REVIEW ARTICLE

Chronic suppurative otitis media: A review

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Summary

Objectives: Chronic suppurative otitis media (CSOM) remains one of the most common childhood chronic infectious diseases worldwide. Although microbial, immunological, and genetically determined factors, as well as Eustachian tube characteristics, are supposed to be involved in the pathogenesis of CSOM, many aspects of the pathogenesis of CSOM still need to be clarified. Optimal treatment strategy has not been established yet. The objective of this review is to present and evaluate the current state of knowledge of CSOM.

Design: Systematic narrative review.

Methods: A PubMed search (1966—January 2005) was performed for studies on epidemiology, pathogenesis, clinical management, and complications of CSOM. All included articles were categorized according to level of evidence.

Results: Five hundred and fifty papers were identified, of which 79 were found to be relevant for this review. The definition of CSOM was found to vary. CSOM is a multifactorial disease. Regarding management of CSOM, there is no consensus as to what the optimal management strategy should entail. No convincing evidence is available for most medical and surgical therapies. Topical quinolones have proven effective, but need further monitoring regarding adverse effects.

Conclusions and recommendations: Important goals in research of CSOM should be achieving consensus about the definition of CSOM and gaining more in-depth knowledge of the pathogenesis of CSOM, especially the role of innate and adaptive immunity. There is also a need for further well-designed studies on the effectiveness of various management strategies for CSOM.

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1. Introduction

Chronic suppurative otitis media (CSOM) remains one of the most common childhood chronic infectious diseases worldwide, affecting diverse racial and cultural groups both in developing and industrialized countries. It involves considerable morbidity and can cause extra- and intracranial complications [1–5].

There are still many questions about the pathogenesis of CSOM and consequently about what the optimal management — medical and/or surgical — should entail.

In this article, the current state of knowledge in epidemiology, pathogenesis, complications, and management of CSOM is reviewed systematically from a clinical perspective, the ultimate aim being to provide the clinician a tool in the management of this condition. In this connection, future research goals will be identified.

2. Search strategy and selection criteria

A PubMed search was done for articles dating from 1966 to January 2005 with the MESH heading “Otitis media, Suppurative” in combination with text- and keywords complications, drug therapy, epidemiology, etiology, genetics, history, immunology, microbiology, pathology, physiopathology, surgery, and therapy. All studies in English on chronic suppurrative otitis media in children and adults, containing these text- and keywords were considered for inclusion.

After critical assessment of the abstracts identified by the initial search, the full content of all potentially relevant papers was reviewed for final selection and data extraction.

The following articles were excluded: those dealing with topics other than CSOM, e.g. cholesteatoma; those focussing on technical aspects of surgery in CSOM; those with information included in more recent updates on CSOM.

Subsequently, a review of identified report bibliographies and a manual search of standard textbooks on ENT surgery was undertaken.

3. Results

The search resulted in 550 citations, which after applying the criteria for inclusion and exclusion left 79 articles for inclusion; 42 additional references were also used. The articles were independently categorized by two authors (M.V. and M.M.R.) in levels of evidence (Table 1) [6].

4. Definition

The textbook definition of CSOM is chronic inflammation of the middle ear and mastoid mucosa in which the tympanic membrane is not intact (perforation or tympanostomy tube) and discharge (otorrhea) is present [7–9]. There is, however, no consensus about the duration of the symptoms. The World Health Organisation (WHO) [10] defines CSOM as “otorrhea through a perforated tympanic mem-
brane present for at least 2 weeks”, while others define “chronic” as symptoms persisting for more than 6 weeks [1,11—14]. Since it is accepted that CSOM is preceded by acute otitis media (AOM) treated either incomplete or unsuccessfully [11,15,16], these variations in definition of duration of symptoms suggest that the transition from otorrhea as a sign of AOM to that of CSOM is not clearly established.

CSOM should be distinguished from tympanostomy tube otorrhea (TTO), which is the most common complication of tympanostomy tube placement [17,18], but as with AOM and CSOM, the transition from TTO to CSOM is not well defined. At the same time, CSOM should be distinguished from chronic OME (otitis media with effusion), in which no perforation or active infection is present, as well as from a chronic perforation of the tympanic membrane (TM), in the absence of middle-ear infection [19]. If a cholesteatoma is present, the term chronic suppurative otitis media with cholesteatoma is used.

