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Forgotten No More: The Superior Turbinate in Migraine-Inducing Mucosal Compression

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ABSTRACT

Headache disorders are among the most common and disabling health conditions, with migraine representing a major contributor to global disease burden. Although the nasal endoscope has greatly advanced the diagnosis and treatment of sinonasal diseases, clinical attention has largely focused on the middle meatus and septal abnormalities, while the superior turbinate has been overlooked. Historically described as the "forgotten turbinate," the superior turbinate may play an important role in rhinogenic headache and migraine through mucosal contact with the nasal septum. This review examines historical observations, clinical studies, neuroanatomical pathways, and biomechanical mechanisms that support mucosal compression between the septum and superior turbinate in particular as a cause of migraine. We review the clinical importance of migraine, a brief history of the clinical investigation into superior turbinate-septal mucosal contact and migraine, the histopathological evidence of mucosal compression injury as the initiating factor in migraine, and the role of nasal nociceptive nerve fibers and the trigeminovascular system in mucosal contact-related migraine. We hope to draw the rhinologist's attention to the superior turbinate in the diagnosis and treatment of the migraine patient.

Keywords: headache, migraine, mucosal contact, superior turbinate, rhinogenic headache

Introduction

The nasal endoscope has revolutionized the diagnosis and treatment of sinonasal disease. However, since its widespread use in the 1980s, rhinologists have focused their attention almost exclusively on the middle meatus and septal abnormalities, paying very little attention to the superior turbinate (ST). The ST has been labeled "the forgotten turbinate"[1] since it is believed to be of minor clinical importance. While this is undoubtedly true with regard to sinus inflammatory disease, evidence suggests the ST is of much greater importance in the diagnosis and management of mucosal contact headache (MCH), also called rhinogenic headache. In this paper, we review the clinical importance of migraine, a brief history of the clinical investigation into ST-septal mucosal contact and migraine, the histopathological evidence of mucosal compression injury as the initiating factor in migraine, and the role of nasal nociceptive nerve fibers and the trigeminovascular system in mucosal contact-related migraine. The relevant clinical data, neuroanatomy, and neurophysiology, and mucosal biomechanics are discussed, as we highlight the importance of mucosal compression (MC) between the ST and septum as a cause of headache, and migraine in particular. By drawing attention to the ST, we hope rhinologists will be better able to diagnose and treat the migraine patient.

History of migraine and mucosal contact

According to the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2021, headache disorders ranked as the second most prevalent condition worldwide, following oral disorders, and tied for the third leading cause of years lived with disability (YLDs), following low back pain, major depressive disorder, and age-related and other hearing loss.[2] Among the 37 conditions affecting the nervous system examined in this study, migraine was the third greatest contributor to DALYs (disability-adjusted life years, one DALY represents one lost year of healthy life), with more than a billion people living with the condition. Only stroke and neonatal brain damage ranked higher, with migraine outpacing conditions, such as dementia. Migraine's prevalence, severity and disabling nature make it particularly important to clinicians.[2]

An historical review will show that headache and/or migraine from ST MC is not a new concept. In 1888, John O. Roe from Rochester, New York was the earliest author to implicate the ST in referred headache. He wrote: "Headaches that have their exciting cause in the nose are reflex in character … occasioned by some abnormal condition which brings together parts that normally should be separate and produces more or less pressure between them…pain reflected from the region of the superior turbinated bone is commonly felt in the frontal and supraorbital region."[3]

Starting in the 1970s Piero Bonaccorsi & V.J. Novak reported on hundreds of patients with various primary headaches, including migraine, who responded to sphenoethmoidectomy to manage middle turbinate (MT) and ST MC.[4,5] Bonaccorsi advocated ethmoidectomy to remove the lateral supports of the MT and ST and then lateralizing or outfracturing the turbinates to alleviate contact against the septum. He also reported that surgery resulted in the resolution of central migraine-associated symptoms such as visual aura.

Clerico[6] in 1996 reported three cases of ST concha bullosa causing migraine (see Figure 1). He noted idiopathic adhesion formation between the ST and septum; ST-septal surfaces fused due to prolonged and intense compressive force. Endoscopic surgery to remove the ST resulted in a dramatic improvement in migraines.

