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Evaluation of the current clinical and bacteriological profile in tubotympanic and atticoantral type chronic suppurative otitis media

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ABSTRACT

Chronic suppurative otitis media (CSOM) is a leading cause of preventable hearing loss in low- and middle-income countries, including Indonesia. It is classified into tubotympanic and atticoantral types, yet local comparative data regarding clinical features and bacteriological profiles remain limited. This study aimed to evaluate and compare the clinical manifestations, microbiological patterns, and antibiotic susceptibility profiles of both CSOM types in a tertiary hospital. A cross-sectional study was conducted from November 2021 to August 2022 involving patients aged ≥17 yr with active CSOM. Data were collected through interviews, otoendoscopy, and pure-tone audiometry. Ear swabs were cultured and antibiotic susceptibility testing was performed. Ear-based analysis was applied for clinical and audiological variables (73 ears from 66 patients), and isolate-based analysis for microbiology. Exploratory comparisons between CSOM types were performed using Chi-square or Fisher's exact tests. Among 73 ears, 50 (68.5%) were tubotympanic and 23 (31.5%) atticoantral. Hearing loss was present in 98.6% of ears, most commonly mixed (47.9%) and conductive (42.5%), with predominantly moderate to severe degrees. Facial nerve palsy occurred in one atticoantral case (1.4%). No significant differences in clinical or audiological profiles were observed between types ($p > 0.05$). Of 76 bacterial isolates obtained from 69 culture-positive samples, Gram-negative organisms predominated (81.6%). *Pseudomonas aeruginosa* was the most frequent pathogen (57.9%), followed by *Proteus mirabilis* (13.1%). *Pseudomonas aeruginosa* showed highest susceptibility to amikacin, meropenem, and piperacillin–tazobactam, with reduced susceptibility to fluoroquinolones and cephalosporins. Gram-positive bacteria were most susceptible to linezolid, tigecycline, tetracycline, and quinupristin/dalfopristin. In conclusion, tubotympanic CSOM remains the predominant subtype, with *P. aeruginosa* as the principal pathogen. Moderate-to-severe hearing loss is common in both disease types. Clinical symptoms alone cannot differentiate CSOM subtypes, underscoring the importance of otoscopic or otoendoscopic examination and culture-guided therapy to optimize management and minimize antimicrobial resistance.

ABSTRAK

Otitis media supuratif kronik (OMSK) merupakan penyebab utama gangguan pendengaran di negara berpendapatan rendah dan menengah, termasuk Indonesia yang sebenarnya dapat dicegah. Kelainan ini digolongkan menjadi tipe tubotimpanik dan attikoantral. Namun, data di Indonesia yang membandingkan karakteristik klinis dan profil bakteriologinya masih terbatas. Penelitian ini bertujuan untuk mengevaluasi dan membandingkan manifestasi klinis, pola mikrobiologi, serta profil kepekaan antibiotik pada kedua tipe OMSK di sebuah rumah sakit rujukan tersier. Penelitian potong lintang ini dilakukan pada periode November 2021 hingga Agustus 2022 pada pasien berusia ≥17 tahun dengan OMSK aktif. Data dikumpulkan melalui wawancara, otoendoskopi, dan audiometri nada murni. Apusan telinga dikultur dan dilakukan uji kepekaan antibiotik. Analisis berbasis telinga digunakan untuk variabel klinis dan

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audiologis (73 telinga dari 66 pasien), sedangkan analisis berbasis isolat digunakan untuk data mikrobiologi. Perbandingan eksploratif antara tipe OMSK dilakukan menggunakan uji Chi-square atau uji Fisher. Dari 73 telinga yang dianalisis, 50 (68,5%) merupakan tipe tubotimpanik dan 23 (31,5%) tipe attikoantral. Gangguan pendengaran ditemukan pada 98,6% telinga, paling sering berupa gangguan pendengaran campuran (47,9%) dan konduktif (42,5%), dengan derajat dominan sedang hingga berat. Kelumpuhan saraf fasialis ditemukan pada satu kasus OMSK tipe attikoantral (1,4%). Tidak ditemukan perbedaan bermakna pada profil klinis maupun audiologis antara kedua tipe OMSK (p > 0,05). Dari 76 isolat bakteri yang diperoleh dari 69 sampel dengan kultur positif, bakteri Gram-negatif mendominasi (81,6%). *Pseudomonas aeruginosa* merupakan patogen terbanyak (57,9%), diikuti *Proteus mirabilis* (13,1%). *Pseudomonas aeruginosa* menunjukkan kepekaan tertinggi terhadap amikasin, meropenem, dan piperasilin–tazobaktam, dengan kepekaan lebih rendah terhadap fluorokuinolon dan sefalosporin. Bakteri Gram-positif paling peka terhadap linezolid, tigecycline, tetrasiklin, dan quinupristin/dalfopristin. Simpulan, OMSK tipe tubotimpanik merupakan subtipe paling dominan, dengan *P. aeruginosa* sebagai patogen utama. Gangguan pendengaran derajat sedang hingga berat umum ditemukan pada kedua tipe penyakit. Gejala klinis saja tidak dapat membedakan subtipe OMSK, sehingga menegaskan pentingnya pemeriksaan otoskopi atau otoendoskopi serta terapi berbasis kultur untuk mengoptimalkan penatalaksanaan dan meminimalkan resistensi antimikroba.