5. Epidemiology

The divergent definitions of CSOM and the inclusion of patients with cholesteatoma in reported prevalences of CSOM, preclude an accurate estimate of the true prevalence and incidence of CSOM. CSOM most often occurs in the first 5 years of life [20], and it is most common in developing countries, in special populations such as children with craniofacial anomalies [21], and in certain racial groups [19]. Highest prevalences of CSOM in children are reported among the Inuits of Alaska, Canada and Greenland, American Indians, and Australian Aborigines, and range from 7% to 46% [19,22—25]. Intermediate prevalences are reported in the South Pacific Islands, Africa, Korea, India, and Saudi Arabia, ranging from 1% to 6% [19,26—28]. The lowest prevalences are found in highly developed industrial countries such as the UK and the US: <1% [19,29].

6. Risk factors

Fliss et al. [30] have identified a history of acute and recurrent otitis media, parental history of chronic otitis media, and crowded conditions (i.e. large families with several siblings, large day care centres) as significant risk factors for CSOM. They could not establish an association between CSOM and allergy, recurrent upper respiratory infections, breastfeeding, sex, parental age, or passive smoking. From a clinical perspective, however, some of these risk factors for AOM are likely to play a role in CSOM [31—33]. No quantitative data on risk factors for CSOM, such as odds ratios or prognostic models that can predict which children will develop CSOM, are available.

7. Pathogenesis

The pathogenesis of CSOM is multifactorial: environmental versus genetically determined factors as well as anatomical and functional characteristics of the Eustachian tube are involved. The following paragraphs describe the main causative factors for CSOM in greater detail.

### Table 1 References categorized in level of evidence

<table>
<thead>
<tr>
<th>Level of evidence</th>
<th>Study design</th>
<th>References (PubMed search)</th>
<th>References (additional)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a</td>
<td>Systematic review of RCTs&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>1b</td>
<td>Individual RCT&lt;sup&gt;a&lt;/sup&gt; (good quality)</td>
<td>—</td>
<td>—</td>
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<tr>
<td>2a</td>
<td>Systematic review of cohort studies</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>2b</td>
<td>Individual cohort study, incl. cohort of cases; RCT&lt;sup&gt;a&lt;/sup&gt; of low quality, e.g. &lt;80% follow-up, allocation bias, low power</td>
<td>18</td>
<td>8</td>
</tr>
<tr>
<td>3a</td>
<td>Systematic review of case-control studies</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3b</td>
<td>Individual case-control study</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>3c</td>
<td>Surveys</td>
<td>15</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>Case-series, poor-quality cohort, and case-control studies</td>
<td>22</td>
<td>9</td>
</tr>
<tr>
<td>5</td>
<td>Expert opinions</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>Other</td>
<td>e.g. in vitro studies, animal models, book chapters, etc.</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>79</td>
<td>42</td>
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</tbody>
</table>

<sup>a</sup> RCT: randomised controlled trial.
8. Eustachian tube function

The Eustachian tube has three important functions with respect to the middle ear: ventilation, protection, and clearance. Both endogenous and exogenous factors can impair these functions and therefore cause OM (otitis media) [5,19,34,35]. When a perforation of the tympanic membrane is present, either spontaneously or due to a tympanostomy tube, the middle ear “gas cushion” is lost, resulting in reflux of nasopharyngeal secretions through the Eustachian tube and consequent contamination of the middle ear with potential respiratory pathogens [11,19,35]. Infants and young children are especially at risk for such reflux because their Eustachian tubes are short, horizontal, and “floppy” [19,35]. Similarly, Down syndrome and craniofacial anomalies such as cleft palate affect both the anatomy and function of the Eustachian tube and so predispose to CSOM [21]. Yuceturk et al. [34] studied Eustachian tube function (automatic Toynbee test, tympanometry, Valsalva’s manoeuvre) in 60 ears with CSOM and 146 control ears, finding Eustachian tube dysfunction in 72% (95% CI, 61—83) versus 35% (95% CI, 27—43), respectively, ($p < 0.05$).

