

PROGNOSTIC SIGNIFICANCE OF CLINICAL, ELECTROPHYSIOLOGICAL, AND IMMUNOLOGICAL CHANGES IN FACIAL NERVE NEUROPATHIES ASSOCIATED WITH CHRONIC SUPPURATIVE OTITIS MEDIA

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Scientific specialty: 1st profile — NEUROLOGY; 2nd profile — OTORHINOLARYNGOLOGY

Abstract. Facial nerve neuropathy associated with chronic suppurative otitis media (CSOM) represents a clinically important peripheral neuropathy in which intratemporal inflammation, compression, ischemia, demyelination, and axonal injury may coexist. The neurological outcome is not determined by otological findings alone; it also depends on the severity of motor dysfunction, axonal preservation, denervation and reinnervation dynamics, brainstem reflex-arc integrity, and the biological intensity of chronic inflammation. This integrative review analyzes the prognostic significance of House–Brackmann and Sunnybrook grading, electroneurography/electromyography, blink-reflex parameters, temporal-bone disease characteristics, and inflammatory markers including IL-6, TNF- α , and CRP. Current evidence supports clinical grading and neurophysiological testing as the core of neurological prognostication. Cholesteatoma and destructive bony changes may aggravate intratemporal facial nerve injury. IL-6 and TNF- α are biologically relevant to chronic middle-ear inflammation and tissue destruction, but direct evidence for their independent prediction of facial nerve functional recovery remains limited. A neurologically centered, multidomain assessment is therefore the most coherent framework for future prospective studies.

Keywords: facial nerve neuropathy, chronic suppurative otitis media, peripheral facial palsy, electroneurography, electromyography, blink reflex, House–Brackmann, Sunnybrook, IL-6, TNF- α , prognosis.

INTRODUCTION

The facial nerve is a mixed cranial nerve with a dominant motor role in facial expression. Peripheral facial nerve injury can result in weakness of the facial musculature, impaired eye closure, oral incompetence, altered facial symmetry, and, during incomplete or misdirected recovery, synkinesis and contracture. From a neurological perspective, the clinically visible paresis is only the surface manifestation of a spectrum that ranges from transient conduction block and demyelination to severe axonal degeneration and incomplete reinnervation [1–4].

Chronic suppurative otitis media is an uncommon but clinically important cause of acquired peripheral facial nerve dysfunction. Persistent middle-ear and mastoid inflammation, granulation tissue, cholesteatoma, erosion of the fallopian canal, and temporal-bone destruction may expose the intratemporal facial nerve to inflammatory, compressive, and ischemic mechanisms [5,6]. The resulting neuropathy is therefore best understood as a peripheral nerve disorder occurring within a specific otological disease environment.

Clinical assessment remains essential. The House–Brackmann scale provides a globally accepted six-grade description of facial function [1], whereas the Sunnybrook Facial Grading System offers a more granular evaluation of resting symmetry, voluntary movement, and synkinesis [2]. However, neither system directly quantifies axonal preservation. Electroneurography (ENoG), facial nerve conduction studies, needle electromyography (EMG), and blink-reflex testing can add objective neurophysiological information and may refine prognostic assessment [3,4,7].

Inflammatory biology represents a further layer. Cytokines such as IL-6 and TNF- α are expressed in cholesteatoma and chronic otitis media and have been linked to inflammatory tissue remodeling and, in some studies, bone destruction [8–12]. Whether these markers also provide independent information about facial nerve recovery is not established. This uncertainty creates a clinically relevant research opportunity: linking neuroinflammation to neurological severity and electrophysiological degeneration rather than studying inflammatory markers in isolation.

Accordingly, the primary scientific profile of this topic is neurology, because the main outcome is the functional recovery of a peripheral cranial nerve. Otorhinolaryngology defines the etiological and anatomical disease context.

OBJECTIVE

To analyze the prognostic significance of clinical, electrophysiological, and immunological changes in facial nerve neuropathies associated with chronic suppurative otitis media, with emphasis on a neurologically centered framework for evaluating functional recovery.

MATERIALS AND METHODS

This paper was prepared as a targeted analytical review. The methodological framework was based on the domains defined for the dissertation topic: clinical-neurological assessment using the House–Brackmann and Sunnybrook systems; neurophysiological evaluation using ENoG/nerve conduction, EMG, and blink-reflex testing; otological assessment of chronic suppurative otitis media and temporal-bone changes; and evaluation of inflammatory activity using IL-6, TNF- α , and CRP.

PubMed-indexed literature was selectively reviewed using combinations of the terms facial nerve neuropathy, facial paralysis, chronic suppurative otitis media, cholesteatoma, electroneurography, electromyography, blink reflex, House–Brackmann, Sunnybrook, IL-6, TNF-alpha, and prognosis. The aim was not to perform a formal systematic review or meta-analysis, but to synthesize clinically relevant evidence that can support a prospective neurological study of CSOM-associated facial nerve neuropathy.

Evidence was organized into four prognostic domains: (1) clinical-neurological severity and secondary motor phenomena; (2) electrophysiological evidence of axonal loss, denervation, and reinnervation; (3) otological and radiological indicators of destructive middle-ear disease; and (4) inflammatory markers reflecting the biological activity of chronic disease. Functional facial nerve recovery, residual paresis, synkinesis, and contracture were considered the principal neurological outcomes.