INTRODUCTION

Chronic suppurative otitis media (CSOM) is a chronic inflammatory disease of the middle ear and mastoid cavity lasting more than six weeks. It is classified into two clinical types namely the tubotympanic type and the atticoantral type. Both types remain major causes of preventable hearing loss particularly in low-income countries including Indonesia.[1,2] A population-based study in Indonesian schoolchildren found otitis media in 2.5% of participants, with 67% classified as CSOM. Otitis media accounted for 57% of mild-to-moderate hearing loss cases. CSOM was the leading cause of moderate hearing impairment and represents a major public health burden in Indonesia.[3]

Clinically, CSOM is characterized by persistent otorrhea, tympanic membrane perforation, and varying degrees of hearing loss.[4] Delayed or inadequate treatment may lead to extracranial complications such as Bezold's abscess and facial nerve paralysis. Intracranial complications including brain abscess and meningitis have also been reported and remain important causes of morbidity and mortality.[5-7]

Bacterial infection plays a central role in the pathogenesis of CSOM. Therefore, understanding local bacteriological profiles is essential for selecting appropriate empirical antibiotic therapy and minimizing the development of antimicrobial resistance.[8] However, comparative data on clinical features and causative microorganisms between the tubotympanic and atticoantral types of CSOM in Indonesia remain limited.[4] This study aimed to compare the clinical characteristics of tubotympanic and atticoantral CSOM and to evaluate the distribution of causative microorganisms and their antibiotic susceptibility patterns to support evidence-based empirical treatment.

MATERIAL AND METHODS

Study Design and Setting

This cross-sectional study was conducted at the Ear Nose and Throat clinic of a tertiary hospital from November 2021 to August 2022. Consecutive sampling was applied. Patients aged ≥17 yr, diagnosed with CSOM by an otologist and had active otorrhea suitable for swab collection were included.

Eligible patients were invited to participate and written informed consent was obtained. Clinical data were collected through structured interviews regarding duration of symptoms and presenting complaints. Physical examination was performed using otoendoscopy to document the characteristics of the tympanic membrane perforation the type of ear discharge and signs of complications including facial nerve paresis. Pure tone audiometry was performed to determine the type and degree of hearing loss.

Microbiological examination

The unit of sampling for microbiological analysis was the affected ear. Each ear with active discharge was treated as an independent sample because several patients presented with bilateral disease. Ear swabs were collected aseptically using sterile cotton swabs under otomicroscopy guidance. Samples were obtained after clinical examination and assessment of otorrhea characteristics. All specimens were transported to the hospital microbiology laboratory using appropriate transport media.

Bacteriological culture and identification were performed according to standard laboratory protocols. Isolate-based analysis was applied for determination of the causative microorganisms, including cases of polymicrobial growth. Antibiotic susceptibility testing was conducted for all bacterial isolates using routine methods based on laboratory standards. Patients with incomplete clinical records or unavailable microbiological culture results were excluded from the final analysis.

Statistical analysis

Ear-based analysis was applied for demographic variables symptoms and audiological findings because each ear with active disease was considered an independent unit of analysis. Isolate-based analysis was used for microorganism profiling. Descriptive statistics were used to report frequencies and percentages.

Categorical variables were categorized as present or absent for clinical symptoms. Hearing loss was categorized based on audiogram findings into normal conductive hearing loss and mixed hearing loss. The degree of hearing loss was classified as normal mild moderate severe and very severe.

Comparative analysis between tubotympanic and atticoantral types for clinical symptoms was performed exploratively to identify potential differences in clinical manifestations. Categorical variables were analyzed using the Chi-square test. Fisher's exact test was applied when the expected cell count was less than five. Statistical analyses were conducted using SPSS software version 25. A p-value of <0.05 was considered statistically significant.

Ethical approval

This study was approved by the Ethics Committee of Universitas Jenderal Soedirman under approval number 010/KEPK/I/2021. Written informed consent was obtained from all participants.

RESULTS

A total of 66 patients were enrolled in this study, yielding 73 affected ears due to bilateral disease in 7 patients. Of the ears examined, 50 (68.5%) were classified as tubotympanic type and 23 (31.5%) as atticoantral type (TABLE 1).

Demographic characteristics

Age and sex distribution based on ear-based analysis are summarized in TABLE 2. Two age peaks were observed, in the 16-20 yr group and the 46–50 yr group, each accounting for 15.1% of affected ears. Tubotympanic CSOM was most frequently observed among individuals aged 41-45 yr (18%), while atticoantral CSOM predominated in the aged 21–25 yr group (30.4%). Female ears accounted for the majority of tubotympanic cases (68%), whereas males predominated in the atticoantral group (56.5%).