Reduced ciliary function of the middle ear and Eustachian tube mucosa has been associated with impairment of clearance of middle-ear secretions and may, therefore, facilitate the progression from AOM/OME into CSOM [36,37]. Gastroesophageal reflux may also contribute to Eustachian tube dysfunction and subsequent middle-ear infection [38,39].

9. Microbiology

In CSOM, bacteria can reach the middle ear either from the nasopharynx through the Eustachian tube or from the external ear canal through a non-intact tympanic membrane [11,19,35]. The aerobic microorganisms most frequently isolated in CSOM are Pseudomonas aeruginosa (in 18—67% of ears), Staphylococcus aureus (14—33%), Gram-negative organisms, such as Proteus spp., Klebsiella spp., and Escherichia spp. (4—43%), and Haemophilus influenzae (1—11%) [13,15,40—49]. The most frequently isolated anaerobic organisms are Bacteroides spp. (1—91%) and Fusobacterium spp. (4—15%) (Table 2) [42—47]. In CSOM, the middle-ear environment is thought to be more tolerant to unusual organisms like P. aeruginosa, S. aureus, and anaerobes; therefore, it is still uncertain whether these bacteria are true pathogens in CSOM or might reflect secondary invaders or contamination from the external auditory canal [2,7]. The large variability in recovery rates of aerobic and anaerobic bacteria may be related to differences in timing of sampling during the course of the disease, prior use of antibiotics, and differences in sampling- and processing techniques, e.g. sterilizing the audi-

### Table 2 Most frequently isolated aerobic and anaerobic microorganisms in chronic suppurative otitis media (percentage per ear)

<table>
<thead>
<tr>
<th>Author [ref.]</th>
<th>Aerobic micro-organisms</th>
<th>Anaerobic micro-organisms</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pseudomonas aeruginosa</td>
<td>Bacteroides spp.</td>
</tr>
<tr>
<td></td>
<td>Staphylococcus aureus</td>
<td>Fusobacterium spp.</td>
</tr>
<tr>
<td></td>
<td>Gram-negative rods</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Haemophilus influenzae</td>
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</table>

<table>
<thead>
<tr>
<th>Author [ref.]</th>
<th>Aerobic micro-organisms</th>
<th>Anaerobic micro-organisms</th>
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<tr>
<td>Kenna et al. [15]</td>
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<td>Fliss et al. [48]</td>
<td>62 14 7—15 10—11</td>
<td>n/a n/a</td>
</tr>
<tr>
<td>Arguedas et al. [13]</td>
<td>26 14 14—22 1</td>
<td>n/a n/a</td>
</tr>
<tr>
<td>Indudharan et al. [40]</td>
<td>36 31 10—15 8</td>
<td>n/a n/a</td>
</tr>
<tr>
<td>Brook and Yocum [46]</td>
<td>20 22 20—43 9</td>
<td>24—61 15</td>
</tr>
<tr>
<td>Brook [44]</td>
<td>22 15 15—35 9</td>
<td>17—71 8—11</td>
</tr>
<tr>
<td>Miro [41]</td>
<td>18 25 5—10 2d</td>
<td>n/a n/a</td>
</tr>
<tr>
<td>Jonsson et al. [42]</td>
<td>30 30 21e 2</td>
<td>23 4</td>
</tr>
<tr>
<td>Erkan et al. [43]</td>
<td>37 23 21—40 n/a</td>
<td>11—34 5</td>
</tr>
<tr>
<td>Papastavros et al. [45]</td>
<td>55 29 6—9 n/a</td>
<td>5—10 n/a</td>
</tr>
<tr>
<td>de Uzeda and Rocha [47]</td>
<td>18 33 31—42g 9</td>
<td>24—91 7—11</td>
</tr>
<tr>
<td>Ikebwe et al. [49]</td>
<td>25 23 4—7g 7</td>
<td>1 n/a</td>
</tr>
</tbody>
</table>

* Minimum and maximum (min—max) data are given when more than one Gram-negative rod or species was identified.
* Data on Klebsiella species not provided.
* Only total number of Haemophilus spp. is provided.
* Data on Klebsiella spp. and Escherichia spp. not provided.
* Percentage per case.
* Data on Escherichia spp. not provided.
tory canal before sampling, transport media, or delays in inoculation [2,7,11]. Fungi are also thought to play a role in CSOM, especially Aspergillus spp. and Candida spp. [50,51]. In some populations, especially those inhabiting hot, humid regions where fungi may well flourish, fungi are isolated in 50% of cases with CSOM [50,51].

