



Congenital myasthenic syndromes: recent advances

David Beeson

Purpose of review

Congenital myasthenic syndromes (CMS) are a group of heterogeneous inherited disorders caused by mutations in genes encoding proteins essential for the integrity of neuromuscular transmission. This review updates the reader on recent findings that have expanded the phenotypic spectrum and suggested improved treatment strategies.

Recent findings

The use of next-generation sequencing is continuing to unearth new genes in which mutations can give rise to defective neuromuscular transmission. The defective transmission may be part of an overall more complex phenotype in which there may be muscle, central nervous system or other involvement. Notably, mutations in series of genes encoding presynaptic proteins are being identified. Further work on mutations found in the *AGRN*-*MUSK* acetylcholine receptor clustering pathway has helped characterize the role of *LRP4* and broadened the phenotypic spectrum for *AGRN* mutations. Mutations in another extracellular matrix protein, collagen 13A1 and in *GMPPB* have also been found to cause a CMS. Finally, there are an increasing number of reports for the beneficial effects of treatment with β 2-adrenergic receptor agonists.

Summary

Recent studies of the CMS illustrate the increasing complexity of the genetics, pathophysiological mechanisms and the need to tailor therapy for the genetic disorders of the neuromuscular junction.

Keywords

collagen 13A1, congenital myasthenic syndromes, N-glycosylation pathway, salbutamol, presynaptic

INTRODUCTION

Congenital myasthenic syndromes (CMS) are a group of heterogeneous inherited disorders caused by mutations in genes encoding proteins that are essential for maintaining the integrity of neuromuscular transmission [1,2]. The syndromes share the clinical feature of fatigable weakness, but age at onset, presenting symptoms, distribution of weakness and response to treatment vary depending on the molecular mechanism resulting from the genetic defect. To date, at least 24 different genes are known to cause CMS (Table 1; [3]). Next-generation sequencing (NGS) is helping to spread the net in identifying causative genes that now include proteins that are ubiquitously expressed but their defective function is most apparent at the neuromuscular junction, and presynaptic proteins in which multiple functional deficits are present, of which defective neuromuscular transmission is one.

In this review, we report the most recent findings in CMS regarding novel causative genes, notable observations for the better established CMS subtypes and an update on treatment. Readers are referred to other reviews for a more general overview of CMS [1,4].

PRESYNAPTIC SYNDROMES

Until recently, mutations in *CHAT*, the gene encoding choline acetyltransferase, were the only presynaptic cause of a CMS. The hallmark phenotypic feature for patients with *CHAT* mutations is episodic apnea that can be fatal. New reports support the original findings of Ohno *et al.* [5] that characterized the mutations as affecting the catalytic activity of the enzyme, the expression levels of the enzyme or a combination of the two [6]. This leads to a use-dependent reduction in the filling of acetylcholine (ACh) molecules into the synaptic vesicles of the motor nerve terminal and thus a reduction in the quantal release of ACh into the synaptic cleft.

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