TABLE 1. Distribution of CSOM type

CSOM Type	Total n=73 [n (%)]
Tubotympanic type	50 (68.5)
Atticoantral type	23 (31.5)

TABLE 2. Age group and gender distribution of CSOM

Characteristics	CSOM TT n=50 [n (%)]	CSOM AA n=23 [n (%)]	Total n=73 [n (%)]
Age group (yr)			
16-20	6 (12)	5 (21.7)	11 (15.1)
21-25	0 (0)	7 (30.4)	7 (9.6)
26-30	7 (14)	1 (4.3)	8 (11)
31-35	3 (6)	5 (21.7)	8 (11)
36-40	4 (8)	0 (0)	4 (5.5)
41-45	9 (18)	0 (0)	9 (12.3)
46-50	8 (16)	3 (13)	11 (15.1)
51-55	2 (4)	1 (4.3)	3 (4.1)
56-60	3 (6)	1 (4.3)	4 (5.5)
61-65	2 (4)	0 (0)	2 (2.7)
66-70	5 (10)	0 (0)	5 (6.8)
71-75	1 (2)	0 (0)	1 (1.4)
Gender			
Female	34 (68)	10 (43.5)	44 (60.3)
Male	16 (32)	13 (56.5)	29 (39.7)

Clinical manifestations and audiological findings

The clinical characteristics of CSOM are shown in TABLE 3. Most ears had a disease duration of >1 yr (68.5%). The most common discharge type was mucopurulent (65.8%), followed by purulent discharge (23.3%). The majority of ears were associated with a smelly discharge (58.9%), subjective hearing loss (98.6%), tinnitus (78.1%), and ear fullness (75.3%). Other symptoms included itching (58.9%), otalgia (57.5%), headache (60.3%), and vertigo (32.9%). Facial nerve paralysis House Brackmann IV was observed in one ear (1.4%), which occurred in the atticoantral group.

Audiometric evaluation demonstrated that mixed hearing loss was the most frequent type (47.9%), followed by conductive hearing loss (42.5%) and normal hearing (9.6%). Regarding severity, moderate hearing loss predominated (34.2%), followed by severe hearing loss (31.5%), with only a small proportion falling into the very severe category (5.5%). Exploratory comparative analysis between the tubotympanic and atticoantral types revealed no statistically significant differences across all clinical symptoms or audiometric variables ($p > 0.05$; TABLE 3).

Microbiological profile

From the 73 ear swabs, microbial growth was detected in 69 samples, while 4 samples (5.3%) showed no growth. In total, 76 microbial isolates were obtained. Most samples demonstrated monomicrobial growth (81.6%), whereas mixed growth occurred in 9.2% of ears (TABLE 4). Gram-negative bacteria predominated (81.6%), with *P. aeruginosa* representing the most frequently isolated organism (57.9% of all isolates). This species was most prominently isolated from the tubotympanic type (36 isolates) compared with the atticoantral type (8 isolates).

Other common Gram-negative organisms included *P. mirabilis* (13.1%), which was mainly isolated from the atticoantral group, *K. pneumoniae* (3.9%), *E. coli* (2.6%), *E. aerogenes* (2.6%), and *E. cloacae complex* (1.3%). *Escherichia coli* and *E. aerogenes* were isolated only in the atticoantral group. Gram-positive bacteria comprised 18.4% of isolates, with *S. aureus* (6.6%) being the most common, followed by *Enterococcus spp*. (5.3%), *S. hominis* (2.6%), and smaller numbers of *S. epidermidis, S. haemolyticus,* and *S. warneri.*

Antibiotic susceptibility patterns

The antibiotic susceptibility profiles of gram-negative and gram-positive isolates are summarized in TABLE 5 and 6 and visualized in the susceptibility heatmap (FIGURE 1). For *P. aeruginosa* isolates, the highest susceptibility was observed to amikacin (65.9%), meropenem (61.4%), and piperacillin–tazobactam (56.8%). Moderate susceptibility was found to gentamicin (52.3%), cefepime (38.6%), and ciprofloxacin (40.9%), while very low activity was demonstrated for tigecycline (2.3%). *Proteus mirabilis* isolates showed high susceptibility to piperacillin–tazobactam, meropenem, and amikacin (100%), and moderate susceptibility to cefepime and gentamicin. All isolates of *K. pneumoniae* were fully susceptible to third- and fourth-generation cephalosporins, carbapenems, fluoroquinolones, and aminoglycosides.

TABLE 3. Clinical characteristics of tubotympanic and atticoantral CSOM.