Recently, concern has risen about secondary fungal overgrowth as a complication of treatment with quinolone eardrops [52].

Recently, bacterial biofilms have gained attention as a source of chronic infections. A biofilm is a population of bacterial cells growing on a surface, enclosed in an exopolysaccharide matrix; being difficult to eradicate, they could be the source of persistent infections [53,54]. Biofilms may attach to damaged tissue, such as exposed osteitic bone and ulcerated middle-ear mucosa, or to otologic implants such as tympanostomy tubes, and are therefore thought to cause persistent infection in CSOM [11,54—56].

10. Immunology and genetics

In general, immunoglobulins IgG and IgA are most important in the defence against mucosal infections like CSOM. Secretory IgA (SIgA) is synthesized locally by plasma cells in the mucosa of the middle-ear cavity and may be important in preventing bacteria from attaching to and colonizing the middle-ear mucosa. Children with CSOM may lack SIgA [57]. IgG-class immunoglobulins facilitate phagocytosis directly or via complement activation. IgG and IgG-subclass concentrations depend on age [58]. Children with recurrent upper respiratory infections may have low specific IgG-subclass (mostly IgG2) antibody levels (10—20% of cases) [59,60]. For CSOM no such data are available. An essential condition for immunoglobulins to act is their adherence to the bacterial wall, i.e. coating [58]. In CSOM, intense SIgA and IgG coating of bacteria is common, but when P. aeruginosa is the causative agent of the infection, no bacterial coating is seen. This may well explain why infections caused by P. aeruginosa are so difficult to eradicate [57,61].

Although many have reported on inflammatory mediators in AOM and OME, evidence about the specific role of such factors in CSOM, like local cytokine production, is not yet available.

Genetic determinants of CSOM are still unknown. Against the background of twin studies in OM, showing a higher concordance rate in OM for monozygotic twins than for dizygotic twins, a genetic component is also likely for CSOM [62,63].

Low serum levels of mannose-binding lectin (MBL) and polymorphisms of Fc gamma receptor have been associated with recurrent upper respiratory infections and otitis media in childhood [64—66]. Further research into these and other modifier genes is necessary to confirm their role in CSOM.

11. CSOM in systemic conditions

CSOM may occur as a part of a systemic condition, e.g. M. Wegener, tuberculosis, and Histiocytosis X [21,67,68], where the mastoid and middle ear may be the localization of this specific inflammation.

12. Complications and sequelae of CSOM

The most common sequela of CSOM is hearing loss, either conductive or sensorineural; this may affect a young child’s language development and school progress. Chronic infection of the middle ear, causing oedema of the middle-ear lining and discharge, tympanic membrane perforation, and possibly ossicular chain disruption, results in a conductive hearing loss ranging from 20 to 60 dB [7,69,70].

There is some evidence that CSOM causes sensorineural hearing loss. Animal studies have shown that inflammatory mediators, penetrating into the inner ear through the round window membrane, can cause the loss of hair cells in the cochlea [71,72]. A recent study in humans has shown loss of outer and inner hair cells in the basal turn of the cochlea in patients with CSOM [73].

Four studies have been identified reporting on sensorineural hearing loss in CSOM.

In one retrospective study of 218 patients (average age 35 years) with unilateral CSOM the bone conduction threshold was 9—14 dB lower in diseased ears than in healthy ears [74]. In another retrospective study of 121 patients (average age 37 years) with unilateral CSOM, the bone conduction threshold was 10—12 dB in the diseased ear compared to 3—4 dB in the healthy ear [75].

