Dear Editor

I read with great interest the article of systematic review and meta-analysis of Bayat et al., which shows a close relationship between loss of hearing and COPD. The article reminds us that the doctor should always make an accurate medical history, because the clinical evaluation is the foundation of what will be the therapy of the patient.

The text does not give explanations on the possible causes that induce hearing loss in patients diagnosed with COPD.

There is a close embryological and functional relationship between the temporomandibular joint (TMJ) and the middle ear. The structure of union between these two anatomical areas is the discomalleolar ligament or Pinto’s ligament, which connects the medial retrodiscal portion of TMJ and the malleus of the middle ear. Studies show that an altered ligament tension due to an abnormal opening of the mouth leads to dysfunction of the middle ear with risk of hearing loss.

The patient with COPD suffers from many comorbidities, including musculoskeletal disorders, arthritis, arthrosis, with degeneration of the joint functions (as for TMJ), as well as obstructive sleep apnea syndrome (OSAS). OSAS causes an abnormal opening of the mouth during the night, with a structural and functional alteration of the neck; there is a close relationship between this syndrome and the decrease in hearing.

From a clinical point of view, signs such as hearing loss, functional impairment of the neck and TMJ disorders lead to Costen’s syndrome.

The latter alters the internal pressure of the middle ear and subsequent decrease in hearing, through muscular (neck), articular (TMJ) and ligament dysfunction (Pinto’s ligament).

We can strongly hypothesize that one of the most important causes of hearing loss in patients with COPD is the secondary presence of Costen’s syndrome.

Disclosure

The author reports no conflicts of interest in this communication.

References


Dear editor

We wish to emphasize some issues concerns our study. Actually, among the well-studied reasons that lead to hearing loss in patients with COPD, we discussed those that have been observed in most studies. The reason which is conflicting is the impact of smoking status or the type of smoking on the degree of impairment in auditory measures, which some studies reported a significant correlation, whereas other evidence demonstrated no association. The first possible reason that received special attention is the effect of COPD and chronic hypoxemia or hypoxia on the auditory function. Moreover, numerous studies have documented another possible reason that locally accompanied by loss of hearing sensitivity due to the high dependence of the transduction mechanism of the inner ear to the change in cochlear oxygen supply such as hypoxia.

Another research suggested poorer central auditory function during hypoxemia in patients with COPD. Besides, some researchers introduced infection as a symptom that needs special consideration in patients with COPD; so, this leads researchers to use antibiotics in COPD patients as a prophylactic treatment to reduce exacerbations as well as to improve quality of life.11,12

Besides, infection is one of the different factors that can cause a type of hearing loss called conductive hearing loss. Although it is interesting to hypothetically consider secondary change in internal pressure of the middle ear and subsequent hearing impairment through temporomandibular joint (TMJ) as a cause of hearing loss in patient with COPD, this will require more research to be done. So far, up to 90% of the individuals with tinnitus suffer from some level of noise-induced hearing loss.14 A group of evidence reported that tinnitus associated with a TMJ falls under this second category.15,16

Another important suggestion is that functional impairment of the neck and TMJ, and consequently Costen’s syndrome, can lead to dysfunctional breathing such as mouth breathing syndrome, respiratory mechanics changes, diaphragmatic muscular dystonia and overuse of accessory inspiratory muscles.17 Consequently, such breathing dysfunction may induce hypoxia through various mechanisms, but not necessarily COPD.

Disclosure

The authors report no conflicts of interest in this communication.

References