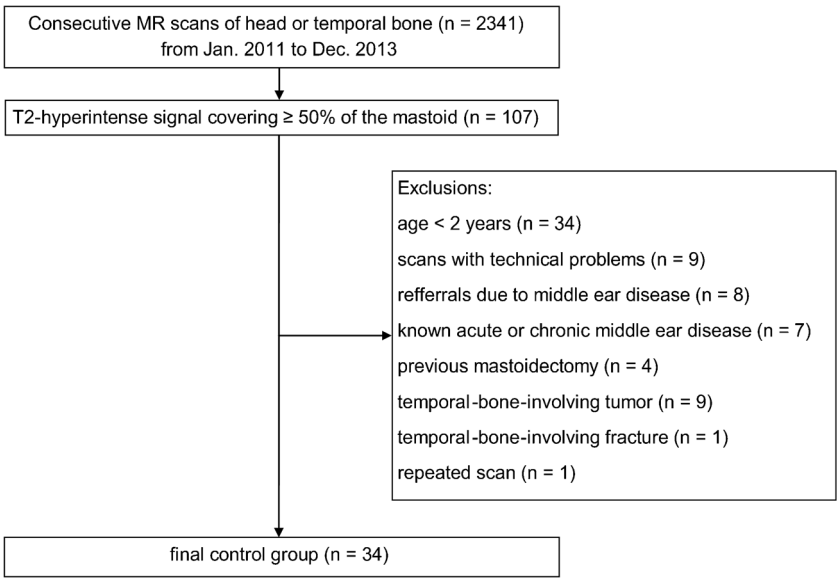

Fig. 1 Flowchart of the control group selection

MR images. Discrepancies were resolved by means of an additional joint-reading session. Only one ear was evaluated for each patient (the first-involved ear in one patient with bilateral AM, and the side with more involvement in seven controls with bilateral incidental T2-hyperintensity). Imaging findings were estimated as described in Table 2.

Mastoid volume was estimated subjectively, based on the experience of the readers. In general, mastoid volume was reported as "reduced" when only small-sized periantral cells were present, if any.

The signal intensities (SI) of intramastoid contents on T2 FSE were compared to the SI of CSF; and on CISS to the SI of the brain parenchyma, by visual evaluation [12]. Diffusion was considered to be restricted when the intramastoid SI on DWI was higher than that of CSF, and the ADC map showed a signal drop. Mean ADC values of intramastoid contents were calculated from measurements of three different ROIs, placed on non-aerated mastoid areas, each in a different section (Figs. 4d and 5d).

Bone destruction was estimated at three subsites (the intercellular septa, and the inner and the outer cortical bony table) from T2 FSE images as loss of morphological integrity, or signal transformation inside the otherwise signal-voided cortical bone. All suspicious and definite changes were reported as pathological.

Signs of labyrinth involvement were either diffuse intralabyrinthine enhancement or perilymph signal drop on CISS. Intratemporal abscess was defined as a non-anatomical collection with restricted diffusion and an enhanced wall. The same criteria may also apply for a cholesteatoma; therefore, these two entities were estimated together as "cholesteatoma or abscess" with no distinction made between them.

Criteria for generalized pachymeningitis (in contrast to perimastoid dural enhancement) were extensive thickening and enhancement of the dura that extended past the borders of the temporal bone.

Table 1 Referring units and otological and infectious background status of the control patients

Otological and infectious status of the control patients (n=34)	No.
No infection or otological disease	13
Infection other than otological	14
Acute middle ear infection	0[a]
Chronic middle ear infection	0[a]
Other, non-infectious otological disease	7
Referring units of the control patients	
Neurology	12
Neurosurgery	3
Oncology and radiotherapy	9
Otorhinolaryngology	9
Internal medicine	1

Note: [a] Patients with acute or chronic middle ear infection were excluded from the controls

Statistical analysis

Differences in gender and in qualitative imaging findings of AM patients and controls were assessed by Fisher's exact test; the distribution of the differences in patients' ages and in their ADC-values were assessed non-parametrically by the Mann-Whitney U-test. A receiver operating characteristic (ROC) analysis with multiple logistic regression was used to calculate the area under the curve (AUC) for ROC to find the optimal ADC threshold for predicting

Table 2 Comparative MRI findings in patients with clinical acute mastoiditis (AM) and in patients with incidental intramastoid T2 hyperintensity (controls)