On the other hand, in two prospective studies, one of 286 patients with unilateral CSOM (average age 50 years) and the other of 87 children with bilateral CSOM (average age 5.5 years), no effect of the infection on bone conduction thresholds was found [69,76]. No evidence was found suggesting that children are more susceptible to develop sensorineural hearing loss in CSOM than adults [69,74—76].

CSOM can result in serious extracranial and intracranial complications. The reported overall extra-
and intracranial complication rate in CSOM varies from 0.7% to 3.2%; extracranial complications alone from 0.5% to 1.4% and intracranial complications from 0.3% to 2.0% [3,79]. The incidence of complications appeared higher in pediatric patients than in adults with CSOM. With CSOM being more frequent in children, however, no adequate comparison between complication rates in children and adults can be made.

The most frequent extracranial complications are facial paralysis, subperiosteal abscess, mastoiditis, and labyrinthitis, with reported incidences of 13—58%, 40—68%, 14—74%, 7—34% of all extracranial complications, respectively [3,4,77—80].

The most common intracranial complications of CSOM are meningitis, cerebral abscess, lateral sinus thrombosis, extradural abscess, otic hydrocephalus, and encephalitis, with reported incidences of 21—72%, 18—42%, 2—26%, 7—16%, 5—11%, and 2% of all intracranial complications, respectively [3,4,77—80].

### 13. Clinical management

#### 13.1. Topical treatment

In developing countries, antiseptic drops, e.g. aluminium acetate, boric acid, iodine powder, and povidone-iodine are commonly used for CSOM because of their low cost and availability [81—83]. Eardrops containing antimicrobial agents either with or without an anti-inflammatory component have been promoted as an effective therapy for CSOM since the 1950s [84—86]. Since the 1990s, fluoroquinolone drops have become available [33,41,87—92].

The effectiveness of ototopical drops was evaluated in a Cochrane Review [82]. CSOM was defined according to WHO criteria, i.e. otorrhea through a perforated tympanic membrane present for at least 2 weeks. Overall success percentages (dry ear) of ototopical drops varied from 40% to 100%. It was concluded that treatment with antibiotic or antiseptic eardrops accompanied by aural toilet was more effective in resolving otorrhea than no treatment (OR 0.37, 95% CI, 0.24—0.57) or aural toilet alone (OR 0.31, 95% CI, 0.23—0.43). Topical antibiotics were not more effective than topical antiseptics (OR 1.34, 95% CI, 0.64—2.81), and topical treatment with antibiotic or antiseptic eardrops was more effective than systemic antibiotics (OR 0.46, 95% CI, 0.30—0.69). The benefit of combinations of topical antibiotics and steroids as compared to topical antibiotics alone has not been evaluated formally. Combined treatment with topical and systemic antibiotics was not more effective than treatment with topical antibiotics alone in terms of otorrhea resolution (OR 1.71, 95% CI, 0.88—3.34). Topical quinolones appeared to be more effective than non-quinolone eardrops (OR 0.26, 95% CI, 0.16—0.41).

Another systematic review by Abes et al. [93] also showed that quinolone eardrops were more effective than non-quinolones. They found a 2.67 times higher cure rate with topical 0.3% ofloxacin otic solution, than with other topical or systemic antibiotics (95% CI, 2.04—3.50). In a third non-systematic review, however, the authors stated that topical quinolones were not superior to topical aminoglycosides [94].

The overall quality of the studies included in these three reviews [82,89,93—97], was generally considered low. The definition of CSOM, duration of discharge (3 weeks—40 years), age range of the patients (21 months—79 years) and follow-up varied considerably. No consistent relation between duration of CSOM or age of the patients and outcome was found. In all studies, outcome was defined as resolution of otorrhea; no data regarding the quality of the tympanic membrane or hearing were given.

The risk of ototoxicity by ototopical preparations has been the subject of discussion for many years [84,98]. Potential ototoxicity of antibiotics, solvents, and antiseptics has been demonstrated in animal studies, but information regarding these adverse effects in humans is scarce [85]. In the studies reviewed by Acuin et al. [82] negligible rates of ototoxicity were found.