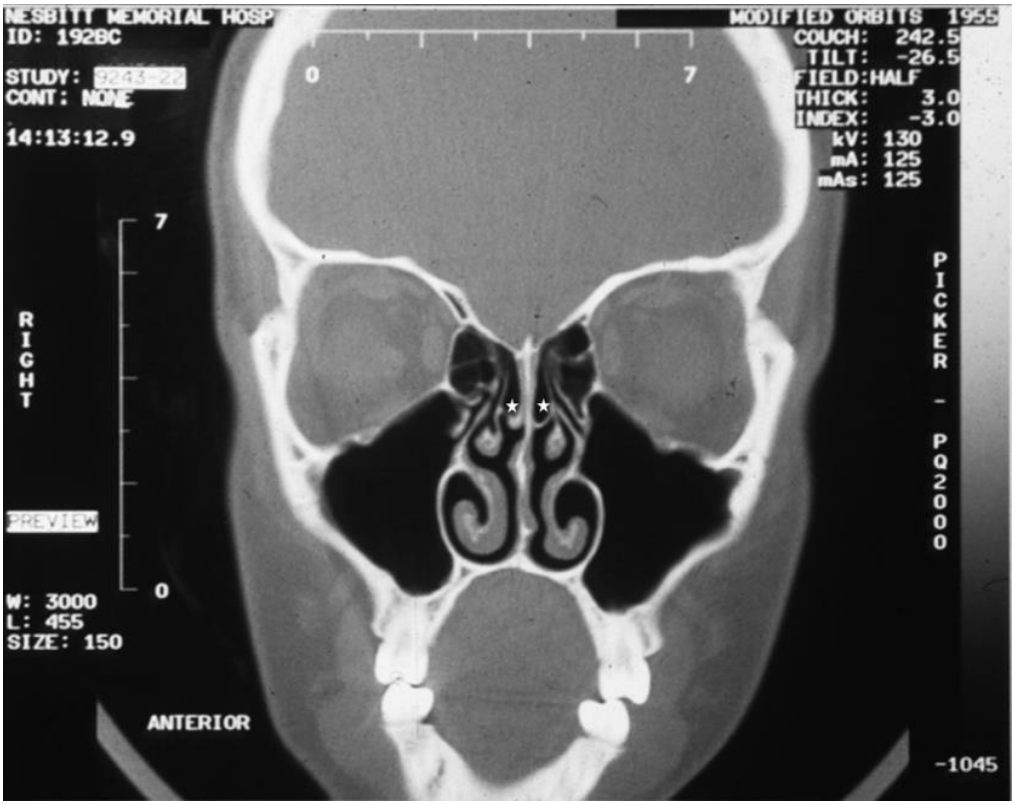

Figure 1: *CT exhibits bilateral septoturbinal contact between the septum and STs. Stars indicate ST concha bullosa.*

Behin et al[7] reported in 2005 endoscopic surgery was successful in treating refractory migraine in 76% of 21 patients. He performed an ethmoidectomy and addressed the ST by lateralizing it.

Ferrero and colleagues[8] in 2014 performed a CT study comparing patients with chronic migraine to controls, finding some level of MC in 51.8% in the chronic migraine group, compared to 36.9% in the control group (difference not statistically significant). However, they found septum-MT MC and septum-MT+ST MC (multiple areas of contact) were more prevalent in the chronic migraine group compared to controls, the difference being statistically significant.

In 2014, Clerico[9] investigated characteristics of pain on nasal stimulation using a monopolar cautery for electrical stimulation and a balloon catheter for mechanical stimulation. His findings largely concurred with a landmark study by McAuliffe and Wolff,[10] but with the exception of finding the ST to be the most pain-sensitive structure in the entire sinonasal cavity.

Though not specifically investigating migraine, Li et al[11] in 2021 reported on 80 patients with chronic headache and CT evidence of MC limited to the ST and the septum (Figure 1). Their surgical approach consisted of septoplasty, lateralization of the MT, and posterior ethmoidectomy with resection of the ST, (the medial wall of the posterior ethmoid sinus). They reported a 95% success rate at 12 months follow-up.

Clerico et al[12] in 2024, like Ferrero et al's CT study, found septoturbinal MC on nasal endoscopy to be a relatively common phenomenon, with an overall MC prevalence of 49% rate, and was associated with migraine. They identified MC most frequently at the ST level and found MT and ST MC in particular were associated with migraine, while inferior turbinate MC was not.