RESULTS

Clinical grading is the first layer of prognostic assessment. Higher House–Brackmann grades reflect greater global motor dysfunction, while Sunnybrook scoring provides additional sensitivity to regional movement and synkinesis [1,2,13]. Recent neurological

data support the value of combining clinical grading with neurophysiology rather than relying on a single scale [7]. In CSOM-associated neuropathy, the same principle is particularly relevant because the severity of the visible facial weakness may not fully reflect the degree of intratemporal axonal injury.

Electrophysiological tests provide the strongest objective link between neurological impairment and prognosis. A marked reduction in CMAP amplitude suggests loss of functioning motor axons. Needle EMG can demonstrate active denervation and later reinnervation. Grosheva et al. showed that EMG, especially when repeated during the course of facial palsy, has substantial prognostic value and may outperform a single ENoG assessment in identifying defective healing [3].

The blink reflex provides complementary information by assessing the trigemino-facial brainstem reflex arc. Preservation or early return of the R1 component has historically been associated with a favorable outcome, whereas persistent absence is more frequently linked to poor recovery [4]. A recent neurology-based study further showed that absent R1 responses together with more severe ENoG abnormalities were associated with poor outcomes [7]. These findings are highly relevant when the topic is positioned primarily within neurology.

Otological disease severity modifies the neural injury. In retrospective series of chronic otitis media-related facial paralysis, cholesteatoma and bony destruction of the facial canal were common findings [5,6]. Shorter delays between onset of facial paralysis and definitive management were associated with better restoration of facial function in one large surgical series [6]. Such observations are consistent with a model in which prolonged compression, inflammation, and ischemia progressively increase axonal damage.

The immunological literature supports biological plausibility for adding inflammatory markers to the prognostic framework. IL-6, TNF- α and other cytokines are expressed in cholesteatoma tissue, and studies have reported associations with inflammatory activity and bone remodeling or destruction [8,10–12]. Serum IL-6 has also been shown to be increased in patients with chronic suppurative otitis media compared with healthy controls [9]. However, the available literature does not yet demonstrate that serum IL-6, TNF- α , or CRP independently predicts facial nerve recovery in CSOM-related neuropathy. Their most defensible current role is as complementary biomarkers that can be correlated prospectively with neurological severity and neurophysiological degeneration.

Domain	Key variables	Neurological interpretation
Clinical	House–Brackmann grade, Sunnybrook score, lagophthalmos, synkinesis, contracture	Defines the functional phenotype and quality of recovery.
Electrophysiology	CMAP amplitude, side-to-side ratio, distal latency, EMG denervation/reinnervation	Estimates axonal preservation, denervation severity, and regenerative activity.
Blink reflex	R1/R2 latency and presence	Provides information on trigemino-facial brainstem reflex-arc integrity.
Otological/radiological	Cholesteatoma, granulation, temporal-bone destruction, facial canal involvement	Characterizes the anatomical and etiological burden affecting the nerve.
Immunological	IL-6, TNF- α , CRP	Reflects inflammatory activity; independent prediction of neurological recovery remains unproven.

DISCUSSION

The major implication of the reviewed evidence is that CSOM-associated facial nerve neuropathy should not be reduced to an otological complication alone. The otological process initiates and sustains the lesion, but the outcome of interest is peripheral cranial nerve recovery. This distinction is important for scientific classification: the first profile is neurology, while otorhinolaryngology is the second profile providing etiological, surgical, and anatomical context.

A neurologically coherent prognostic model should prioritize variables that directly represent nerve function. House–Brackmann and Sunnybrook scores describe the clinical phenotype. ENoG and EMG quantify neural injury and regenerative dynamics. Blink-reflex testing extends the evaluation to the reflex arc. These measures can be followed

longitudinally and linked to residual weakness, synkinesis, and contracture—outcomes that are directly relevant to neurological recovery.

Otological variables remain indispensable because they explain the mechanism and intensity of the insult. Cholesteatoma, granulation tissue, facial canal dehiscence, and temporal-bone destruction can be considered modifiers of neural damage rather than substitutes for neurological assessment. Their prognostic role is strongest when interpreted together with electrophysiological evidence of axonal loss.

The immunological component has particular research value. IL-6 and TNF- α are biologically active in chronic middle-ear inflammation and cholesteatoma, but the evidence base is currently stronger for disease activity and tissue destruction than for facial nerve recovery itself [8–12]. Therefore, a future prospective study should test whether these markers correlate with House–Brackmann/Sunnybrook severity, CMAP reduction, denervation activity, and blink-reflex abnormalities. Demonstrating such links would provide a genuine neuroinflammatory dimension to the dissertation topic.

From a methodological standpoint, this approach avoids prematurely creating a new 'prognostic criterion' or score. It is scientifically more appropriate to determine the prognostic significance of existing clinical, electrophysiological, and immunological changes, identify independent associations, and only then consider whether a validated composite model is justified.

CONCLUSION

- Clinical-neurological and electrophysiological variables constitute the core of prognostic assessment in facial nerve neuropathy associated with chronic suppurative otitis media.
- House–Brackmann and Sunnybrook grading, ENoG/EMG, and blink-reflex testing are complementary and collectively provide a more complete description of facial nerve injury and recovery than otological findings alone.
- Cholesteatoma and destructive temporal-bone changes are important modifiers of intratemporal facial nerve injury and should be integrated into neurological interpretation.
- IL-6, TNF- α , and CRP are biologically plausible adjunctive markers, but their independent prognostic value for facial nerve functional recovery requires prospective validation.
- The topic is therefore appropriately positioned with Neurology as the first scientific profile and Otorhinolaryngology as the second.

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