Secondary fungal overgrowth causing otitis externa has been reported as a side effect of treatment with topical quinolones. With the growing enthusiasm for the use of these eardrops, the incidence of this complication is found to increase [52].

#### 13.2. Systemic treatment

Systemic antibiotics are advised both as initial therapy for CSOM [21,51,96] and as secondary when topical therapy fails [1,7,11,15,99—102]. In Table 3, the results of the available studies on systemic treatment of CSOM are summarized; the success rate of systemic antibiotics appears to be quite high, approximately 70%. Overall, the methodological quality of these studies was low. Because of heterogeneity of study populations and study design the data could not be pooled, nor could subgroups of patients with better or poorer outcome be identified.

An expert panel called together by the American Academy of Otolaryngology—Head and Neck Surgery concluded recently that systemic therapy should only be considered in patients with CSOM.
showing signs of complicated or invasive infections or signs of systemic disease [84]. Consensus is lacking as to which antibiotic to use systemically as well as about the duration of treatment in CSOM [11,103,104]: both broad-spectrum antibiotics, as well as culture-directed therapy, have been advocated as initial oral therapy for CSOM [11,103].

### 13.3. Surgical treatment

Tympanomastoidectomy has been advocated as the surgical treatment of choice in CSOM since the 1970s [7,16,105]. However, no prospective, randomised, controlled trials justifying this guidance have been published [82]. Only three retrospective studies on surgical treatment for CSOM are available. Vartiainen et al. [105] reported the outcome of 221 ears with CSOM in children and adults managed with either a one-stage tympanomastoidectomy (84%) or a mastoidectomy, with planned second-stage tympanoplasty (15%). The overall success rate, defined as dry ear plus an intact, mobile eardrum, was 73% (95% CI, 67—79). No differences were found between results in children and adults. Another report by the same authors, limited to children with CSOM, showed an equally successful outcome of (tympano)mastoidectomy, namely 74% (95% CI, 59—89) [16]. Balyan et al. [106] analyzed the surgical outcome in 323 patients (age range 4—68 years) with CSOM managed by: (I) tympanoplasty and mastoidectomy (discharging ears); (II) tympanoplasty alone (discharging ears); or (III) tympanoplasty alone (dry ears). Graft success rates in groups I—III were 91% (95% CI, 83—98), 86% (95% CI, 73—99), and 90% (95% CI, 85—93), respectively. Mean residual air-bone gaps were 17, 20, and 19 dB, respectively. The success percentage of surgery appeared to be higher in children below 16 years of age. The effect of duration of symptoms on outcome was not studied. The authors concluded that results of tympanoplasty combined with mastoidectomy are no better than tympanoplasty alone in patients with CSOM.

Studies comparing surgical versus medical therapies for CSOM are not available.

Regarding the timing of mastoid surgery and tympanoplasty for CSOM in children, opinions vary. Procter [9] recommends that mastoid surgery should be delayed until puberty when possible, while Bluestone et al. [7] and Vartiainen [16,105] consider mastoid surgery indicated in all cases of CSOM that do not respond to conservative treatment, regardless of age.

Regarding tympanoplasty alone in children with CSOM, some authors recommend this operation only in children older than 10—12 years of age because of

<table>
<thead>
<tr>
<th>Author</th>
<th>N (Average age range)</th>
<th>Type of study</th>
<th>Antibiotic type</th>
<th>Duration of treatment</th>
<th>Type of surgery</th>
<th>Route of administration</th>
<th>Initial/secondary</th>
<th>Success rate (%)</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Khanna et al. [51]</td>
<td>110 (n/a)</td>
<td>RCT</td>
<td>Various types of AB</td>
<td>14 Days</td>
<td>Initial</td>
<td>Oral</td>
<td>2 Weeks</td>
<td>85 vs. 75</td>
<td>2 Weeks</td>
</tr>
<tr>
<td>Fliss et al. [1]</td>
<td>48 (4—12 years)</td>
<td>RCT</td>
<td>Ceftazidime vs. aztreonam</td>
<td>10—14 Days</td>
<td>Secondary</td>
<td>IV</td>
<td>12 Days</td>
<td>100 Both</td>
<td>12 Days</td>
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<td>36 (1—17 years)</td>
<td>Case report</td>
<td>Various types of AB</td>
<td>3—35 Days</td>
<td>Secondary</td>
<td>IV</td>
<td>4—20 Months</td>
<td>89 vs. 76</td>
<td>4—20 Months</td>
</tr>
<tr>
<td>Dagan et al. [101]</td>
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<td>Cohort of cases</td>
<td>Ceftazidime</td>
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<td>IV/M</td>
<td>12 Months</td>
<td>67 vs. 53</td>
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<td>Case report</td>
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<td>RCT</td>
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<td>Oral</td>
<td>12 Months</td>
<td>73 vs. 53</td>
<td>12 Months</td>
</tr>
</tbody>
</table>