In 2025, Clerico et al[13] described the histopathology of ST MC in four migraine patients. Abnormalities included loss of epithelium and basement membrane, dystrophic calcification, fibroblast deposition, fewer glands, stromal edema, and nerve fiber hypertrophy (see Figure 2). These findings are consistent with mechanical compression-induced tissue injury. The authors could find no previous reports of MC histopathology in the medical literature. They coined the term "migraine-inducing mucosal compression" (MIMuC) to describe their findings.

From this limited histopathologic data, understanding pressure injury biomechanics is important to understanding the microscopic and molecular mechanisms of MICuC.

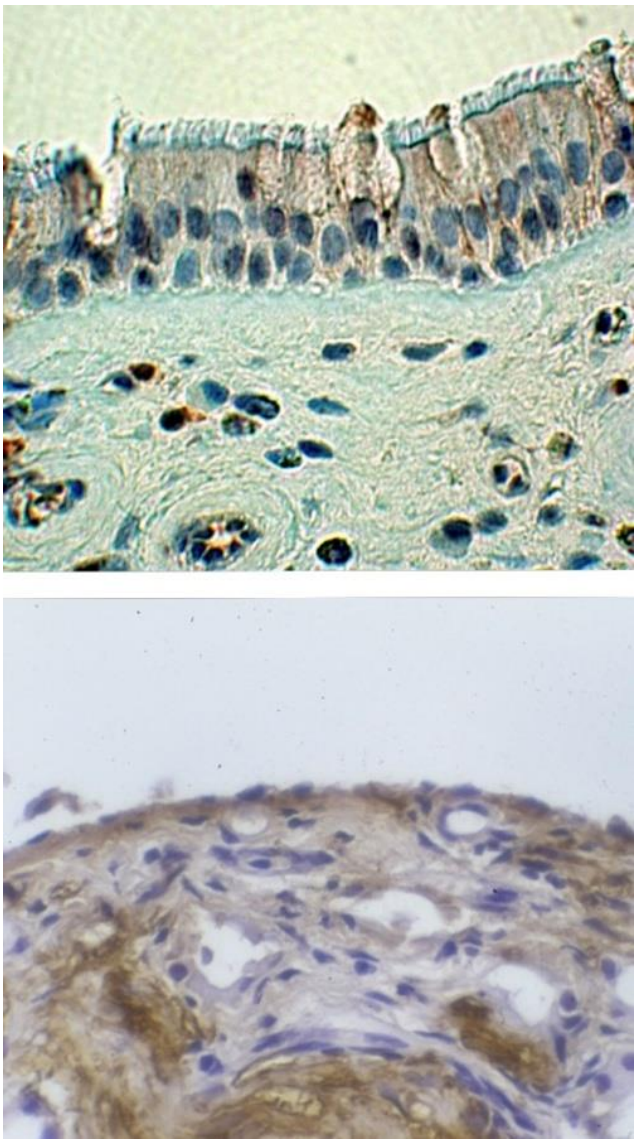

Figure 2. *Top image shows normal ST stained with S100 stain showing intact epithelial surface, loose stroma, sparse nerves. Bottom image from area of ST MC showing denuded epithelial surface, compressed stroma, neural hypertrophy (indicated by brown stain).*

Compression injury

Compression injuries occur when severe or sustained mechanical loading of tissue causes deformation of cells, inflammation, capillary collapse, and ischemia. Mechanical forces fragment cellular cytoskeleton, causing deformation of cell membranes, membrane leakage, and cell rupture. When the pressure on soft tissues exceeds capillary closing pressure (32 mm Hg), cells and tissues are deprived of oxygen and nutrients.[14] Ischemia and cell death follow.