Clinical symptoms and manifestation	CSOM TT n=50 [n (%)]	CSOM AA n=23 [n (%)]	Total n=73 [n (%)]	p
Onset				
2-6 mo	12 (24)	4 (17.4)	16 (21.9)	0.418
6 mo – 1 yr	6 (12)	1 (4.3)	7 (9.6)	
>1 yr	32 (64)	18 (78.3)	50 (68.5)	
Characteristic of ear discharge				
Mucopurulent	34 (68)	14 (60.9)	48 (65.8)	0.832
Purulent	11 (22)	6 (26.1)	17 (23.3)	
Dry	5 (10)	3 (13)	8 (11)	
Smelly Ear				
Yes	28 (56)	15 (65.2)	43 (58.9)	0.626
No	22 (44)	8 (34.8)	30 (41.1)	
Sensation of hearing loss				
Yes	49 (98)	23 (100)	72 (98.6)	1*
No	1 (2)	0 (0)	1 (1.4)	
Tinnitus				
Yes	41 (82)	16 (69.6)	57 (78.1)	0.374
No	9 (18)	7 (30.4)	16 (21.9)	
Ear-fullness				
Yes	40 (80)	15 (65.2)	55 (75.3)	0.285
No	10 (20)	8 (34.8)	18 (24.7)	
Itchy Ear				
Yes	32 (64)	11 (47.8)	43 (58.9)	0.294
No	18 (36)	12 (52.2)	30 (41.1)	
Otalgia				
Yes	28 (56)	14 (60.9)	42 (57.5)	0.892
No	22 (44)	9 (39.1)	31 (42.5)	
Headache				
Yes	27 (54)	17 (73.9)	44 (60.3)	0.175
No	23 (46)	6 (26.1)	29 (39.7)	
Vertigo				
Yes	17 (34)	7 (30.4)	24 (32.9)	0.974
No	33 (66)	16 (69.6)	49 (67.1)	
Nerve VII paralysis				
Yes	0 (0)	1 (4.3)	1 (1.4)	0.315*
No	50 (100)	22 (95.7)	72 (98.6)	
Hearing loss				
Normal	5 (10)	2 (8.7)	7 (9.6)	0.982
Conductive	21 (42)	10 (43.5)	31 (42.5)	
Mixed	24 (48)	11 (47.8)	35 (47.9)	
Hearing loss degree				
Normal	5 (10)	2 (8.7)	7 (9.6)	0.569
Mild	12 (24)	2 (8.7)	14 (19.2)	
Moderate	16 (32)	9 (39.1)	25 (34.2)	
Severe	15 (30)	8 (34.8)	23 (31.5)	
Very Severe	2 (4)	2 (8.7)	4 (5.5)	

Chi-square test was used. Variables marked with (*) were analyzed using Fisher's exact test.

TABLE 4. Profile and frequency of isolated bacteria

Total ears (n=73)	Total	CSOM TT	CSOM AA
Bilateral ears (n)	7	7	0
Total isolates [n (%)]	76	53	23
Monomicrobial	62 (81.6)	43	19
Mixed growth	7 (9.2)	5	2
No growth	4 (5.3)	2	2
Gram-negative isolates [n (%)]	62 (81.6)		
Pseudomonas aeruginosa	44 (57.9)	36	8
Proteus mirabilis	10 (13.1)	2	8
Klebsiella pneumoniae	3 (3.9)	3	0
Escherichia coli	2 (2.6)	0	2
Enterobacter aerogenes	2 (2.6)	0	2
Enterobacter cloacae complex (ECC)	1 (1.3)	1	0
Gram-positive isolates [n (%)]	14 (18.4)		
Staphylococcus aureus	5 (6.6)	4	1
Enterococcus sp	4 (5.3)	3	1
Staphylococcus hominis	2 (2.6)	1	1
Staphylococcus epidermidis	1 (1.3)	1	0
Staphylococcus haemolyticus	1 (1.3)	1	0
Staphylococcus warneri	1 (1.3)	1	0

