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When Myosin Affects Hearing: A Case of MYH7B-Related Sensorineural Hearing Loss

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Abstract: Sensorineural hearing loss (SNHL) is a common congenital disorder with a significant genetic contribution. The widespread use of whole exome sequencing (WES) has aided in the identification of various genes associated with congenital hearing loss. MYH7B, a member of the myosin heavy chain family encoding myosin heavy chain, has recently been linked to hereditary SNHL. We describe a 10-year-old girl with bilateral SNHL and growth lag. WES reported a heterozygous missense variant in MYH7B. To date, only one family with 3 affected siblings with hearing loss linked to MYH7B mutations has been identified. This case report contributes to the limited literature available on MYH7B-related hearing loss.

Keywords: *Sensorineural Hearing Loss, MYH7B Gene, Growth Lag, Auditory Dysfunction.*

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I. INTRODUCTION

Sensorineural hearing loss (SNHL) is the most common congenital sensory disorder worldwide, affecting approximately 1-2 per 1000 live births (1). A large proportion of congenital SNHL has a genetic basis, with autosomal recessive inheritance accounting for nearly two-thirds of non-syndromic cases. Among implicated genes, GJB2 is the most frequently reported, followed by SLC26A4, MYO15A, and OTOF (2). The MYH7B gene, which encodes sarcomeric myosin heavy chain, essential for cytoskeletal organisation and intracellular transport, has recently been linked to auditory dysfunction (3).

II. CASE PRESENTATION

A 10-year-old girl presented with complaints of growth lag and impaired hearing. She was the firstborn child of a second-degree consanguineous couple. Antenatal history was unremarkable, with no exposure to infections, radiation or teratogenic drugs. She was delivered at term by an uneventful vaginal delivery with a birth weight of 2.3 kg. There was no history suggestive of birth asphyxia, neonatal sepsis, significant hyperbilirubinemia or neonatal intensive care admission.

Hearing difficulties, including delayed speech and language development, were recognised at two years of age. A detailed audiological evaluation using brainstem evoked response audiometry demonstrated bilateral severe sensorineural hearing loss. There was no significant prior history of recurrent otitis media, seizures or developmental regression. No family history of hearing loss, neuromuscular disease or cardiomyopathy was reported.

On physical examination, she weighed 13.7 kg and measured 114 cm in height. Both parameters were below the 3rd percentile for age, indicating proportionate growth lag. No facial dysmorphisms were observed. Both fine and gross motor milestones were age-appropriate. There was a significant history of constipation, managed with diet modification and oral laxatives. The rest of the systemic examination was within normal limits, with no evidence of cardiomyopathy. Given the early-onset SNHL and consanguineous background, whole exome sequencing was performed, and a heterozygous missense variant in the MYH7B gene, c.5000T>A pMet1667Lys (48x/109x), was identified. She was fitted with bilateral hearing aids and initiated on speech therapy. A multidisciplinary follow-up has been planned.

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III. DISCUSSION

Sensorineural hearing loss is characterized by marked genetic diversity, and whole exome sequencing has become an essential tool for diagnosing unexplained auditory impairment. Primarily known for muscle-related functions, MYH7B encodes a type II myosin crucial for actin-myosin interactions in the cochlear hair structure. Disruption of these interactions leads to impaired auditory function (3.4).

Haraksingh et al. reported autosomal recessive SNHL due to compound heterozygous MYH7B mutations, with all three affected siblings inheriting one variant from each unaffected parent (5).

The absence of myocardial or neuromuscular involvement in this child is consistent with previously reported MYH7B-associated phenotypes, which appear to be largely confined to auditory dysfunction (3). The observed growth lag and constipation are unlikely to be attributable to MYH7B-related disease and may represent incidental or multifactorial findings.

IV. CONCLUSION

This report contributes to the limited clinical evidence supporting a role for MYH7B in hereditary hearing loss. It also emphasizes the challenges inherent in interpreting variants in emerging disease genes. Reporting such cases enhances understanding of gene-disease associations and supports future variant interpretation. Ongoing multidisciplinary care and long-term follow-up remain crucial for the optimal management of affected children and their families.

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- Conflict of Interest: None

- Ethical considerations: Institute ethics committee approval deferred as per institute policy (Case report and patient details anonymised). Parental written consent for publication obtained.

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