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Antimicrobial Resistance in Chronic Suppurative Otitis Media with Special Reference to ESBL-Producing Pseudomonas aeruginosa and MRSA in Central India

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ABSTRACT

Background: Chronic suppurative otitis media (CSOM) is frequently complicated by persistent discharge, recurrent infection, biofilm-associated bacterial survival, and repeated antimicrobial exposure. These features create favourable conditions for the emergence of multidrug-resistant organisms, particularly Pseudomonas aeruginosa and Staphylococcus aureus. Resistance surveillance is therefore essential for safe empirical therapy and antimicrobial stewardship.

Aim: To analyze antimicrobial resistance patterns among bacterial isolates from CSOM cases at a tertiary care hospital in Central India, with special reference to extended-spectrum beta-lactamase (ESBL)-producing P. aeruginosa and methicillin-resistant S. aureus (MRSA).

Methods: The manuscript is based on a hospital-based study of 100 clinically diagnosed CSOM patients. Ear-discharge specimens were processed for aerobic bacterial culture, fungal culture, organism identification, and antimicrobial susceptibility testing. Disc diffusion-based susceptibility interpretation followed accepted laboratory standards.[7] Cefoxitin screening was used for methicillin resistance in S. aureus, while ceftazidime and ceftazidime-clavulanic acid were used for phenotypic ESBL detection among multidrug-resistant P. aeruginosa isolates.[8] [10]

Results: Aerobic bacterial growth was obtained in 81% of samples. P. aeruginosa was the most common isolate (35%), followed by S. aureus (24%). Among 24 S. aureus isolates, 7 were MRSA, giving an MRSA proportion of 29.2%. Linezolid showed the highest overall sensitivity against S. aureus (80.8%), followed by azithromycin and gentamicin (76.79% each). Among 35 P. aeruginosa isolates, imipenem was the most active agent (80.0% sensitivity), followed by meropenem (74.3%), amikacin (65.7%), piperacillin-tazobactam (63.0%), cefepime (62.9%), gentamicin (60.0%), ceftazidime (57.1%), ciprofloxacin (57.1%), levofloxacin (54.3%), and aztreonam (51.4%). Universal resistance was observed to ampicillin, ceftriaxone, and cotrimoxazole. ESBL-producing P. aeruginosa was reported in 31.4% of P. aeruginosa isolates, with markedly higher resistance than non-ESBL isolates.

Conclusion: CSOM in this cohort showed clinically significant resistance among both Gram-negative and Gram-positive pathogens. Carbapenems remained the most active agents against P. aeruginosa, but the detection of ESBL-producing isolates and MRSA indicates that culture-guided therapy should be preferred over repeated empirical therapy. Institutional CSOM antibiograms should be updated regularly to support otological antimicrobial stewardship.

Keywords: Chronic suppurative otitis media; antimicrobial resistance; Pseudomonas aeruginosa; ESBL; MRSA; antibiogram; Central India.

INTRODUCTION

Chronic suppurative otitis media is a persistent inflammatory disease of the middle ear that usually presents with recurrent or continuous otorrhoea through a perforated tympanic membrane. In addition to producing discomfort and hearing

impairment, CSOM may act as a reservoir for organisms exposed to repeated topical and systemic antimicrobial therapy. The chronic discharge, altered middle-ear mucosa, tissue debris, and moist local environment allow bacteria to persist for long periods. In many cases, the infection becomes difficult to eradicate because organisms form biofilms or acquire resistance mechanisms that reduce the effectiveness of conventional therapy.[1] [2]

The relationship between CSOM and antimicrobial resistance is clinically important. Patients often receive multiple courses of topical ear drops or oral antibiotics before culture is performed. Such partial and repeated exposure can suppress susceptible organisms while allowing resistant strains to persist. P. aeruginosa is especially important because it is intrinsically resistant to several commonly used antibiotics and can acquire additional resistance through beta-lactamase production, efflux pumps, altered porins, biofilm formation, and other adaptive mechanisms.[3] [4] Its nutritional flexibility, iron-acquisition systems, quorum-sensing networks, and capacity to adapt to different host microenvironments help explain why it persists in chronic infections even when competing organisms are suppressed.[5] In chronic ear disease, its ability to survive in moist environments and resist host clearance makes it a frequent cause of persistent otorrhoea.