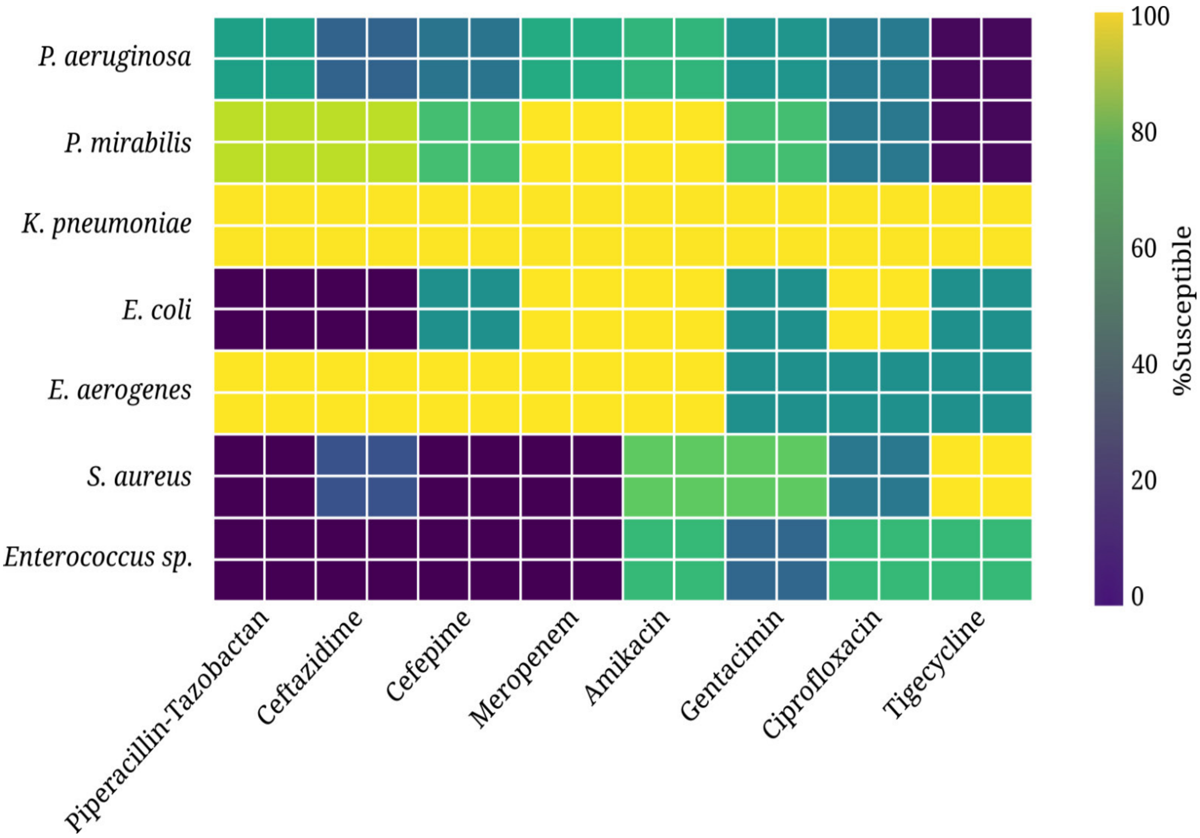

FIGURE 1. Antibiotic susceptibility patterns of gram-negative and gram-positive bacteria.

TABLE 5. Antibiotic susceptibility of Gram-negative isolates [n (%)].

Antibiotic	*P. aeruginosa* (n= 44)		*P. mirabilis* (n= 10)		*K. pneumoniae* (n=3)		*E. coli* (n= 2)		*E. aerogenes* (n= 2)		*E. cloacae complex* (ECC) (n= 1)	
	TT (n=36)	AA (n=8)	TT (n=2)	AA (n=8)	TT (n=3)	AA (n=0)	TT (n=0)	AA (n=2)	TT (n=0)	AA (n=2)	TT (n=1)	AA (n=0)
Ampicillin	NT	NT	1 (50)	4 (50)	0 (0)	-	-	0 (0)	-	NT	NT	-
Ampicillin/ Sulbactam	NT	NT	1 (50)	5 (62.5)	3 (100)	-	-	0 (0)	-	NT	NT	-
Piperacillin/ Tazobactam	19 (52.7)	6 (75)	2 (100)	7 (87.5)	3 (100)	-	-	2 (100)	-	2 (100)	1 (100)	-
Cefazolin	0 (0)	0 (0)	NT	NT	NT	-	-	NT	-	0 (0)	0 (0)	-
Ceftazidime	10 (27.8)	4 (50)	2 (100)	7 (87.5)	3 (100)	-	-	0 (0)	-	2 (100)	1 (100)	-
Ceftriaxone	NT	NT	1 (50)	5 (62.5)	3 (100)	-	-	0 (0)	-	1 (50)	1 (100)	-
Cefepime	13 (36.1)	4 (50)	1 (50)	6 (75)	3 (100)	-	-	1 (50)	-	2 (100)	1 (100)	-
Aztreonam	NT	NT	2 (100)	7 (87.5)	3 (100)	-	-	0 (0)	-	1 (50)	1 (100)	-
Ertapenem	NT	NT	2 (100)	6 (75)	3 (100)	-	-	1 (50)	-	1 (50)	1 (100)	-
Meropenem	21 (58.3)	6 (75)	2 (100)	8 (100)	3 (100)	-	-	2 (100)	-	2 (100)	1 (100)	-
Amikacin	24 (66.7)	5 (62.5)	2 (100)	8 (100)	3 (100)	-	-	2 (100)	-	2 (100)	1 (100)	-
Gentamicin	18 (50)	5 (62.5)	1 (50)	6 (75)	3 (100)	-	-	1 (50)	-	1 (50)	1 (100)	-
Ciprofloxacin	14 (38.9)	4 (50)	1 (50)	3 (37.5)	3 (100)	-	-	2 (100)	-	1 (50)	1 (100)	-
Tigecycline	0 (0)	1 (12.5)	0 (0)	0 (0%)	3 (100)	-	-	1 (50)	-	1 (50)	1 (100)	-
Nitrofurantoin	NT	NT	0 (0)	0 (0)	0 (0)	-	-	NT	-	0 (0)	1 (100)	-
Trimethoprim/ Sulfamethoxazole	NT	NT	NT	NT	NT	-	-	NT	-	0 (0)	1 (100)	-
Cotrimoxazole	NT	NT	NT	NT	3 (100)	-	-	1 (50)	-	NT	NT	-