Imaging findings	AM No. (%)	(n)	controls No. (%)	(n)	*p*-value	Kappa coefficient
Reduced mastoid volume	2 (6)	35	10 (29)	34	0.012	0.70
T2 hyperintensity coverage of temporal bone subsites						
≥50 % of the tympanic cavity	26 (74)	35	13 (38)	34	0.004	0.80
≥50 % of the mastoid antrum	28 (80)	35	19 (56)	34	0.041	0.78
(Near) Total coverage of mastoid	27 (77)	35	20 (59)	34	0.126	0.93
Signal intensities and diffusion characteristics of intramastoid contents						
Hypointense to CSF on T2 FSE	30 (86)	35	2 (6)	34	<0.001	0.80
Hypointense to brain on CISS	11 (39)	28	0 (0)	2	0.520	0.55
Diffusion restriction[a]	18 (62)	29	0 (0)	27	<0.001	0.64
Enhancement characteristics						
Intense intramastoid	18 (51)	35	0 (0)	29	<0.001	0.63
Periosteal	24 (69)	35	1 (3)	29	<0.001	0.80
Perimastoid meningeal	15 (43)	35	1 (3)	29	<0.001	0.57
Bone destruction						
Mastoid septa	12 (34)	35	0 (0)	34	<0.001	0.50
Inner cortex	4 (11)	35	0 (0)	34	0.114	nc
Outer cortex	10 (29)	35	0 (0)	34	0.001	0.80
Bone destruction in general	17 (49)	35	0 (0)	34	<0.001	0.63
Complications						
Abscess or cholesteatoma[b]	8 (24)	34	0 (0)	34	0.005	0.68
Labyrinth involvement	5 (14)	35	0 (0)	29	0.058	0.37
Extracranial abscess	7 (20)	35	0 (0)	34	0.011	1.00
Pachymeningitis	2 (6)	35	1 (3)	29	1.000	0.55
Leptomeningitis	2 (6)	35	0 (0)	29	0.497	0.66
Epidural abscess	3 (9)	35	0 (0)	34	0.239	1.00
Subdural empyema	0 (0)	35	0 (0)	34	nc	nc
Sinus thrombosis	3 (9)	35	1 (3)	31	0.616	1.00

Note: [a] diffusion restriction = intramastoid signal intensity on DWI higher than of CSF + signal drop in ADC; [b] intratemporal abscess or cholesteatoma

clinical AM in mastoid cavities with bright signal on T2WI, along with corresponding sensitivities, specificities, and positive and negative predictive values. Inter-rater agreement was measured with Cohen's kappa statistic. Statistical analyses were conducted with NCSS 8 software (NCSS LLC, Kaysville, UT). *p* values less than 0.05 were considered statistically significant.

Results

Prevalence of mastoid T2-hyperintensity

Out of 2341 consecutive MR scans [of those, 684 (29 %) from children's hospital] that were reviewed for the control group, 107 (4.6 %) showed T2 hyperintensity covering≥50 % of the mastoid air cells. Nearly one-third (31.8 %) of those patients were younger than 2 years old and were not included in the study. Out of 1498 MR scans [486 (32 %) from children's hospital] that were separately evaluated for any evidence of intramastoid T2-hyperintensity, 326 (21.8 %) were abnormal, and 72 (4.8 %) of them had≥50 % of the mastoid cavity covered by hyperintense signal. Left-right side distribution was 17 / 18 in the AM group, and 14 / 20 in the control group, respectively (p=0.631).

MR imaging characteristics

Differences between the MR imaging characteristics of the AM and control groups are demonstrated in Table 2. The inter-reader agreement, according to classification given by Landis and Koch, was moderate to perfect, except for the labyrinth involvement [14]. In most categories, a substantial agreement was achieved. Cohen's kappa-values are given in Table 2.

