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Otolaryngologic-cutaneous associations, mechanisms, and diagnostic distinctions: a narrative review

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Abstract

Reported otolaryngologic-cutaneous relationships arise through distinct mechanisms, including direct infectious spread, shared inflammatory predisposition, local sensory-neuroimmune signaling, and neuropathic-like symptoms without nerve injury. This structured narrative review evaluates clinical and translational evidence for these categories and distinguishes neurogenic inflammation from neuropathic pain and itch. PubMed/MEDLINE was searched through August 12, 2026, with supplementary targeted association searches on September 1, 2026. Acute bacterial rhinosinusitis may cause periorbital or orbital cellulitis through direct anatomic spread. Associations of chronic rhinosinusitis with rosacea and allergic rhinitis with atopic dermatitis or eczema remain observational and do not establish causality or temporal sequence. Sensory-neuropeptide signaling is best established for pruritus and remains suggestive for rosacea. Intense or persistent nociceptor activation can amplify neurogenic inflammation and produce burning, stinging, itch, or allodynia that resembles neuropathic symptoms without the somatosensory lesion required for neuropathic disease. Clinical evaluation should classify findings as infectious, shared inflammatory or atopic, neurogenic inflammatory, neuropathic-like, or neuropathic and should direct treatment to the established disorder. Human evidence does not support targeted treatment of a shared otolaryngologic-cutaneous neuroimmune pathway.

Key words: neurogenic inflammation; rhinosinusitis; rosacea; atopic dermatitis; pruritus.

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Introduction

Scope and definitions

Neurogenic inflammation is an inflammatory response initiated or amplified by sensory-neuron activation and peripheral neuropeptide release, producing vasodilation, plasma extravasation, and immune signaling.[1,2] It may accompany inflammatory or nociceptive pain without a demonstrable nerve lesion. Neuropathic pain, in contrast, is caused by a lesion or disease of the somatosensory nervous system.[3] Concurrent sinonasal and cutaneous findings may reflect distinct infectious, inflammatory, sensory-neuroimmune, or neuropathic-like processes.[4] This structured narrative review evaluates the strength and directness of evidence linking ear, nose, and throat disorders with cutaneous findings and outlines clinically relevant diagnostic and therapeutic implications.

Methods

This structured narrative review was guided by the Scale for the Assessment of Narrative Review Articles (SANRA) domains of importance, aims, search description, referencing, scientific reasoning, and presentation of relevant outcome data.[5] PubMed/MEDLINE was searched from database inception through August 12, 2026 using three concept-based strategies. The otolaryngologic search was: ((“neurogenic inflammation”[Title/Abstract] OR “neurogenic inflammatory”[Title/Abstract]) AND (otolaryngolog*[Title/Abstract] OR rhin*[Title/Abstract] OR sinonasal[Title/Abstract] OR rhinitis[MeSH Terms] OR sinusitis[MeSH Terms] OR “nasal mucosa”[MeSH Terms] OR nose[Title/Abstract] OR ear[Title/Abstract] OR laryngeal[Title/Abstract] OR larynx[Title/Abstract])) AND English[Language].

The cutaneous search was: ((“neurogenic inflammation”[Title/Abstract] OR “neurogenic inflammatory”[Title/Abstract]) AND (skin[Title/Abstract] OR cutaneous[Title/Abstract] OR dermato*[Title/Abstract] OR rosacea[MeSH Terms] OR rosacea[Title/Abstract] OR pruritus[MeSH Terms] OR prurit*[Title/Abstract] OR “dermatitis, atopic”[MeSH Terms] OR “atopic dermatitis”[Title/Abstract])) AND English[Language]. The neuropathic-differential search was: (((“neuropathic pain”[Title/Abstract] OR “neuropathic itch”[Title/Abstract] OR “neuropathic pruritus”[Title/Abstract]) AND (“trigeminal neuralgia”[Title/Abstract] OR “postherpetic neuralgia”[Title/Abstract])) OR “neuropathic itch”[Title] OR “neuro-

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pathic pruritus"[Title]) AND (diagnos*[Title/Abstract] OR pathophysiolog*[Title/Abstract] OR classific*[Title/Abstract] OR differenti*[Title/Abstract]) AND (review[Publication Type] OR guideline[Publication Type]) AND English[Language]. Because these strategies required neurogenic-inflammation or neuropathic terminology, supplementary PubMed/MEDLINE searches were conducted on September 1, 2026 for the chronic rhinosinusitis (CRS)-rosacea and rhinitis-eczema or atopic dermatitis associations. The exact searches were ("chronic rhinosinusitis"[Title/Abstract] OR CRS[Title/Abstract]) AND rosacea[Title/Abstract] and ("allergic rhinitis"[Title/Abstract] OR rhinitis[MeSH Terms]) AND ("atopic dermatitis"[Title/Abstract] OR eczema[Title/Abstract]). The Al-Balbeesi CRS-rosacea study and Steiner rhinitis-eczema study were identified through these targeted searches rather than citation chasing. Reference lists of relevant articles and reviews were screened to identify additional mechanistic sources.