TT= tubotympanic type; AT= atticoantral type; NT = not tested

TABLE 6. Antibiotic susceptibility of Gram-positive isolates [n (%)]

Antibiotic	*S. aureus* (n=5)		*Enterococcus* sp. (n=4)		*S. hominis* (n=2)		*S. epidermidis* (n=1)		*S. haemolyticus* (n= 1)		*S. warneri* (n=1)	
	TT (n=4)	AA (n=1)	TT (n=3)	AA (n=1)	TT (n=1)	AA (n=1)	TT (n=1)	AA (n=0)	TT (n=1)	AA (n=0)	TT (n=1)	AA (n=0)
Benzyl-penicillin	0 (0)	0 (0)	2 (66.7)	0 (0)	0 (0)	0 (0)	0 (0)	-	0 (0)	-	0 (0)	-
Ampicillin	NT	NT	2 (66.7)	0 (0)	NT	NT	NT	-	NT	-	NT	-
Oxacillin	0 (0)	NT	NT	NT	0 (0)	0 (0)	NT	-	0 (0)	-	0 (0)	-
Gentamicin	3 (75)	1 (100)	1 (33.3)	0 (0)	1 (100)	1 (100)	1 (100)	-	0 (0)	-	1 (100)	-
Ciprofloxacin	1 (25)	1 (100)	2 (66.7)	1 (100)	0 (0)	1 (100)	1 (100)	-	0 (0)	-	1 (100)	-
Levofloxacin	1 (25)	1 (100)	1 (33.3)	1 (100)	0 (0)	1 (100)	1 (100)	-	0 (0)	-	1 (100)	-
Moxifloxacin	1 (25)	1 (100)	NT	NT	0 (0)	1 (100)	1 (100)	-	0 (0)	-	1 (100)	-
Erythromycin	2 (50)	0 (0)	2 (66.7)	0 (0)	0 (0)	1 (100)	0 (0)	-	0 (0)	-	1 (100)	-
Clindamycin	1 (25)	0 (0)	NT	NT	0 (0)	1 (100)	0 (0)	-	0 (0)	-	1 (100)	-
Quinupristin/ Dalfopristin	2 (50)	1 (100)	0 (0)	0 (0)	1 (100)	1 (100)	1 (100)	-	1 (100)	-	1 (100)	-
Linezolid	3 (75)	1 (100)	2 (66.7)	0 (0)	1 (100)	1 (100)	1 (100)	-	1 (100)	-	1 (100)	-
Vancomycin	1 (25)	0 (0)	0 (0)	0 (0)	1 (100)	1 (100)	0 (0)	-	1 (100)	-	1 (100)	-
Tetracycline	3 (75)	1 (100)	2 (66.7)	0 (0)	0 (0)	1 (100)	1 (100)	-	NT	-	0 (0)	-
Tigecycline	4 (100)	1 (100)	2 (66.7)	NT	1 (100)	1 (100)	1 (100)	-	NT	-	1 (100)	-
Nitrofurantoin	4 (100)	1 (100)	2 (66.7)	0 (0)	1 (100)	1 (100)	1 (100)	-	1 (100)	-	1 (100)	-
Rifampicin	2 (50)	1 (100)	1 (33.3)	NT	1 (100)	1 (100)	1 (100)	-	1 (100)	-	1 (100)	-
Trimethoprim	NT	NT	NT	NT	NT	NT	NT	-	1 (100)	-	1 (100)	-
Cotrimoxazole	3 (75)	1 (100)	1 (33.3)	NT	0 (0)	1	1 (100)	-	NT	-	NT	-
Piperacillin/ Tazobactam	NT	NT	NT	NT	NT	NT	NT	-	NT	-	NT	-

TT= tubotympanic type; AT= atticoantral type; NT = not tested; NT = not tested.

Gram-positive bacteria displayed the greatest susceptibility to linezolid, tigecycline, nitrofurantoin, quinupristin/dalfopristin, and tetracycline, particularly among *S. aureus* and coagulase-negative staphylococci. Conversely, resistance to beta-lactams and macrolides was frequently observed among these organisms. Methicillin Resistant *S. aureus* (MRSA) was seen positive in 80% of *S. aureus* cases.

The heatmap illustrates a clear predominance of broad-spectrum susceptibility among Gram-negative isolates to carbapenems and aminoglycosides, while highlighting variable resistance patterns to fluoroquinolones and cephalosporins, particularly in *P. aeruginosa* (FIGURE 1). Gram-positive organisms demonstrated consistently high susceptibility to linezolid and tigecycline.