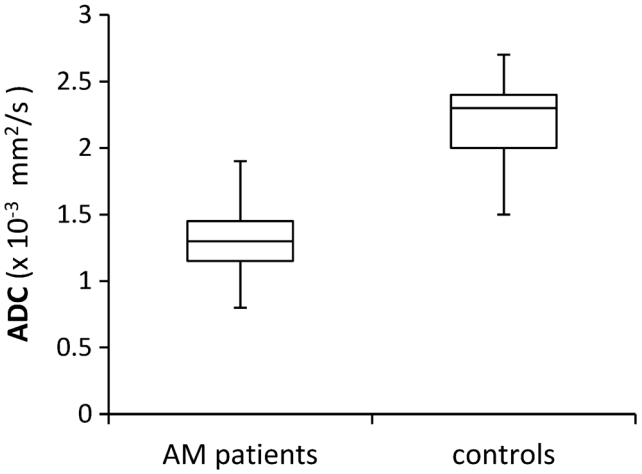

Fig. 2 ADC values of the intramastoid contents in AM patients and controls

Pneumatisation and T2-hyperintensity of the middle ear and mastoid

Reduced mastoid volume was found in two (5.7 %) patients with AM, and in ten (29.4 %) controls (p=0.012). Total or near total coverage of the mastoid by T2-hyperintensity was found in 27 (77.1 %) AM patients, and in 20 (58.8 %) controls (p=0.126). T2-hyperintensity covering>50 % of the tympanic cavity was present in 26 (74.3 %) patients, and in 13 (38.2 %) controls (p=0.004). In the mastoid antrum, > 50 % coverage was found in 28 (80.0 %) AM patients, and in 19 (55.9 %) controls (p=0.041).

Intramastoid signal intensities

On T2 FSE, the intramastoid SI was hypointense to CSF in 30 (85.7 %) AM patients and in two (5.9 %) controls. CISS sequence was only available in two controls, and showed no lowering of intramastoid contents' SI when compared to WM. SI on CISS among 28 AM patients was isointense or hypointense to WM in 11 (39.3 %) (p=0.520).

Diffusion characteristics

Diffusion restriction of intramastoid contents occurred in 18 (62.1 %) of 29 AM patients, and in none of the controls (p<0.001). The mean ADC value of intramastoid contents in the AM group was $1.33\pm0.28\times10^{-3}$ mm^2/s (range, $0.8 – 2.0\times10^{-3}$ mm^2/s), and in the control group it was 2.11 ± 0.31 (range, $1.5 – 2.7\times10^{-3}$ mm^2/s) (p<0.001) (Fig. 2).

The optimal ADC cutoff threshold of 1.72×10^{-3} mm^2/s based upon the logistic regression analysis yielded AUC of 0.983, sensitivity of 96.0 % and specificity of 88.9 %, PPV of 92.3 %, and NPV of 94.1 % for predicting clinical AM in our study setting with similar-sized AM and control groups. In reality, the prevalence of AM patients in clinical MRI practice is considerably lower than of those with incidental mastoid T2-hyperintensity. Figure 3 shows the trends in PPV and NPV dependence of AM prevalence, with PPV being 100 % up to the ADC-value of 1.4, regardless of the AM prevalence. The DWI measurements and ADC-values are dependent on MRI unit and technical parameters; therefore, logistic regression analysis was performed using only data from the same, most frequently used unit (1.5 T Avanto) and DWI sequence. A similar cutoff threshold of 1.71×10^{-3} mm^2/s with AUC of=0.963 was in fact achieved from the whole cohort with different MRI units used (Figs. 4 and 5).

Enhancement characteristics

Intense intramastoid enhancement was detected in 28 patients (51.4 %), but in none of the 29 controls (p<0.001). Enhancement of the outer periosteum occurred in 24 patients (68.6 %) vs. one (3.4 %) of the 29 controls (p<0.001); perimastoid dural enhancement occurred in 15 (42.9 %) patients vs. one (3.4 %) control (p<0.001).

Bone destruction

Destruction of the intramastoid bony septa was suspected in 12 (34.3 %), of inner cortical bone in four (11.4 %), and of outer cortical bone in ten (28.6 %) patients, and in none of the controls (p<0.001 for mastoid septa, p=0.114 for inner cortex, p=0.001 for outer cortex). Any bone destruction in general was suspected in 17 (48.6 %) patients (p< 0.001).

