

Review Article

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The Ear-Temporomandibular Joint Complex: A Narrative Review of Anatomical and Pathological Connections



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Abstract

Background: The ear and temporomandibular joint (TMJ) share common embryological origins. Disorders of the ear and TMJ frequently share the same aetiological origins.

Materials and methods: A PubMed literature search between 1970 and 2023 was performed to reveal anatomical and pathological connections between the ear and TMJ.

Results: Various anatomical communications exist between the ear and TMJ. Pathological processes that can affect both structures include infectious, inflammatory and neoplastic processes.

Conclusion: Otologists and dentists care for diseases in the ear and TMJ should recognize the possible anatomical and pathological connections between both areas.

Keywords: External ear; Middle ear; Temporomandibular joint; Temporomandibular disorder; Otoneurologic symptoms

Abbreviations: TMJ: Temporomandibular Joint; TMD: Temporomandibular Disorder; JVA: Joint Vibration Analysis; RA: Rheumatoid Arthritis

Introduction

The temporomandibular joint (TMJ) lies directly Infront of the ear. Tissues of both structures have common sensory innervation from the auriculotemporal nerve [1]. They both receive arterial blood supply from the superficial temporal and maxillary arteries [2]. Several structures pass between both compartments [3]. Many pathological disorders, such as infectious and inflammatory processes, can traverse both areas [4]. Recently, much interest has surged in the reporting of otoneurologic symptoms in relation to dysfunction of the TMJ and related structures (Costen's syndrome) [5]. Also, surgery to the middle ear can have deleterious effect on TMJ structures [6]. Recent literature recommends that otologists should be familiar with temporomandibular disorder (TMD), as it is a common presentation to their practice [7]. On the other hand, the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) did not include otoneurological symptoms, despite including headache in the classification, as a subjective symptom.⁸ Hearing loss, blocked-ear sensation, subjective tinnitus, and vertigo are quite common symptoms in TMD patients (Costen's

syndrome) [9]. This is true for both arthrogenous and myogenous subtypes of TMD [10]. Objective audiometric and vestibular function tests support the otological origin of these symptoms in patients with TMD [9,11,12]. Additionally, ear pain is one of the most common presentations of TMD [13]. The TMJ and middle ear share common embryologic origin from the first branchial arch, emphasizing the notion that the TMJ and ear might be considered as a unit [14]. From these perspectives, the author reviews the anatomical and pathological connections between both regions. This would serve as a road-map for both otologists and dentists caring for pathology in this area. As far as the author is aware, and after a PubMed literature search from 1970 till 2023, no study had comprehensively discussed this connection between the ear and the TMJ.

Embryological considerations

The development of the human TMJ and ear structures follows a synchronous process that progresses through various stages.

These developmental stages in the human embryo and fetus putatively mirror the evolutionary scenario in the various living animals [15]. The human TMJ has evolved to be a robust, load-bearing joint [16]. Initially, the mandible and middle ear structures have a common ancestor, Meckel's cartilage, which is the cartilage of the first branchial arch [17]. Having three middle ear ossicles (malleus, incus and stapes) is a unique mammalian feature [18]. The posterior portion of Meckel's cartilage transforms into the malleus and incus [19]. The incudo-malleolar joint is considered analogous to the primary jaw joint that persists in fish [20]. Oral movements in the human fetus are essential for development of the secondary condylar cartilage that becomes responsible for synovial fluid production and formation of the disc to evolve into the two-compartment TMJ [21]. During the process of TMJ development, the masticatory and hearing organs separate by the development of the temporal bone [22]. Part of the sheath of the lateral pterygoid muscle persists in adults as the discomalleolarligament, while the proximal remnants of Meckel's cartilage persist in adults as the anterior malleolar and sphenomandibular ligaments (collectively termed the malleomandibular ligament) [23]. In hereditary disorders involving the TMJ, the external and middle ear are almost always severely affected. This is especially so in Treacher Collins syndrome and its variants [24]. A further embryological perspective is found in defects of development of the temporal bone, leading to the persistence of foramen of Huschke, which is associated with herniation of TMJ tissues into the external and middle ear [25].

Anatomical considerations

The TMJ is a synovial articulation between the condyle of the mandible inferiorly, and the glenoid fossa of the squamous temporal bone and articular eminence from the zygomatic bone; superiorly [26]. Posteriorly, the glenoid fossa presents the postglenoid process, which contributes to forming the upper wall of the external auditory meatus [27]. This represents the squamotympanic fissure, which continues medially as the petrotympanic fissure that forms the anterosuperior border of the petrous temporal bone and separates the TMJ from the middle ear [28,29]. Continuation between both compartments is provided by canaliculi in the petrotympanic fissure, which transmits the chorda tympani nerve, the discomalleolar and anterior malleolar ligaments, and the anterior tympanic artery [30]. It has been suggested that the extratemporal portion of the discomalleolar ligament would limit extreme anterior disc movement during wide mouth opening [31]. On the other hand, anterior disc displacement could cause increased tension on the discomalleolar and anterior malleolar ligaments, leading to a subtle movement of the malleus (in the order of microns), which would cause increased stiffness of the middle ear [11]. This movement of the malleus would be transmitted to the stapes, leading to altered polarization of inner ear hair cells, providing a plausible otologic cause for Costen's syndrome symptoms in anterior disc displacement [32].

