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CLINICAL FEATURES OF OTORHINOLARYNGOLOGICAL PATHOLOGY IN CHRONIC KIDNEY

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Abstract
This scientific article presents a review of current knowledge on chronic kidney pathology, in particular chronic kidney failure, in both the pre- and post-transplantation periods, as well as pathological changes affecting the organs of the ear, nose, and throat.

Keywords: Chronic kidney disease, kidney transplant recipient, otorhinolaryngological complications, sensorineural hearing loss, otoacoustic emissions, short-latency auditory evoked potentials.

Introduction

As the global prevalence of chronic kidney disease (CKD) continues to increase, the number of patients with systemic dysfunctions associated with CKD, including otorhinolaryngological disorders, is also expected to rise [1]. The effects of CKD on body systems result from the accumulation of nitrogenous waste products, so-called “uremic toxins,” in various tissues, electrolyte imbalance, local chemical reactions due to ammonia, as well as immunological, vascular, and coagulation changes [3]. Ototoxic and immunosuppressive agents used in the treatment of CKD also contribute to a range of systemic complications [3]. It has been established that immunosuppression in patients after kidney transplantation predisposes them to various infections, particularly opportunistic infections, and to the development of malignancies [4,5]. CKD affects the vast majority of organ systems; however, this review focuses primarily on otorhinolaryngological complications of CKD in both the pre- and post-transplantation periods.

Certain otorhinolaryngological dysfunctions associated with CKD have been studied in greater detail than others. The most frequently analyzed head and neck abnormalities in patients with CKD, including kidney transplant recipients (KTRs), include sensorineural hearing loss, epistaxis, candidiasis, halitosis, xerostomia, dysgeusia, lip cancer, and thyroid gland cancer in kidney transplant recipients. In addition, this review also reports correlations between CKD and other conditions, including rhinosinusitis, rhinocerebral

mucormycosis, sudden sensorineural hearing loss, deep neck infections, mucosal ulcerations, lichenoid changes, oral hairy leukoplakia, tinnitus, vertigo, olfactory loss, tympanosclerosis, voice disorders, gingival hyperplasia, and head and neck cancer.

Aim of the study:

To provide an overview of current knowledge on CKD and the effects of its treatment on ENT organs.

Epistaxis is a common symptom caused by CKD [3]. The nasal cavity is one of the most frequent sites of bleeding in patients with uremia. In patients with CKD, epistaxis is mainly caused by the accumulation of toxic elements that are normally excreted by the kidneys in healthy individuals. Other factors predisposing CKD patients to epistaxis include anemia and coagulation disorders [5]. Other sinonasal manifestations, namely chronic and acute rhinosinusitis, invasive fungal rhinosinusitis, or fungal ball, are mainly observed in organ transplant recipients as a result of immunosuppression [10]. Maxillary sinus mucosal cyst (MSMC) is a benign and usually asymptomatic condition caused by obstruction of the duct of a seromucous gland of the sinus mucosa, leading to mucus accumulation and cystic dilatation of the affected gland [8]. Studies have shown that olfactory function may be impaired in patients with CKD. It has been reported that patients with CKD, particularly those with end-stage renal disease undergoing dialysis, exhibit a moderate but significant reduction in olfactory function [7,8].

Oropharyngeal lesions are very common in patients with CKD. CKD-associated systemic inflammation leads to malnutrition, predisposes patients to cardiovascular disease, and increases mortality in this population. Most oropharyngeal abnormalities in this group result from elevated salivary urea levels [10]. Patients with CKD have been found to exhibit various oropharyngeal disorders, including halitosis, xerostomia, periodontitis, dysgeusia, candidiasis, parotitis, abnormal lip pigmentation, burning mouth sensation, and ulcerations.

Sore throat is a frequent complaint among patients with CKD. It is generally caused by oropharyngeal dryness and ulcerations resulting from reduced salivary secretion, dehydration, and the action of commensal bacteria that degrade urea. According to a meta-analysis conducted by Ruospo et al., mucosal ulcers were observed in 8.6% of dialysis-dependent end-stage renal disease patients (n = 832) and in 1.3% of kidney transplant recipients (n = 453). Patients with CKD frequently suffer from oropharyngeal candidiasis. It has been reported that candidiasis developed in 37% of individuals with CKD [14].