S. aureus is another major CSOM pathogen. Methicillin resistance changes therapeutic choices and is an infection-control concern, particularly in hospitals where MRSA may circulate between community and healthcare settings. Accurate detection of MRSA by cefoxitin screening is widely accepted because cefoxitin is a strong inducer of mecA-mediated resistance and provides reliable phenotypic recognition of methicillin resistance.[10] If MRSA is not identified, patients may receive beta-lactams that are unlikely to succeed.

Extended-spectrum beta-lactamase production in P. aeruginosa is also a matter of concern. Although ESBL testing has traditionally been emphasized in Enterobacteriaceae, phenotypic ESBL-like resistance in P. aeruginosa can compromise cephalosporins and is often associated with multidrug resistance.[8] [9] Indian studies have highlighted the need to detect ESBL-producing Pseudomonas species in clinical isolates, particularly in tertiary-care settings where broad-spectrum antibiotic exposure is common.[12]

The present paper uses data from a CSOM study conducted at Index Medical College Hospital & Research Centre, Indore, to examine antimicrobial susceptibility patterns in CSOM isolates, with detailed attention to P. aeruginosa and S. aureus resistance. The broader microbiological spectrum is acknowledged, but the main focus is the resistance pattern that should guide empirical policy, culture-based therapy, and institutional stewardship.

MATERIALS AND METHODS

Study design and population

The study included 100 clinically diagnosed CSOM patients who attended the otorhinolaryngology outpatient department of a tertiary-care hospital. The source protocol enrolled eligible patients consecutively and used ear-discharge samples for microbiological evaluation. The key resistance-related objectives were to determine antibiotic susceptibility patterns of bacterial isolates and to detect drug resistance in P. aeruginosa.

Microbiological identification

Specimens were collected aseptically from active ear discharge and processed for aerobic bacterial culture. Organisms were identified by colony morphology, Gram staining, and biochemical tests according to standard diagnostic microbiology practice.[6] Fungal culture was also performed in the original study, but fungal susceptibility was not the main focus of the present resistance analysis.

Antimicrobial susceptibility testing

Disc diffusion susceptibility testing was performed using antibiotic panels appropriate to Gram-positive cocci, Gram-negative bacilli, and P. aeruginosa. Interpretation was based on standard zone-size criteria and contemporary laboratory guidance.[7] For S. aureus, the tested agents included linezolid, azithromycin, gentamicin, cefoxitin, cotrimoxazole, ciprofloxacin, clindamycin, doxycycline, penicillin, ampicillin, and fosfomycin. Cefoxitin was used to identify MRSA. For P. aeruginosa, the tested drugs included imipenem, meropenem, piperacillin-tazobactam, cefepime, ceftazidime, amikacin, gentamicin, ciprofloxacin, levofloxacin, aztreonam, and selected agents expected to show intrinsic non-susceptibility.

Detection of ESBL phenotype in Pseudomonas aeruginosa

Multidrug-resistant P. aeruginosa isolates were screened phenotypically for ESBL production using ceftazidime and ceftazidime-clavulanic acid discs placed 15 mm apart from centre to centre. After incubation, a zone increase of at least 5 mm around the clavulanate-containing disc compared with ceftazidime alone was interpreted as ESBL production. This

approach is consistent with phenotypic inhibitor-based ESBL detection principles used in clinical microbiology studies.[8] [9]

Data analysis

The study used descriptive statistics. Frequencies and percentages were calculated for isolates, resistance phenotypes, and antibiotic susceptibility. Because this manuscript was prepared from summarized study tables, advanced statistical modelling was not performed. The ESBL-positive P. aeruginosa proportion is presented as reported in the source results table, while the detailed ESBL-positive versus ESBL-negative resistance comparison is interpreted as a complete-panel subset of phenotypically resistant isolates.

RESULTS

Overall isolate distribution relevant to resistance

Aerobic bacterial culture was positive in 81 of 100 CSOM samples. P. aeruginosa was the leading isolate, recovered from 35% of the total study population. S. aureus followed at 24%. Coagulase-negative staphylococci represented 9%, while Gram-negative enteric bacilli included K. pneumoniae, E. coli, Citrobacter species, and Proteus vulgaris. This isolate distribution is important for empirical therapy because the two leading organisms require different antimicrobial strategies.