Immediately medial to the glenoid fossa is the bony Eustachian

tube [33]. Recent advances in otological endoscopy concluded that the bony Eustachian tube is indeed the protympanum [34]. Several links exist between the Eustachian tube and masticatory apparatus. Movement of the TMJ, such as during yawning and chewing helps to briefly open the Eustachian tube, which is necessary for its ventilatory function for the middle ear cleft [4]. Immediately above the bony Eustachian tube is the tensor tympani muscle, which via its connection to the malleus, causes tension on the tympanic membrane, via a reflex mechanism to loud sound [35]. On the other hand, baroreceptors in the middle ear and tympanic membrane can, via reflex mechanisms, delicately affect the ventilation of the middle ear, via actions on the Eustachian tube musculature [36]. In this regard, the tensor tympani muscle has been considered a (strange chewing muscle) [37]. Tension on tensor tympani, in conjunction with tension on masticatory muscles, have been hypothesized to play an essential role in the pathogenesis of otologic symptoms in patients with TMD [38,39]. Increased tone in these muscles, supplied by the mandibular nerve, appears to be centrally mediated, as revealed by brain imaging studies [40,41].

During mouth opening and closure, the condyle of the mandible exhibits both rotation and gliding movements [42]. In a normal TMJ, movement of the condyle-disc complex is a smooth, silent process [43]. This is due to the excellent lubrication system inside the TMJ [44]. However, disc abnormalities may lead to abnormal sounds in the joint which are transmitted to the inner ear. The sounds include clicking in reducing disc displacement or crepitus in degenerative joint disease [45,46]. Recently, joint vibration analysis (JVA) has been applied as a useful, simple tool to screen for sounds in the TMJ, as well as to categorize the sounds as click or crepitus, based on the vibration frequency of the sound in the disordered joint. Therefore, this tool bridges the gap between clinical findings and sophisticated TMJ imaging [47]. Elicited sounds from the disordered TMJ reach the neighboring cochlea by the recently described soft tissue conduction; a form of sound conduction different from air conduction and bone conduction [48]. Soft tissue conduction from the TMJ to the cochlea takes place along the vascularized retro discal tissue as well as the Eustachian tube [49,50]. Apart from the associated clinical complaints, the abnormal sounds have been described as a (noisy annoyance) [51].

Pathological considerations

The TMJ and ear are anatomically very close to each other. Therefore, pathology in one region can readily spread to the other region. Notable pathologies include sequelae of traumatic, infectious, inflammatory, and neoplastic processes. Traumatic events may be macrotrauma to the jaw region or microtrauma to the TMJ (bruxism) [52]. Motor vehicle accidents, assaults, sports injuries, and falls are the most common causes of TMJ and ear injuries, that are associated with fractures [53]. Fractures of the TMJ are frequently complicated by external ear canal fractures, soft tissue injuries, and tympanic membrane perforations [54].

More seriously, are associated petrous temporal bone fractures, which may be complicated by facial nerve paralysis, sensorineural hearing loss, cerebrospinal fluid leak, and/or traumatic brain injury [55]. Apart from fracture, traumatic posterior or superior TMJ dislocations may damage the external ear canal [56].

Bruxism is a diurnal or nocturnal parafunctional activity, associated with clenching or grinding of the teeth. Awake bruxism occurs in response to stress or anxiety, while sleep bruxism is associated with micro-arousals and changes in the autonomic nervous system [57]. Bruxism has been postulated to cause TMJ microtrauma and/or masticatory muscle hyperactivity [58]. Regarding the TMJ, mechanical studies have revealed that bruxism causes abnormal loads on the TMJ disc, contributing to its deterioration [59]. TMJ microtrauma is associated with a low-grade inflammation in the joint. Various inflammatory molecules sensitize pain receptors, with the TMJ pain being referred to the ear via the auriculotemporal nerve [60]. Additionally, masticatory muscle hyperactivity may involve contraction of the tensor tympani muscle (causing stiffness of the middle ear system), and contraction of the medial pterygoid muscle (causing Eustachian tube dysfunction) [11,50]. These factors are putatively associated with sensation of ear fullness, hearing loss, tinnitus, and vertigo (Costen's syndrome) [61].