DISCUSSION

This study provides an updated overview of the clinical characteristics, microbiological profile, and antimicrobial susceptibility patterns of CSOM in an Indonesian tertiary-care setting. Chronic suppurative otitis media is characterised by long-term inflammation of the middle ear and mastoid mucosa, with a perforated tympanic and persistent ear discharge.[2] There are two types of CSOM i.e. tubotympanic type and atticoantral type.[9] The tubotympanic type was the dominant presentation, accounting for approximately two-thirds of affected ears. This distribution is consistent with previous reports indicating that the tubotympanic CSOM remains the most prevalent subtype worldwide, particularly in developing countries, where delayed presentation and barriers to early treatment are common.[1,4,10]

Two age peaks were observed at 16–20 yr and 46–50 yr. The predominance of younger patients may be related to increased susceptibility to upper respiratory tract infections that predispose individuals to CSOM.[4] The second peak in middle-aged adults may reflect the influence of comorbid conditions that impair immune defenses and delay disease resolution.[9] Regarding sex distribution, this study found a higher proportion of female patients, particularly in the tubotympanic group (68%). This finding is consistent with previous reports suggesting that women tend to exhibit greater healthcare-seeking behavior, resulting in higher detection rates.[4] In contrast, males predominated in the atticoantral group (56.5%), which has been associated with exposure to risk activities such as swimming.[4] Nevertheless, there is no evidence that CSOM occurrence is intrinsically related to gender, as anatomical differences between males and females do not influence disease susceptibility.[11,12]

Clinically, 68.5% of ears had symptoms persisting for > 1 yr, indicating possible patient neglect and failure of previous treatment. Treatment failure may also be associated with antibiotic resistance.[4] Nearly all ears were associated with hearing loss (98.6%), while most patients experienced tinnitus (78.1%) and ear fullness (75.3%). These findings confirm the substantial functional burden imposed by CSOM.[2] Mucopurulent otorrhea was the most frequent discharge type observed in both CSOM subtypes (65.8%), consistent with previous studies and reflecting ongoing chronic infection and persistent mucosal inflammation.[4] Complications of CSOM may be classified into extracranial and intracranial types. Facial nerve paralysis graded as House Brackmann IV occurred in 1.4% of cases and was found exclusively in the atticoantral group, highlighting the known association between cholesteatoma, granulation tissue, and the development of extracranial or intracranial complications.[5,13] In addition, cholesteatoma can release neurotoxic substances that contribute to bone destruction and subsequent facial nerve compression, further explaining the mechanism of facial nerve involvement.[9]

Audiological assessment in both CSOM types demonstrated a predominance of mixed hearing loss (47.9%), followed by conductive hearing loss (42.5%). These findings are consistent with the multifactorial pathophysiology of CSOM, which includes tympanic membrane perforation, ossicular chain disruption, chronic inflammatory changes, and possible inner-ear involvement with cochlear hair cell damage.[4,14]. Hearing loss severity was most commonly in the moderate (34.2%) to severe (31.5%) range in both groups. This degree of impairment may be influenced by the site and size of tympanic membrane perforations, as posterior perforations are associated with greater decibel loss, likely due to reduced protection of the round window membrane from direct sound wave impact.[11]

Exploratory comparison between the tubotympanic and atticoantral types revealed no statistically significant differences in clinical symptoms or hearing outcomes. Although the atticoantral type is traditionally considered more aggressive, the absence of significant differences in this study may reflect similar disease chronicity at presentation in both groups or the limited statistical power due to sample size.[11] These findings highlight that clinical symptoms alone cannot reliably differentiate CSOM subtypes or indicate disease severity. Therefore, accurate diagnosis must rely on a comprehensive otoscopic or otoendoscopic examination, which allows clinicians to identify key markers of severity such as cholesteatoma, granulation tissue, and the extent of mucosal disease. These structural findings are far more informative for prognosis and management decisions than symptoms reported by the patient. Further evaluation using microbiological culture and antibiotic susceptibility testing is essential to guide targeted antimicrobial therapy, particularly in cases with poor response to empirical treatment. In addition, temporal bone CT scanning plays a critical role in assessing complications and surgical planning, especially when cholesteatoma or extensive bony erosion is suspected.[9,15]

Microbiological analysis demonstrated growth in most samples, with monomicrobial infection predominating (81.6%), while mixed bacterial growth was observed in only 9.2% of cases. No microbial growth was detected in 5.3% of ears, which is likely related to prior antibiotic exposure, as most patients were referred to this tertiary hospital after receiving previous treatment.[14] The low rate of polymicrobial infection supports previous findings that CSOM in its chronic stage is commonly dominated by a single pathogenic organism rather than mixed flora. The etiological profile of CSOM is known to vary depending on environmental conditions, socioeconomic and demographic factors, as well as personal hygiene practices, all of which may influence bacterial colonization and infection patterns.[12]