Soft tissue compression injury can occur whenever tissues become trapped between rigid structures. The nasal cavity is perhaps the only location in the body where mucosal tissue can be compressed in this manner. The sensitive nasal mucosa of the septum and ST can be compressed between the perpendicular plate of the ethmoid bone of the septum medially and the turbinate bone laterally. Like compression injuries in general, mechanical compression injury in the nasal cavity is likely influenced by duration, depth, intensity, surface area, and tissue type (resilience of tissue). Most literature sources report soft tissue injury resulting from external mechanical force; however, MC involves internal compression, where tissue is caught between two anatomical structures, both supplied by afferent nerve pathways. Tissue injury leads to the release of inflammatory mediators, such as bradykinin and prostaglandins that trigger peripheral sensory nerve activity.[15]

Since the mucosa of the septum and ST are both innervated by branches of the same nerve (anterior and posterior ethmoid nerves mainly), this could represent two separate points of injury located along a single nerve pathway, which may produce a stronger perception of pain than would occur with a single injury site. Hummel et al[16] surmised that in the nasal mucosa, the length of stimulus and pain threshold is inversely proportional, i.e., longer stimulus times can lower pain threshold. In studies of MCH and MIMuC, individuals who experienced headache for years would obviously have a correspondingly long stimulus duration time. As a result, those with MIMuC experience and are expected to have a lower pain threshold.

The depth and strength of compressive forces influence submucosal damage as well as whether capillary closing pressure is surpassed. A reduction in capillary blood flow resulting from this pressure may result in ischemia and later cell death. The total compressed area also determines the number of the sensory neurons activated; larger regions of injury can increase pain intensity. The ability of tissues to withstand mechanical stress also impacts injury susceptibility. Regions within the nasal cavity with thicker subepithelial layers (such as the inferior turbinate) may provide greater resistance to damage and are therefore less likely to produce a painful sensation.[15]

The Nasal Mucosa Nociception and Migraine

Nasal mucosa receives sensory innervation from terminal branches of the ophthalmic (V1) and maxillary (V2) divisions of the trigeminal nerve and is responsive to painful stimuli. The ophthalmic division provides most of the sensory input to the cerebral vasculature and the dura mater. The nasociliary nerve, a branch of V1, gives rise to the anterior and posterior ethmoid nerves, which transmit sensory information from the superior regions of the nasal cavity.

Sensory endings of the trigeminal nerve contain multiple receptor types capable of detecting different forms of stimulation. Aδ (A-delta) and C-nociceptors transmit painful sensation from nociceptive stimulation. Nociceptors, derived from the Latin nocere “to harm,” are peripheral sensory neurons that detect potentially harmful stimuli. Aδ fibers are myelinated afferent fibers of moderate diameter that transmit brief, well-localized sensations of sharp pain. These fibers are among some of the smallest myelinated axons in the body and measure between 2 to 5 μm in diameter. In contrast, C-fibers lack myelin and are smaller, ranging 0.2 to 1.5 μm in diameter. They conduct impulses more slowly and are responsible for diffuse, poorly localized pain sensations that are commonly associated with prolonged or persistent stimuli.[16]

Functionally, both Aδ fibers and C fibers respond to mechanical stimuli. When stimulated, both fiber types release neuropeptides including calcitonin gene-related peptide (CGRP), pituitary adenylate cyclase activating polypeptide (PACAP), substance P, and neurokinin A, which contribute to nociceptive signaling.

Unlike tissues protected by stratified squamous epithelium, the nasal cavity lacks this more durable epithelial covering. As a result, external stimuli can readily reach the free nerve endings within the nasal mucosa, increasing its susceptibility to painful stimulation. During trigeminal nociception, strong chemical agents such as capsaicin (found in cayenne pepper), mechanical forces like compression or cutting, or extremes of temperature activate nociceptors. These stimuli generate electrochemical signals that travel along peripheral nerves to the trigeminal ganglion, then to the trigeminal nucleus within the brainstem, and subsequently to the thalamus before ultimately reaching the somatosensory cortex.[17]

Studies on MCH emphasize the importance of mucosal contact occurring in the MT and ST regions. As noted previously, these areas receive their primary innervation from the anterior and posterior ethmoid branches of V1, with additional contributions from terminal branches of V2. The anterior ethmoid nerve has also been shown to contain autonomic fibers,[18] which may help explain why mucosal contact in this region can trigger the autonomic symptoms frequently associated with migraine.

Research involving both animal models and human subjects has demonstrated that sensory nerve endings are not evenly distributed throughout the nasal mucosa. Instead, localized regions of increased or decreased trigeminal sensitivity are present. One example is the observation that the medial (septal) surface of the middle turbinate exhibits greater sensitivity than its

lateral side.[19] Although more research is needed, this finding suggests that mucosal contact between the uncinate process and the lateral portion of the middle turbinate, previously proposed as a cause of MCH,[20] may be less clinically significant than contact occurring between the nasal septum and the MT/ST. The uneven distribution of sensory fibers may also help explain why some individuals with MC remain asymptomatic and why small, localized areas of contact may not produce clinically meaningful symptoms.