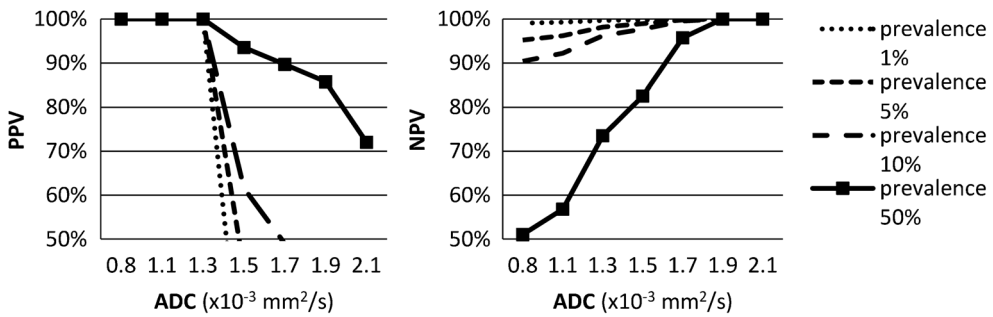

Fig. 3 PPV and NPV of ADC threshold values, depending on AM prevalence

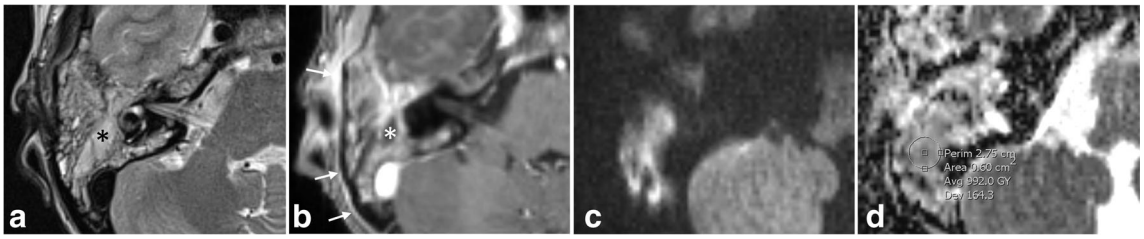

Fig. 4 Right-sided AM in a 19-year-old woman. **A:** Axial T2WI showing intramastoid SI hypointense to CSF. **B:** Axial postgadolinium T1 MPRAGE image showing intense enhancement in the walls of the fluid-filled mastoid antrum (*asterisk*) and surrounding cells, and periosteal enhancement (*arrows*) along the outer cortical bone surface. **C:** Bright signal on DWI (b=1000 s/mm^2) is due to purulent secretions inside the mastoid. **D:** An ADC value of 0.992×10^{-3} mm^2/s confirms intramastoid diffusion restriction and is compatible with AM diagnosis

Complications

Intratemporal abscess or cholesteatoma was suspected in eight patients (23.5 %), and in none of the controls (p=0.005). Extracranial abscesses were detectable in seven (20.0 %) patients, and in none of the controls (p=0.011). Epidural abscesses occurred in three (8.6 %) patients, and in none of the controls (p=0.239). Signs of labyrinth involvement were detectable in five (14.3 %) patients, and in none of the controls (p=0.058). Pachymeningitis was detected in two (5.7 %) patients and in one (3.4 %) of the controls, leptomeningitis in two (5.7 %) patients and none of the controls, with no significant differences between the groups. Sinus thrombosis was present in three (8.6 %) AM patients and in one (3.2 %) control patient, with no significant differences.

Discussion

Intramastoid T2 hyperintensity—although half as frequent as incidental mucosal swelling in paranasal sinuses—is a relatively common finding in MR imaging [9, 15]. The reported frequency of intramastoid T2 hyperintensity varies but is higher for children (12–27 %) than for adults (5 %) [7–9, 15].

Correlations between mastoid T2-hyperintensity and clinical history or signs of otitis media are controversial; stronger associations to clinically relevant disease have been reported in adults [7–9, 15]. Intramastoid bright signal on T2-WI has been shown to be a source of AM misdiagnosis by radiologists, potentially leading to unnecessary investigations or treatment [10, 11].

Middle ear effusion usually results from Eustachian tube (ET) dysfunction and middle ear pressure decrease; common causes are adenoid hypertrophy, inflammatory diseases of the middle ear or upper airways, or allergy [16]. It can sometimes be an indicator of a more serious occult pathology, such as neoplastic or vascular diseases, or previous irradiation, especially in adults [17, 18]. Bacterial AM causes pronounced vascular congestion, fluid and inflammatory cellular extravasation, proliferation of granulation tissue, and bone destruction with intracranial or extracranial complications in advanced cases [19]. Therefore, additional MR features, including intramastoid signal changes or increased contrast uptake, are to be expected in AM.

Our results of an overall frequency of 21.8 % for incidental mastoid T2-hyperintensity are similar to those obtained by previous studies [7, 8, 15]. The frequency of pronounced (≥50 %) mastoid opacification was much lower in our data, at only 4.8 %. A substantial portion of those patients were younger than 2 years old and not included in our comparative study.