In the present study, gram-negative organisms predominated, with *P. aeruginosa* identified as the most common pathogen, accounting for 57.9% of all isolates. This finding is consistent with previous reports that recognize *P. aeruginosa* as the principal causative organism in CSOM.[16] Its dominance is attributed to its ability to survive in moist and unhygienic environments, form biofilms, produce various virulence factors, and demonstrate intrinsic resistance to multiple antibiotic classes.[4,11,17] *Proteus mirabilis* was the second most frequent isolate (13.1%) and was found predominantly in the atticoantral group. Gram-positive bacteria were less commonly isolated, primarily *S. aureus* (6.6%)

and *Enterococcus* species (5.3%). Additionally, *S. epidermidis* (1.3%) was detected, which is considered part of the normal flora of the external auditory canal and may represent contamination rather than true pathogenic involvement.[14,18]

Antibiotic susceptibility testing revealed clinically significant resistance trends among the isolated pathogens. *Pseudomonas aeruginosa* showed the highest susceptibility to amikacin, meropenem, and piperacillin-tazobactam, indicating that aminoglycosides and carbapenems remain the most reliable options for complicated or refractory CSOM cases. In contrast, reduced susceptibility to fluoroquinolones and cephalosporins was evident. This finding is clinically concerning because topical fluoroquinolones are widely used as first-line empirical therapy for CSOM.[15] Excessive and inappropriate use of antibiotics has been recognized as a major driver of increasing antimicrobial resistance among CSOM pathogens, particularly in regions with limited culture-guided treatment practices.[8,12]

Resistance in *P. aeruginosa* is mediated by efflux pump overexpression, target-site mutations causing fluoroquinolone resistance, and β-lactam resistance through AmpC overproduction and extended-spectrum β-lactamase production.[17] Biofilm formation further promotes bacterial persistence and treatment failure. Its dominance in CSOM is supported by its ability to survive under minimal nutritional conditions, adhere to damaged middle ear epithelium via pili, and produce virulence factors such as proteases, lipopolysaccharides, pyocyanin, and bacteriocins that enable immune evasion and competition with other microorganisms.[16]

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From a therapeutic perspective, fluoroquinolone antibiotics remain commonly used as empirical agents for CSOM because of their favorable safety profile, low risk of ototoxicity, and wide availability in topical formulations.[15,16] However, reliance on empirical fluoroquinolone therapy alone may contribute to further resistance and limit their clinical effectiveness. Our study also demonstrated reduced susceptibility to fluoroquinolones, supporting this emerging concern. In contrast, our isolates showed higher susceptibility to carbapenems and aminoglycosides, which demonstrated the most consistent activity against gram-negative pathogens. Nevertheless, the use of aminoglycosides requires careful consideration because of their potential ototoxic effects.[16]

Notably, MRSA accounted for 80% of *S. aureus* isolates in this study, supporting concerns regarding the rising prevalence of community-acquired MRSA in CSOM and underscoring the need for ongoing regional antimicrobial surveillance.[4,14] Gram-positive organisms retained high susceptibility to linezolid and tigecycline.

These findings emphasize the importance of culture-guided therapy in CSOM, particularly in cases unresponsive to initial empirical treatment. Standard management begins with aural toilet and topical antibiotic therapy to achieve microbial eradication, followed by tympanic membrane repair once infection control is established.[15] Although topical antibiotics remain the mainstay of treatment, systemic antibiotics may be required when no appropriate ototopical option is available based on susceptibility testing.[9] Routine microbiological evaluation is therefore essential to optimize antibiotic selection and support antimicrobial stewardship efforts in the face of rising antimicrobial resistance.[8]

Several limitations of this study should be considered. First, the study employed a cross-sectional, single-center design, which limits generalizability to other populations and settings. Second, microbiological analysis was restricted to conventional culture techniques without molecular pathogen identification or resistance mechanism characterization. Third, the relatively small number of isolates for some bacterial species limited robust comparative analysis between subgroups.

CONCLUSION

This study confirms that tubotympanic CSOM is the predominant subtype in Indonesia, with *P. aeruginosa* as the principal pathogen. Mixed and conductive hearing loss are the most common findings, with moderate-to-severe impairment frequently affecting both disease types. Although no significant clinical or audiological differences are observed between tubotympanic and atticoantral CSOM, each subtype carries distinct clinical considerations, including the higher risk of complications in the atticoantral subtype. Given the dominance of *P. aeruginosa* and its resistance patterns, empirical therapy should prioritize agents with reliable local activity and be regularly aligned with regional susceptibility data. This study highlights that clinical symptoms alone cannot differentiate CSOM subtypes. Therefore, comprehensive otoscopic or otoendoscopic evaluation combined with culture-guided antibiotic selection remains essential to optimize management and limit the progression of antimicrobial resistance.

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