Transient receptor potential (TRP) channels are membrane-bound ion channels composed of protein complexes found in the plasma membranes of many different cell types. One member of this family, transient receptor potential ankyrin 1 (TRPA1), functions as a receptor capable of responding to a variety of stimuli. TRPA1 is highly expressed in C fibers involved in neuropathic pain pathways and serves as an important sensor for painful and thermal stimuli.[21] Because TRPA1 receptors are present not only on intracranial axons but also on extracranial branches that innervate structures such as the nasal mucosa, skull periosteum, and pericranial muscles, nociceptive stimulation of extracranial tissues may activate intracranial fibers through an axon reflex mechanism.[21] This process can lead to the release of vasoactive neuropeptides within meningeal tissue, increased intracranial blood flow, and activation of pathways involved in migraine pathophysiology.

Experimental work in animal models supports this mechanism. Painful stimulation applied to the nasal and sinus cavities of rats has been shown to increase meningeal blood flow and promote plasma protein leakage from the dura.[22] These findings indicate a physiological connection between nociceptive stimulation within the nasal cavity and the activation of dural afferent pathways through antidromic signaling. The resulting dural neurogenic inflammation is a process commonly associated with migraine.[23]

The Trigeminovascular System and Migraine

In 1979, Moskowitz and colleagues first described the trigeminovascular system (TVS) in the context of migraine,[24] emphasizing the importance of the trigeminal nerve and its projections, which contain vasoactive neuropeptides that extend to the meninges and dural vasculature. Their work highlighted how activation of trigeminal pathways leads to the release of these neuropeptides, producing vasodilation and a form of sterile neurogenic inflammation.

Before this model was proposed, migraine with or without aura (associated visual changes) was thought to originate within the central nervous system. Earlier theories focused on cortical spreading depression and other intracranial events as the primary mechanisms responsible for initiating migraine symptoms. More recent models, however, suggest that migraine begins with activation of the trigeminovascular system within the peripheral nervous system, particularly at the trigeminal ganglion.[25,26,27] From this structure, V1 and V2 branches of the trigeminal nerve extend to innervate the meninges and intracranial blood vessels.

Pain signals arising from the trigeminal ganglion converge centrally within the trigeminocervical complex, which is located in the brainstem and upper cervical spinal cord. From this region, second-order neurons project upward to higher brain centers, including the thalamus, hypothalamus, and somatosensory cortex. Activation of this pathway is believed to generate the characteristic pain of migraine through the release of neuropeptides, most notably CGRP and PACAP, within the dura. These substances are potent vasodilators and have been shown to provoke migraine attacks. As a result, vasodilation, once considered the primary cause of migraine, is now viewed more likely as a secondary consequence of trigeminovascular activation.[27]

While the concept of MIMuC substantiates that migraine begins with peripheral activation of the trigeminovascular system, it implies that the initiating stimulus may occur distal (more peripheral) to the trigeminal ganglion. Evidence indicates that mucosal contact within the nasal cavity, particularly between the septum and MT/ST, may serve as the initial nociceptive trigger. This stimulus then send signals back toward the trigeminal ganglion, after which the established trigeminovascular pathway proceeds as previously described.

Importantly, this concept does not challenge the current understanding of migraine pathophysiology but instead shifts the proposed site of initiation slightly further along the peripheral pathway from the trigeminal ganglion to the terminal branches of V1 and V2 within the nasal mucosa. This perspective supports the growing view that migraine attacks may arise from peripheral triggers rather than solely from processes within the central nervous system.[25,26,27]

Practical approaches

Since the ST can play a crucial role in the migraine patient, evaluation and management of ST MC is critical. Unfortunately, the ST is frequently difficult to visualize on nasal endoscopy, and sufficient topical or local anesthesia to and lateralization of the MT is often necessary to accomplish this feat (see Figure 3). Once adequately visualized, the ST can then be topically anesthetized or injected with local anesthetic at the time the patient has a migraine to determine if the ST MC is the cause of headache. This technique of diagnostic nerve block has been described elsewhere.[15] Once the diagnosis of MIMuC from ST MC has been made, the ST can then be either lateralized (outfractured) or partially resected under general anesthesia for definitive management. The senior author prefers subtotal superior turbinectomy, which has been described elsewhere.[15]