Eligible evidence included English-language human observational and interventional studies, guidelines, case reports, and peer-reviewed reviews directly addressing neuroimmune mechanisms or clinically relevant otolaryngologic-cutaneous associations. Animal and *in vitro* studies were retained only when human mechanistic evidence was unavailable and are identified as preclinical. Conference abstracts were excluded from clinical-effectiveness conclusions; one conference abstract was retained solely to characterize the limited evidence underlying neurogenic switching. Duplicate publications and articles without a direct otolaryngologic, cutaneous, or neuroimmune focus were excluded. Purely infectious or structural disorders were retained only when needed to distinguish direct complications from proposed remote neuroimmune mechanisms. Studies were selected purposively according to relevance, methodological strength, and clinical applicability and were synthesized narratively. Formal duplicate screening, risk-of-bias or certainty assessment, and meta-analysis were not performed.

Search results

The otolaryngologic, cutaneous, and neuropathic-differential strategies retrieved 164, 643, and 156 records, respectively. Because the searches overlapped and selection was purposive, these yields were not combined into a unique-record total. The final narrative synthesis comprised 50 cited sources, including sources identified through the supplementary targeted searches and citation chasing.

Neurogenic inflammation in ear, nose, and throat disorders

Mechanisms linking neurogenic inflammation and cutaneous findings

Activation of mucosal or cutaneous sensory afferents can generate two related but separable outputs. Orthodromic action potentials carry nociceptive pain-related signals and pruriceptive input (itch-evoking sensory signals) toward the central nervous system. Peripheral axon-reflex signaling provides a second output.[2,6,7] Unmyelinated C fibers are the principal effector fibers; thinly myelinated A-delta fibers contribute primarily to nociception.[6] Table 1 summarizes these mechanisms and their distinction from neuropathic disease.

Acute nociceptor activation can initiate an axon-reflex response, whereas intense or persistent noxious stimulation can amplify or sustain peripheral release of substance P and calcitonin gene-related peptide, promoting vasodilation, plasma extravasation, immune signaling, and sensory sensitization.[2,6] Glutamate and nerve growth factor can further modulate neuronal excitability and immune signaling. Neurogenic inflammation does not require a nerve lesion, although nociceptor sensitization may produce neuropathic-like symptoms.

Substance P activates the neurokinin-1 receptor and promotes nitric oxide and prostaglandin E2 signaling, which can contribute to vasodilation, plasma extravasation, and peripheral sensitization. It also modulates innate and adaptive immune responses.[2]

Calcitonin gene-related peptide is released from peripheral sensory neurons, promotes vasodilation, and can sensitize afferents in concert with substance P.[2,6] It also modulates innate immune cells and may influence type 2 inflammatory responses.[2]

Nerve growth factor released by fibroblasts and immune cells signals through tropomyosin receptor kinase A on sensory neurons and can influence mast cells and eosinophils. These effects contribute to peripheral sensitization and mast-cell degranulation.[6]

Neurogenic switching is a hypothesis that neural activation at one anatomical site may provoke inflammation at a distant site.[9] In an otolaryngologic animal model, intranasal formalin or capsaicin increased nasal and dural plasma extravasation and activated trigeminal nociceptive pathways.[10] Other cited evidence consists of reports outside the otolaryngologic setting and animal experiments.[11-14] These findings suggest a sinonasal-trigeminovascular connection

Table 1. Neurogenic inflammation, neuropathic-like symptoms, and neuropathic disease.

Stage	Component	Key events
Sensory-neuron activation	Predominantly C fibers; A-delta fibers in nociception	Orthodromic nociceptive or pruriceptive signaling and peripheral axon-reflex activation.[2,6,7]
Local neurogenic inflammation	Substance P, CGRP, and NGF	Intense or persistent nociceptor activation can amplify neuropeptide release, vasodilation, plasma extravasation, immune signaling, and sensory sensitization.[1,2,6]
Mechanistic distinction	Neuropathic-like symptoms *versus* somatosensory lesion or disease	Neurogenic inflammation may produce burning, pruritus, hyperalgesia, or allodynia without nerve injury; neuropathic pain or itch requires a lesion or disease.[2,3,7,8]

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but do not demonstrate that localized ear, nose, or throat inflammation causes remote cutaneous disease in humans.