Isolate group	Frequency	Percentage of total cases
Pseudomonas aeruginosa	35	35%
Staphylococcus aureus	24	24%
Coagulase-negative staphylococci	9	9%
Klebsiella pneumoniae	5	5%
Escherichia coli	3	3%
Citrobacter species	3	3%
Proteus vulgaris	2	2%
Fungal growth	9	9%
No growth	10	10%

Staphylococcus aureus resistance pattern

Among 24 S. aureus isolates, 17 were methicillin-sensitive S. aureus and seven were MRSA. Therefore, 29.2% of S. aureus isolates were methicillin resistant. This is clinically important because MRSA limits beta-lactam utility and requires careful antibiotic selection. Overall S. aureus susceptibility was highest for linezolid at 80.8%, followed by azithromycin and gentamicin at 76.79% each. Cefoxitin susceptibility was 69.2%, corresponding to the methicillin-sensitive group. Cotrimoxazole sensitivity was 61.5%, ciprofloxacin 57.7%, and clindamycin and doxycycline 38.5% each. Low sensitivity was observed for penicillin and ampicillin at 23.1% each and fosfomycin at 19.2%.

Antibiotic	Sensitive n (%)	Resistant n (%)
Linezolid	20 (80.8%)	4 (19.2%)
Azithromycin	19 (76.79%)	5 (23.1%)
Gentamicin	19 (76.79%)	5 (23.1%)
Cefoxitin	17 (69.2%)	7 (30.8%)
Cotrimoxazole	15 (61.5%)	9 (38.5%)
Ciprofloxacin	14 (57.7%)	10 (42.3%)
Clindamycin	10 (38.5%)	14 (61.5%)
Doxycycline	10 (38.5%)	14 (61.5%)
Penicillin	6 (23.1%)	18 (76.9%)
Ampicillin	6 (23.1%)	18 (76.9%)
Fosfomycin	5 (19.2%)	19 (80.8%)

The MRSA subset retained comparatively better susceptibility to azithromycin and linezolid in the source analysis, but resistance to several commonly used agents remained substantial. These data support mandatory susceptibility testing when S. aureus is isolated from persistent CSOM, especially if patients have prior treatment exposure.

Coagulase-negative staphylococci

Coagulase-negative staphylococci showed complete sensitivity to azithromycin and cefoxitin in the source data, with 71.4% sensitivity to linezolid and ciprofloxacin. Gentamicin, cotrimoxazole, and ampicillin each showed 57.1% sensitivity, while

clindamycin, doxycycline, penicillin, and fosfomycin showed high resistance. Although coagulase-negative staphylococci may sometimes represent colonization, their recovery from chronically discharging ears should be interpreted in clinical context, particularly when isolated in pure growth or associated with inflammatory microscopy.

Pseudomonas aeruginosa susceptibility pattern

P. aeruginosa was the most important resistance target in the study because it was the most frequent isolate and showed variable susceptibility across clinically used antipseudomonal drugs. Imipenem was the most active drug, with 80.0% sensitivity, followed by meropenem at 74.3%. Among non-carbapenem options, amikacin showed 65.7% sensitivity, piperacillin-tazobactam 63.0%, cefepime 62.9%, gentamicin 60.0%, ceftazidime 57.1%, ciprofloxacin 57.1%, levofloxacin 54.3%, and aztreonam 51.4%.

Antibiotic	Sensitive n (%)	Resistant n (%)
Imipenem	28 (80.0%)	7 (20.0%)
Meropenem	26 (74.3%)	9 (25.7%)
Amikacin	23 (65.7%)	12 (34.3%)
Piperacillin-tazobactam	22 (63.0%)	13 (37.0%)
Cefepime	22 (62.9%)	13 (37.1%)
Gentamicin	21 (60.0%)	14 (40.0%)
Ceftazidime	20 (57.1%)	15 (42.9%)
Ciprofloxacin	20 (57.1%)	15 (42.9%)
Levofloxacin	19 (54.3%)	16 (45.7%)
Aztreonam	18 (51.4%)	17 (48.6%)
Ampicillin	0 (0.0%)	35 (100%)
Ceftriaxone	0 (0.0%)	35 (100%)
Cotrimoxazole	0 (0.0%)	35 (100%)

The universal resistance to ampicillin, ceftriaxone, and cotrimoxazole is not unexpected and confirms that these drugs should not be relied on for P. aeruginosa CSOM. More concerning is the moderate resistance to ceftazidime, ciprofloxacin, and levofloxacin, because these agents are commonly used in otological practice. If fluoroquinolone susceptibility continues to decline, empirical topical or systemic fluoroquinolone strategies may become less reliable.

