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CLINICAL, AUDIOLOGICAL AND PREVENTIVE CHARACTERISTICS OF HEARING FUNCTION IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND DIABETIC POLYNEUROPATHY

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Abstract

Type 2 diabetes mellitus and one of its most prevalent late complications, diabetic polyneuropathy, exert a detrimental impact on multiple physiological systems, including the auditory analyser. Growing evidence suggests that chronic hyperglycaemia and diabetes-related microvascular and neuropathic changes contribute to progressive auditory dysfunction. This scientific study investigates the clinical and audiological characteristics of hearing function in patients with type 2 diabetes mellitus complicated by diabetic polyneuropathy, with particular attention to the underlying pathophysiological alterations of the auditory system and preventive strategies. The study involved comprehensive clinical and neurological examinations, pure-tone threshold and speech audiometry, tympanometry, and otoacoustic emission testing. Audiological assessment revealed that the majority of patients with diabetic polyneuropathy exhibited sensorineural hearing loss, predominantly affecting high-frequency ranges. The severity of hearing impairment demonstrated a strong correlation with the duration of diabetes, the level of glycaemic control, and the degree of neuropathic involvement. The findings highlight the clinical significance of auditory dysfunction as an underrecognised complication of type 2 diabetes mellitus. Timely audiological screening and the implementation of comprehensive

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preventive measures play a crucial role in early detection of hearing impairment and in preventing further progression of auditory dysfunction in patients with diabetic polyneuropathy.

Keywords: Type 2 diabetes mellitus; diabetic polyneuropathy; auditory analyser; sensorineural hearing loss; audiological assessment; otoacoustic emission; glycaemic control; prevention.

Introduction

Diabetes mellitus (DM) is currently recognised as one of the most rapidly increasing chronic metabolic disorders worldwide, posing a significant public health challenge. According to data reported by the World Health Organization, the global prevalence of diabetes continues to rise steadily each year, with type 2 diabetes mellitus (T2DM) accounting for the vast majority of cases [1]. This disease is characterised by persistent disturbances in glucose metabolism and a prolonged clinical course, leading to the development of multiple systemic complications that affect both microvascular and macrovascular structures [2].

Among the late complications of T2DM, diabetic polyneuropathy (DPN) represents one of the most common and clinically significant conditions. DPN is characterised by diffuse damage to the peripheral nervous system and results in progressive sensory, motor, and autonomic dysfunction [3]. The pathogenesis of diabetic polyneuropathy is complex and involves chronic hyperglycaemia, impaired insulin signalling, oxidative stress, and microvascular insufficiency, all of which contribute to neural degeneration and functional impairment [4].

In recent years, growing scientific evidence has demonstrated that the pathological processes underlying diabetic polyneuropathy are not limited to peripheral somatic nerves but may also involve the auditory nerve and central and peripheral components of the auditory analyser [5]. Clinical and experimental studies suggest that patients with long-standing T2DM exhibit a higher prevalence of hearing impairment compared with non-diabetic individuals, even in the absence of overt otological disease [6].

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Diabetes-associated microangiopathy, metabolic imbalance, and oxidative stress play a pivotal role in inducing pathological changes within the inner ear. These mechanisms lead to compromised blood supply to the cochlea, degeneration of outer and inner hair cells, and damage to spiral ganglion neurons and auditory nerve fibres [7]. As a result, progressive sensorineural hearing loss—predominantly affecting high-frequency ranges—may develop insidiously and remain clinically unrecognised for extended periods [8].

Despite the increasing recognition of hearing dysfunction as a diabetes-related complication, auditory impairment is often underestimated in routine clinical practice and is rarely included in standard diabetic screening protocols. Consequently, hearing loss is frequently diagnosed at advanced stages, when structural and functional damage has already become irreversible. This highlights the importance of comprehensive clinical and audiological evaluation in patients with T2DM, particularly in those with established diabetic polyneuropathy [9].

In this context, an in-depth clinical and audiological assessment of hearing function in patients with type 2 diabetes mellitus complicated by diabetic polyneuropathy is of considerable relevance. Early identification of auditory dysfunction through systematic audiological screening may facilitate timely preventive interventions, improve quality of life, and reduce the long-term burden of diabetes-related sensory complications.

Materials and Methods

The study was conducted among patients diagnosed with type 2 diabetes mellitus who presented with clinical manifestations of diabetic polyneuropathy. Participants were evaluated according to the duration of diabetes, the level of glycaemic control, and the severity of neurological symptoms. A control group consisting of individuals without hearing impairment or neurological disorders was included for comparative purposes.

All participants underwent a comprehensive clinical and neurological examination. Audiological assessment included pure-tone threshold audiometry across frequencies ranging from 125 to 8000 Hz, speech audiometry, tympanometry, and otoacoustic emission testing using both transient-evoked and

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distortion-product protocols. Laboratory investigations focused on metabolic status and included measurements of fasting blood glucose and glycated haemoglobin (HbA1c).