Voice changes are frequently observed in patients with chronic kidney failure and are mainly attributed to the effects of CKF on the respiratory and phonatory systems [15]. Patients with CKF commonly demonstrate vocal fold edema, decreased pulmonary function, or abnormal coordination between the central nervous system and peripheral phonatory structures, all of which subsequently lead to voice changes. It has been found that patients with end-stage renal disease undergoing hemodialysis may experience

transient post-dialysis hoarseness as a result of dehydration caused by hemodialysis, reduction in vocal fold size, and increased subglottic pressure. Rare cases of laryngospasm caused by hypocalcemia secondary to CKD have also been reported, resulting from increased reflex excitability of the recurrent laryngeal nerves at neuromuscular junctions.

Sensorineural hearing loss (SNHL) is a common otorhinolaryngological manifestation in patients with CKD. CKD is considered an important independent risk factor for SNHL. SNHL in CKD patients is usually bilateral and occurs more frequently than in the general population [6,7]. The prevalence of SNHL among patients with CKD ranges from 28% to 77%. It is mainly diagnosed in patients with long-standing CKD and worsens over time [7,8]. The highest prevalence of SNHL has been reported in individuals with an estimated glomerular filtration rate above 45 mL/min/1.73 m^2.

The high prevalence of SNHL in patients with CKD may result from several structural and functional similarities between the kidneys and the inner ear [8]. The most important similarity is active electrolyte and fluid transport occurring in the glomerular basement membrane and the stria vascularis of the cochlea [8]. This is due to the presence of the Na^+/K^+-ATPase pump and the enzyme carbonic anhydrase [8]. In addition, the cochlea and kidneys have been found to share similar antigenicity [8]. This is supported by certain diseases and syndromes (e.g., Alport syndrome) that affect both the inner ear and the kidneys.

The most widely discussed ototoxic drugs used in the treatment of CKF are aminoglycosides and furosemide [6]. Vitamin D deficiency and reduced Na^+/K^+-activated ATPase activity are also associated with SNHL [8]. It has been suggested that inhibition of Na^+/K^+-activated ATPase, which is crucial for maintaining the proper ionic gradient in the inner ear, may be the main cause of sensorineural auditory dysfunction in patients with uremia [8]. Another dysfunction predisposing CKF patients to SNHL is endolymphatic hydrops [8]. Endolymphatic hydrops has previously been associated with low-frequency SNHL and may explain hearing improvement after hemodialysis [8]. Uremia-induced nervous system dysfunctions, referred to as “uremic neuropathy,” may also lead to changes in the auditory nerve and auditory pathways [8]. This observation has been confirmed by brainstem auditory evoked response (BAER) testing in patients with CKD by various authors [7]. It has been noted that cases of SNHL in patients with CKD were more often due to cochlear dysfunction rather than retrocochlear auditory pathology [8]. The formation of amyloid deposits in the cochlea caused by prolonged hemodialysis may also result in hearing impairment [8]. Finally, hearing loss may result from the toxic effects of aluminum on the inner ear in these patients [8]. Moreover, the duration of hemodialysis has been reported to be the only independent predictor of sensorineural hearing loss [6].

SNHL should be confirmed by audiological testing. The most common audiometric abnormality observed in patients with CKF was high-frequency hearing loss with a notch at 6 kHz [8]. Speech discrimination appeared to be unaffected in these patients [8].

Distortion-product otoacoustic emissions (OAE) are evoked responses generated by simultaneous stimulation of the cochlea with two pure tones [10]. OAE testing is sensitive for detecting cochlear dysfunction, even at a subclinical level [10]. OAEs were absent in a significant proportion of patients with CKD in various studies [10]. Brainstem auditory evoked response is an objective non-invasive electrophysiological test that measures the retrocochlear portion of the auditory pathway up to the brainstem level in response to sound stimuli [11].