* RCT: randomised controlled trial.
a higher incidence of surgical failures in younger children [107,108]. Other authors state that age does not affect the outcome of tympanoplasty in children with CSOM [16,109,110].

In cases of therapy-resistant CSOM radical mastoidectomy may be considered, with or without mastoid obliteration [111—114]. In one retrospective case study of 16 patients (average age 44 years) who had undergone a radical revision mastoidectomy, 80% obtained a dry ear (95% CI, 60—99) [111]. In another case-control study of 30 patients with CSOM who had been managed by revision mastoidectomy with mastoid obliteration using a temporoparietal fascial flap, 96% (95% CI, 89—100) had a dry ear at 12 months follow-up versus 10% (95% CI, 1—21) of 30 conservatively treated patients with CSOM [114].

14. Hearing revalidation

Bone-anchored hearing aids (BAHA) became available in the 1980s. They are considered a good alternative to conventional bone-conduction hearing aids in patients with chronic draining ears [115—118]. A retrospective study of 69 patients with CSOM previously using a conventional hearing aid showed a reduction of discharge in 84% of patients following fitting with a BAHA; 58% of patients were more satisfied with their BAHA than with their previous aid [116]. Audiologically, patients with CSOM do not perform better with BAHAs than with air conducting aids [115,118].

Up till the 1980s CSOM was considered a contraindication for cochlear implantation because of the risk of spread of the infection to the intracranial space [119]. However, two recent studies which included 19 patients with CSOM, one with an average follow-up period of 18 months and the other 4 years, showed that cochlear implantation could be safely achieved in these patients [120,121].

15. Discussion

A large number of studies have been published about CSOM, but the great variability of these studies precluded pooling of the results in a meta-analysis or a systematic review. Therefore, this review is narrative. As such, it might be prone to bias in the selection of articles, interpretation of results, and recommendations.

To minimize such bias, we performed an elaborate PubMed search using a MESH heading, which ensured us of finding all articles about suppurative otitis media. We included all articles that provided us actual and relevant information about CSOM; all other subjects than CSOM were excluded. This study shows that most articles on CSOM are expert opinions or case-series, using very variable definitions of CSOM, small study populations, and short follow-up duration, hence overall yielding poor evidence.

We, therefore, believe that future research in CSOM should be directed to:

1. Achieving consensus regarding the definition of the disease, including its duration. The WHO definition of CSOM as “otorrhea present for at least 2 weeks”, is not of much help to the clinician who is generally faced with patients with a much longer duration of symptoms. The various stages of this disease, especially the transitional phase from AOM to CSOM, need to be defined more clearly.

2. Identifying the risk factors and pathogenesis of CSOM. It is assumed that factors involved in AOM also have an important role in CSOM, but good evidence for this assumption is lacking. Optimal management of the disease and advice to the parents should be based on the knowledge of the risk factors and pathogenesis of CSOM. In particular, the role of innate and adaptive immunity, e.g. modifier genes, as well as the role of bacterial biofilms needs further study, as these can be targets for new therapies.

3. Developing prognostic models including factors that can predict which children will develop CSOM.

4. Since so few well-designed studies of current medical and surgical therapies are available, management of CSOM is still controversial. Topical quinolones are a promising option in the management of CSOM. Prospective and well-controlled studies are needed to establish the role of both medical and surgical therapies, including the optimal duration of treatment, for various stages of CSOM.

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