ESBL-producing Pseudomonas aeruginosa

The study reported ESBL-positive P. aeruginosa in 31.4% of P. aeruginosa isolates. In the comparative resistance table available from the source analysis, ESBL-positive isolates showed markedly greater resistance than ESBL-negative isolates. Complete resistance among ESBL-positive isolates was recorded for amikacin, cefepime, ceftazidime, ciprofloxacin, gentamicin, and aztreonam, while piperacillin-tazobactam resistance was 80%. Carbapenem resistance was also higher among ESBL-positive isolates, with 50% resistance to imipenem and meropenem in the comparative subset.

Antibiotic	Resistance in ESBL-positive subset	Resistance in ESBL-negative subset
Imipenem	50%	8%
Meropenem	50%	16%
Amikacin	100%	8%
Cefepime	100%	12%
Ceftazidime	100%	20%
Ciprofloxacin	100%	20%
Gentamicin	100%	16%
Aztreonam	100%	28%
Piperacillin-tazobactam	80%	20%

These findings show that ESBL phenotype in P. aeruginosa is not an isolated cephalosporin-resistance event. It appears to be part of a broader multidrug-resistance pattern affecting aminoglycosides, fluoroquinolones, antipseudomonal cephalosporins, monobactams, and beta-lactam/beta-lactamase inhibitor therapy. The reduced carbapenem susceptibility within the ESBL-positive subset is particularly concerning because carbapenems were otherwise the most active antipseudomonal agents.

Enterobacteriaceae and other Gram-negative bacilli

K. pneumoniae isolates showed 100% sensitivity to piperacillin-tazobactam, meropenem, and cotrimoxazole, while sensitivity to doxycycline was 80%. Amoxicillin-clavulanate, cefotaxime, ciprofloxacin, and ceftriaxone showed 66.7% sensitivity. However, cefepime resistance was 100%, and resistance to amikacin, imipenem, ceftazidime, gentamicin, and cefazolin was 66.7%.

Citrobacter species showed complete sensitivity to piperacillin-tazobactam and moderate sensitivity to several other agents, including amoxicillin-clavulanate, imipenem, levofloxacin, cefotaxime, cotrimoxazole, ciprofloxacin, cefazolin, meropenem, doxycycline, and ceftriaxone. E. coli isolates showed complete sensitivity to piperacillin-tazobactam, ceftriaxone, and cefotaxime, but complete resistance to gentamicin, ciprofloxacin, and aztreonam. Proteus vulgaris isolates showed complete sensitivity to piperacillin-tazobactam, ampicillin, ciprofloxacin, meropenem, and ceftriaxone, but complete resistance to cefepime and ceftazidime. These smaller isolate groups should be interpreted cautiously because the numbers were low.

DISCUSSION

The present resistance-focused analysis confirms that CSOM in this tertiary-care setting cannot be treated as a microbiologically simple infection. P. aeruginosa and S. aureus together accounted for most bacterial isolates, and both included clinically important resistant phenotypes. Similar studies from India have reported that local antibiograms vary considerably, reinforcing that treatment policies should be based on institutional surveillance rather than assumptions imported from other regions.[11] [16] [17] [18] Resistance concerns in otitis media are not limited to chronic disease; systematic evidence from children with acute otitis media and ear discharge also shows that bacterial prevalence and antimicrobial resistance vary across settings, strengthening the argument for local surveillance rather than universal empirical assumptions.[15]

The most important finding is the resistance profile of P. aeruginosa. Imipenem and meropenem were the most active drugs, but their sensitivity rates of 80.0% and 74.3% still indicate that a meaningful minority of isolates were resistant. Carbapenems are often considered reserve agents and should not be used casually for uncomplicated CSOM. Their relatively high activity in this study supports their role in severe or complicated infections when indicated by culture, but the presence of carbapenem resistance among ESBL-positive isolates warns against overdependence.