Otogenic septic arthritis of the TMJ is due to invasion of the joint space by microbes from a severe infection of the external or middle ear [62]. Malignant (necrotising) otitis externa is a severe infection of the external ear, that progresses to cause osteomyelitis of the temporal bone [63]. TMJ involvement, with osteomyelitis of the mandibular condyle, has been previously described in uncontrolled cases with malignant otitis externa [64]. Septic arthritis of the TMJ may also be a complication of acute otitis media, especially in young children with a partially developed temporal bone [65]. Early radiological features show joint effusion, which may later progress to abscess formation, then TMJ ankylosis [66]. Apart from acute infectious disorders, a recent study had confirmed a higher incidence of internal derangement of the TMJ in patients with chronic suppurative otitis media, compared to controls [67]. It was postulated by the author that the inflammatory process in the middle ear could spread to the TMJ through the canaliculi in the petrotympanic fissure. This would presumably alter the lubrication system in the TMJ, leading to disc displacement [67]. Furthermore, harvesting of the temporalis fascia, a common procedure in ear surgery, can alter the biomechanics of the TMJ, leading to internal derangement [6].

Osteoarthritis of the TMJ, associated with degenerative joint disease, is classified as a low-grade inflammatory condition of the joint [68]. On the other hand, rheumatoid arthritis (RA) of the TMJ is considered a high-grade inflammatory joint disorder [69]. RA affects 1 per cent of the population and it is considered as the prototype of joint inflammatory disorders [70]. Pathologically, the characteristic synovial pannus destroys the TMJ structures,

and leads to long-term sequelae [71]. The incidence of clinical involvement of the TMJ in adult RA patients has been reported to vary from 1% to 20%, depending on the stage of the disease [72]. A recent study on patients with RA of the TMJ revealed an incidence of otalgia of 54.6 per cent; subjective hearing loss of 51.8 per cent; subjective tinnitus in 48.9 per cent; and vertigo in 64.5 per cent of patients. The incidence of these otologic symptoms was much higher in patients with RA of the TMJ than in control subjects ($P=0.001$) [73]. The same authors, in another study, reported that TMD patients presented to an otology clinic with Costen's syndrome symptoms, whereas they presented to the rheumatology clinic with degenerative joint disease and its sequelae [74]. Notably, the author reported that the incidence of otologic symptoms was higher in RA of the TMJ than in patients with TMD. In a review of literature they concluded that, in addition to the altered input from the inner ear to the brain stem, maladaptive plasticity in central nervous system pathways (neuroinflammation), plays an essential role in the maintenance of the otologic symptoms in patients with RA of the TMJ [75-78].

The sensory nerve supply to the TMJ and external ear comes from the auriculotemporal nerve. Additionally, the auriculotemporal nerve carries post ganglionic parasympathetic nerve fibers to the parotid gland [79]. Nociceptive nerve fibers from the ear and TMJ converge onto the subnucleus caudalis in the brain stem [80]. Referred otalgia is a common symptom of painful TMD [81]. Masticatory muscle entrapment of the auriculotemporal nerve is hypothesized to be an important cause of ear pain, temple headaches, and migraine [82,83]. Auriculotemporal neuralgia; either primary or secondary to trauma and surgery, causes severe, shock-like pain around the ear, condyle of the mandible and temporal region [84,85]. Frey syndrome (auriculotemporal syndrome) is most encountered after parotid gland surgery, that is characterized by gustatory sweating and flushing in the region of the ear and cheek. It is due to aberrant reinnervation of postganglionic parasympathetic neurons to nearby denervated sweat glands and cutaneous blood vessels [86].

Owing to the proximity of the ear, TMJ, and parotid gland, a malignant tumour in any of these areas can spread to other areas. The determination of the anatomical origin of the tumor has important prognostic implications [87]. Cancer of the ear canal or middle ear cleft frequently presents as an aural polyp. The biopsy of the aural polyp must be a deep biopsy to avoid misdiagnosis [88]. The anterior spread of the tumour along the fissures of Santorini or foramen of Huschke results in involvement of the TMJ [89]. This would lead to trismus and mandibular deviation during mouth opening [90]. Notably, the report of glandular carcinomas in the ear merit search for a parotid primary or metastatic spread from a distant primary [91,92]. Malignant tumours of the parotid gland may involve the TMJ [93]. Furthermore, deep lobe malignant parotid tumours may present as TMD, resulting in delay in diagnosis [94].

Conclusion

Otologists should have a broad understanding of the TMJ region, especially because TMD frequently presents with otologic complaints. On the other hand, TMJ disorder may be due to primary aetiology in the ear. The DC/TMD should include otologic symptoms in the classification of disorders of the masticatory system.

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