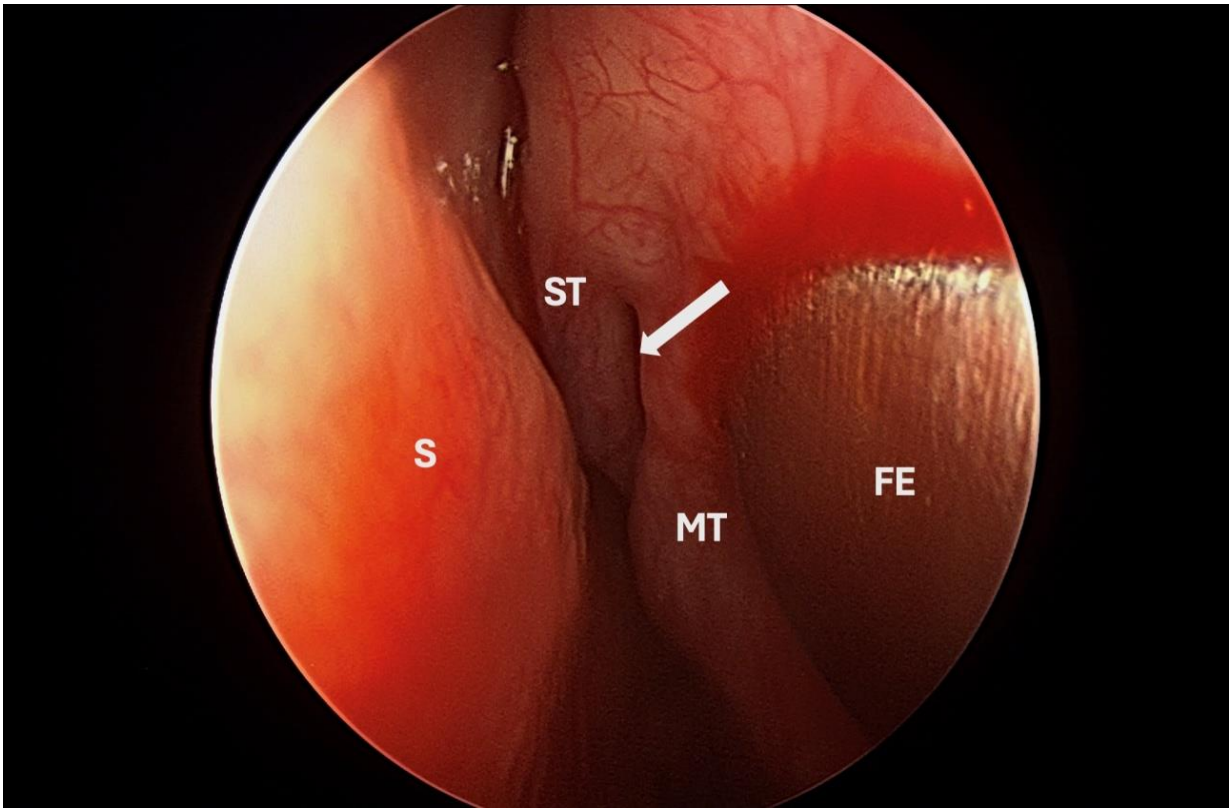

Figure 3: *S=septum, ST=superior turbinate, ARROW points to superior meatus, MT=middle turbinate, FE=Freer elevator*

Conclusion

The ST has been found to be the most sensitive structure in the nasal cavity, and MC at that level occurs most frequently in migraine patients. High success rates at treating migraine have been reported with alleviating ST MC. Mucosal contact with the nasal cavity, particularly between the septum and MT/ST, may act as an initial nociceptive trigger that activates trigeminal nerve endings and initiates the established migraine pathophysiologic pathway. This does not contradict current models of migraine pathophysiology but is consistent with these models. Clinical and histopathological evidence points to the ST as having an important role in MIMuC. However, the topic has yet to be fully explored, and further clinical and experimental studies are needed to clarify and better understand this relationship.

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