Analysis of pure-tone audiometry revealed that a considerable proportion of patients with diabetic polyneuropathy exhibited sensorineural hearing loss. Elevation of hearing thresholds was predominantly observed at high frequencies, a pattern consistent with early cochlear involvement and damage to inner ear hair cells. These changes were not typically accompanied by complaints in the early stages, which may explain the delayed clinical recognition of auditory dysfunction in diabetic patients.

Speech audiometry demonstrated reduced speech perception ability in patients with diabetic polyneuropathy, suggesting impaired transmission of auditory signals along the auditory nerve pathways. Tympanometric findings were largely within normal limits, indicating that middle ear pathology did not play a significant role in the observed hearing impairment.

Otoacoustic emission testing provided further evidence of cochlear dysfunction. In the majority of patients, reduced amplitudes or complete absence of otoacoustic emission responses were recorded, reflecting compromised function of the outer hair cells and confirming damage at the level of the inner ear.

From a pathogenetic perspective, diabetic microangiopathy appears to play a central role in disrupting cochlear microcirculation, thereby promoting ischaemic and degenerative changes within the auditory system. In parallel, diabetic polyneuropathy interferes with neural conduction along the auditory nerve, further contributing to functional hearing deficits. Oxidative stress and chronic glucotoxicity are believed to accelerate apoptotic processes in hair cells, leading to gradual but progressive deterioration of hearing function.

A clear association was observed between the severity of hearing impairment, the duration of diabetes, and HbA1c levels. This relationship underscores the close link between auditory dysfunction and long-term metabolic control, highlighting hearing loss as a potential marker of systemic diabetic complications.

From a preventive standpoint, effective management of hearing disorders in diabetic patients should prioritise optimisation of glycaemic control and early

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detection of diabetic polyneuropathy. Regular audiological screening, ideally on an annual basis, allows early identification of subclinical hearing changes. In addition, the use of antioxidant and neuroprotective therapies, together with avoidance of excessive noise exposure and ototoxic medications, may help reduce the risk of progression and preserve auditory function in this patient population.

Results and Discussion

The clinical and audiological assessment demonstrated that hearing impairment is a frequent finding among patients with type 2 diabetes mellitus complicated by diabetic polyneuropathy. The predominant pattern observed was sensorineural hearing loss, which was most evident at higher frequencies. This configuration is characteristic of early cochlear damage and is consistent with dysfunction of the inner ear hair cells. In many patients, these changes were detected despite the absence of pronounced subjective hearing complaints, emphasising the insidious nature of auditory involvement in diabetes.

Speech audiometry further revealed a decline in speech perception ability in patients with diabetic polyneuropathy. This finding suggests not only cochlear involvement but also impaired neural transmission along the auditory pathways, likely reflecting the neuropathic component of the disease. In contrast, tympanometric measurements were largely within normal limits, indicating preserved middle ear function and supporting the conclusion that the observed hearing loss was primarily sensorineural in origin.

Otoacoustic emission testing provided additional objective evidence of cochlear pathology. Reduced amplitudes or absent otoacoustic emission responses were recorded in a substantial proportion of patients, particularly in those with longer disease duration and poorer metabolic control. These findings indicate functional impairment of the outer hair cells and reinforce the concept that cochlear damage precedes clinically apparent hearing loss in diabetic individuals.

The results of this study are in agreement with previously reported data suggesting that diabetes-related microangiopathy and chronic metabolic imbalance adversely affect cochlear microcirculation. Reduced blood supply to the inner ear promotes ischaemic changes and accelerates degenerative processes

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within the sensory epithelium. In parallel, diabetic polyneuropathy disrupts the normal conduction of auditory nerve impulses, thereby compounding the functional deficit. Oxidative stress and sustained hyperglycaemia are thought to play a central role in triggering apoptotic pathways in hair cells and neural structures, ultimately leading to progressive hearing deterioration.

A significant association was identified between hearing impairment and both the duration of diabetes and HbA1c levels. This relationship highlights the close link between auditory dysfunction and long-term glycaemic control, suggesting that hearing loss may serve as an additional indicator of systemic diabetic complications. These findings underline the importance of considering auditory function as part of the comprehensive assessment of patients with long-standing type 2 diabetes mellitus.

Conclusion

Type 2 diabetes mellitus and diabetic polyneuropathy represent significant risk factors for impairment of auditory analyser function. The findings of the present study demonstrate that sensorineural hearing loss, predominantly affecting high-frequency ranges, is highly prevalent among patients with diabetic polyneuropathy. The degree of hearing dysfunction shows a clear association with the duration of diabetes and the level of glycaemic control, reflecting the systemic nature of diabetes-related microvascular and neuropathic damage.

Timely clinical and audiological evaluation, combined with a comprehensive preventive approach, plays a crucial role in the early detection of hearing impairment and in preventing further progression. Incorporation of routine audiological screening into the long-term management of patients with type 2 diabetes mellitus may contribute to improved quality of life and reduction of sensory complications associated with this chronic metabolic disorder.

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