Several authors have reported delayed neural auditory conduction in patients with CKF, manifested by prolonged BAER wave latencies [11]. It was concluded that conduction times in BAER testing improved after a hemodialysis session; however, hearing did not return to normal [11]. Differences in BAER responses before and after hemodialysis may be attributable to varying calcium ion (Ca^{2+}) levels [11]. In this regard, it has been suggested that hearing loss may be inversely correlated with the number of hemodialysis sessions [6]. Interestingly, studies have shown that patients with CKF were 1.57 times more likely to develop sudden sensorineural hearing loss than the general population [12]. The risk was even higher in patients with CKF and concomitant diabetes mellitus [12]. Although potential etiological factors for SSNHL have been proposed, its etiology in this population remains unclear [12]. Kan et al. reported that patients with CKF and concomitant SSNHL demonstrated a poorer recovery prognosis compared with individuals without CKF when treated with systemic glucocorticosteroids, which are the first-line treatment for SSNHL [12]. In contrast, promising results in the treatment of SSNHL in patients with CKF were achieved using intratympanic steroid injections.

Abnormal kidney function, defined by impaired GFR in patients with CKD, leads to serum phosphate accumulation [13]. Serum phosphate can bind free calcium-producing precipitates and cause subsequent calcification [13]. A reduction in free serum calcium stimulates the parathyroid glands to produce parathyroid hormone (PTH) [13]. Calcification in patients with CKD has been observed mainly in arteries and internal organs [13]. Calcification may also occur in the lamina propria of the middle ear mucosa; however, studies on this topic are limited [13]. Matrix vesicles have been identified as important elements in abnormal tissue calcification [14]. They are also involved in other types of calcifications, including tympanosclerosis [14]. It has been suggested that patients with CKD are more prone to developing myringosclerosis, a type of tympanosclerosis affecting the tympanic membrane [14]. However, no correlation was observed between serum phosphorus, calcium, magnesium, or PTH levels and the occurrence of myringosclerosis [13,14]. According to El-Anwar et al., an increased risk of myringosclerosis was found in patients with CKD undergoing hemodialysis for more than three years, whereas the study by Kaldas et al. did not reveal a similar association between hemodialysis duration and myringosclerosis formation [13,14]. The exact correlation between CKD and the development of both tympanosclerosis and myringosclerosis requires further investigation.

Electrolyte and osmotic changes associated with CKD that affect the cochlea may also influence the labyrinth [14]. Patients with CKD have an increased risk of developing vestibular dysfunction compared with the healthy population [15]. Although the exact cause of vertigo in patients with CKD remains unclear, proposed potential etiological factors include retention of toxic products with subsequent vasculopathy, vestibulocochlear neuropathy, and vascular calcification within the labyrinth.

Conclusion

Otorhinolaryngological disorders are not uncommon complications of CKD and its treatment. Patients with CKD are prone to developing predominantly recurrent epistaxis, opportunistic infections including oropharyngeal candidiasis or rhinocerebral mucormycosis, taste and smell disturbances, deep neck infections, voice disorders, mucosal abnormalities, gingival hyperplasia, sensorineural hearing loss, tinnitus, and vestibular dysfunction. Individuals receiving immunosuppressive therapy after kidney transplantation have an increased risk of carcinogenesis, both related and unrelated to latent viral infection.

Otorhinolaryngological disorders associated with CKD are often persistent, difficult to control, significantly impair quality of life, and may be life-threatening. Given the high prevalence of otorhinolaryngological complications caused both by CKD itself and its treatment, we conclude that patients with CKD, including those after organ transplantation, require frequent and long-term follow-up by an experienced otorhinolaryngologist. This is particularly important because some of these complications may be reversible with early detection and treatment. In addition, progression of certain disorders can be slowed with appropriate therapy, and some may improve after treatment correction. While the predisposition to several otorhinolaryngological complications in patients with CKD and their association with CKD have been well explained, the correlation between other complications and CKD remains unclear. This is mainly due to the lack of large cohort studies and the inconsistency of existing results. Therefore, further research on this topic is required.

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