Our previous study showed at least 50 % involvement of the mastoid cavity in most (94 %) patients with clinical AM [12]. The degree of incidental mastoid effusion is highly variable, as it can range from a few cells to total obliteration. Minimal fluid retention in normal clinical radiology practice would hardly draw any attention, whereas major mastoid effusion does require a mention about its clinical significance in the radiology report. Therefore, we enrolled only patients with T2-hyperintensity that covered≥50 % of the mastoid in our control group.

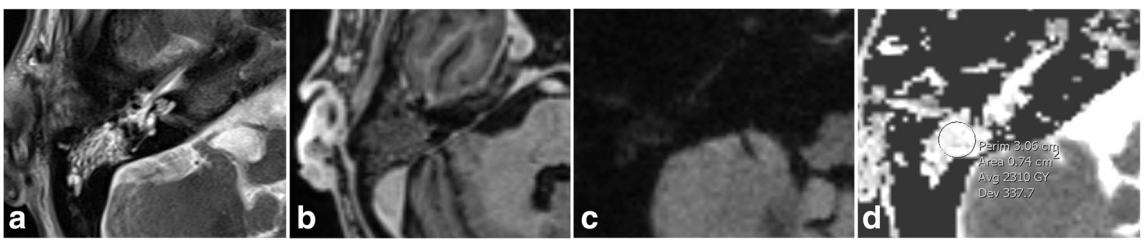

Fig. 5 Incidental mastoid effusion in a 67-year-old man. **A:** Axial T2-weighted image showing intramastoid signal intensity isointense to CSF. **B:** Axial postgadolinium T1 MPRAGE image showing only faint mucosal enhancement in the mastoid cells. **C:** No diffusion restriction is visible on DWI (b=1000 s/mm^2). **D:** An ADC value of 2.310×10^{-3} mm^2/s is compatible with non-purulent mastoid effusion

Substantial (≥50 %) obliteration of the mastoid antrum and tympanic cavity was significantly more frequent in AM patients than in controls. This finding is in line with our previous study, in which total obliteration of the tympanic cavity indicated a clinically more serious disease with higher likelihood of surgical treatment [12].

With regard to mastoid volume reduction, more abnormalities were detected in the control group. Reduced mastoid volume often associates with Eustachian tube dysfunction or previous middle ear infection [20, 21]. Experimental modelling has shown that in prolonged Eustachian tube dysfunction, small middle ear and mastoid volume actually predisposes to the development of negative middle ear pressure and effusion [22].

Signal intensity of the intramastoid contents on T2 TSE was hypointense to CSF in 86 % of the patients vs. only 6 % of the controls, which was significantly different. This difference would probably be even more pronounced in CISS, but could not be determined, because only two patients in the control group presented with this sequence.

Restricted diffusion in the mastoid contents was not found in any of the controls, whereas it was detected in 62 % of the AM patients, which is in concordance with previous knowledge on diffusion restriction in purulent secretions [23–25]. The mean ADC value of the intramastoid contents was significantly higher in the controls. In our study, the optimal ADC threshold for differentiating AM from incidental mastoid effusion was 1.72×10^{-3} mm^2/sec. It must be noted that this applies only to our study setting and that different thresholds may be achieved with other MR imaging units and technical parameters.

Intense intramastoid enhancement, and perimastoid periosteal and dural enhancement were rare in controls (only one case for each), and none had bone destruction. A few parameters, including destruction of inner cortical table, labyrinth involvement, and epidural abscesses, were not statistically significantly different between the AM group and the control group, because of too few positive findings in each group. However, none of the controls showed any abnormalities in these categories.

Our study has several limitations due to its retrospective nature. Uniform imaging protocol was lacking and different MR units were used. Potential reading bias arising from different imaging protocols must be noted. The otological status of the controls was not systematically estimated; instead, our estimation was based on information in the medical records. The AM group contained patients with aggressive disease, who were scanned in the hyperacute phase, and patients with poor clinical response to antibiotic treatment. Thus, time intervals between the AM diagnosis and MR imaging were variable.

In conclusion, in this study, MR imaging findings of patients with incidental mastoid T2-hyperintensity were found to differ from those with clinical AM in several aspects. Hypointense-to-CSF signal intensity on T2-WI, diffusion restriction, intense intramastoid enhancement, enhancement of the perimastoid dura or periosteum, bone destruction, and perimastoid extracranial abscesses rarely occurred as incidental findings. Lack of any of these features made true AM unlikely even when the whole mastoid was filled by fluid, but the presence of one or more of these features would probably justify an ENT consultation to rule out relevant mastoid pathology.

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