The moderate activity of piperacillin-tazobactam, cefepime, amikacin, gentamicin, ceftazidime, and fluoroquinolones is clinically relevant. P. aeruginosa resistance is driven by multiple mechanisms, including intrinsic permeability barriers, efflux systems, beta-lactamase production, adaptive stress responses, and biofilm-associated tolerance.[3] [4] In a biofilm-rich chronic ear environment, in vitro susceptibility may not always translate into rapid clinical clearance. Therefore, treatment should include appropriate aural toileting and assessment for anatomical disease, not antibiotic therapy alone.[2]
The ESBL finding is particularly important. ESBL-positive P. aeruginosa showed broad resistance across multiple drug categories. Reports from Indian tertiary-care settings have documented ESBL production in P. aeruginosa and emphasized phenotypic screening because failure to detect ESBL may lead to ineffective cephalosporin therapy.[8] [9] [12] Recent studies of ear infections from other regions have also identified ESBL-producing bacteria and multidrug-resistant pathogens as emerging problems in otological infections.[13] [14] The present data add Central Indian institutional evidence to this concern.

The MRSA rate of 29.2% among S. aureus isolates also has practical implications. MRSA in CSOM may lead to failure of beta-lactam therapy and can complicate outpatient management. Cefoxitin-based detection is a practical and reliable phenotypic approach, and its inclusion in routine CSOM panels is justified.[10] The relatively higher activity of linezolid should be interpreted cautiously because linezolid is generally a reserve systemic agent and not an automatic first-line choice for ear discharge. Culture results should be interpreted together with disease severity, route of therapy, ototoxicity concerns, tympanic membrane status, and the feasibility of topical treatment.

The resistance patterns among smaller Gram-negative groups are hypothesis-generating rather than definitive. The complete sensitivity of several Enterobacteriaceae to piperacillin-tazobactam or meropenem may reflect small sample size. Conversely, complete resistance in two or three isolates should not be overgeneralized. Nevertheless, these findings are useful for local surveillance because even small numbers can signal emerging resistance when tracked over time.

A major implication of this study is the need for a CSOM-specific antimicrobial policy. General hospital antibiograms may be dominated by urinary, bloodstream, or respiratory isolates and may not accurately represent chronic ear-discharge organisms. CSOM isolates are exposed to unique pressures, including topical antibiotic use, recurrent moisture, canal

colonization, and biofilm formation. Therefore, a separate otological antibiogram prepared at regular intervals would help clinicians choose empirical therapy more rationally while awaiting culture reports. A practical policy could stratify patients into uncomplicated, recurrent, previously treated, and complicated categories. Uncomplicated cases may be managed initially with meticulous aural toilet and locally recommended topical therapy, but recurrent discharge, previous antibiotic exposure, granulation tissue, suspected cholesteatoma, diabetes, immunosuppression, or severe otalgia should trigger culture before escalation. Laboratory reports should clearly identify P. aeruginosa, MRSA, ESBL phenotype, and multidrug resistance, because these findings alter both treatment choice and follow-up intensity.

Antimicrobial stewardship in CSOM should follow several principles. First, repeated empirical antibiotic use should be avoided when discharge persists. Second, bacterial culture and susceptibility testing should be obtained before escalating therapy in recurrent or nonresponding cases. Third, fungal culture should be considered when bacterial cultures are negative or when discharge persists after antibacterial therapy. Fourth, reserve antibiotics should be protected and used only when culture results, clinical severity, or complications justify them. Finally, clinicians should combine antimicrobial therapy with local care, patient education, follow-up, and surgical evaluation when structural disease is suspected.[19]

Limitations

The study was limited to a single tertiary-care hospital, and the findings may not fully represent community-level CSOM. The sample size for non-Pseudomonas Gram-negative bacilli was small, limiting interpretation of percentage susceptibility in those groups. Anaerobic culture was not performed, so anaerobic pathogens and their resistance patterns were not assessed. Molecular confirmation of ESBL genes, carbapenemase genes, or mecA-mediated MRSA was not included. Long-term clinical response, recurrence, and hearing outcomes were also unavailable.

CONCLUSION

This resistance-focused analysis shows that CSOM isolates from a tertiary-care hospital in Central India include important resistant pathogens. P. aeruginosa was the leading organism and showed best susceptibility to imipenem and meropenem, but ESBL-positive isolates demonstrated extensive multidrug resistance, including reduced carbapenem susceptibility in the comparative subset. S. aureus was the second most common organism, with MRSA accounting for nearly one-third of S. aureus isolates. These findings strongly support routine culture, susceptibility testing, MRSA screening, and phenotypic ESBL detection in persistent or recurrent CSOM. Local CSOM-specific antibiograms should guide empirical choices and reduce unnecessary exposure to ineffective or reserve antibiotics.

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