Cutaneous and mucosal sensory neurons plausibly contribute to local neuroimmune responses after allergen, irritant, or pathogen exposure. Direct infection, primary dermatologic disease, and shared atopic inflammation may also explain concurrent ear, nose, throat, and skin findings. Current human evidence is insufficient to attribute remote erythema, rash, or edema after an otolaryngologic insult to neurogenic switching alone.

Dermatologic associations of sinonasal disease

Acute rhinosinusitis and orbital complications

Rhinosinusitis involves inflammation of the sinonasal mucosa and may impair mucociliary clearance.[15,16] Acute bacterial rhinosinusitis may cause periorbital or orbital cellulitis through contiguous spread across the thin lamina papyracea or through valveless venous channels.[17,18] This is an anatomic infectious complication rather than a remote manifestation of neurogenic inflammation. Pain with eye movement, ophthalmoplegia, or proptosis should prompt urgent evaluation for orbital involvement.

Chronic rhinosinusitis and rosacea

Evidence connecting CRS with rosacea remains observational. Rosacea is a chronic facial inflammatory disorder characterized by flushing, erythema, telangiectasia, papules, or pustules.[19] Cathelicidin expression in CRS mucosa and rosacea skin provides a possible shared inflammatory pathway.[20,21] In a case-control study of 28 female Saudi participants with rosacea and 20 age- and sex-matched dermatologic controls, radiographic evidence of CRS was present in 19 of 28 (67.9%) and 4 of 20 (20%), respectively (p=0.003).[22] The small cross-sectional study used plain radiography and therefore demonstrates co-occurrence without establishing temporal sequence or causality.

Allergic rhinitis and atopic dermatitis

Allergic rhinitis and atopic dermatitis or eczema frequently coexist and share type 2 inflammatory mediators, including interleukin 4, interleukin 13, immunoglobulin E, mast cells, and eosinophils.[23-28] In the cited cross-sectional Swiss study of young adults, 68 participants reported seasonal rhinitis plus eczema and 48 (71%) had a positive sensitization test.[28] This percentage represents sensitization within a symptom-defined subgroup, not the prevalence of rhinitis among patients with clinically diagnosed atopic dermatitis. Shared pathways support atopic multimorbidity but do not establish that either condition causes the other or determine temporal order.

Cutaneous neuroimmune symptoms and neuropathic differentials

Rosacea: suggestive neuroimmune evidence

Rosacea involves immune and neurovascular dysregulation, but evidence establishing neurogenic inflammation as a causal mechanism remains limited.[29-31] A targeted metabolomics study incorporated human samples and experimental models and linked altered amino-acid signaling with vasoactive neuropeptide and nitric oxide pathways; key mechanistic findings remained preclinical.[31] Erythematotelangiectatic rosacea may include flushing, visible vessels, burning, or stinging.[32] Improvement of erythema with the alpha-1 adrenergic agonist oxymetazoline in two case reports demonstrates a vasoconstrictive treatment effect and does not establish neurogenic inflammation as the underlying mechanism.[33] The proposed association is therefore biologically plausible but hypothesis-generating, and current evidence does not show that CRS initiates or worsens rosacea through neurogenic pathways.

Pruritus and neurogenic itch

Pruritus is transmitted predominantly by unmyelinated C-fiber pruriceptors through histamine-dependent and histamine-independent pathways; thinly myelinated A-delta fibers may also contribute.[7] Mediator-induced itch in the absence of a nerve lesion may accompany neurogenic inflammation. Neuropathic itch, by comparison, results from a lesion or disease of somatosensory pathways and requires separate classification.

Neurogenic inflammation does not require a somatosensory nerve lesion, but nociceptor sensitization can produce burning pain, pruritus, hyperalgesia, or allodynia that resembles neuropathic symptoms.[2,3,7] These neuropathic-like symptoms do not by themselves establish neuropathy. Postherpetic itch and postherpetic neuralgia result from varicella-zoster-associated nerve injury and are neuropathic disorders.[34-36] Small-fiber neuropathy is a peripheral neuropathy involving thinly myelinated A-delta and unmyelinated C fibers, which mediate pain, temperature, itch, and autonomic function. Diagnosis requires compatible clinical signs plus structural or functional small-fiber abnormalities; sensory symptoms alone are insufficient (Table 2).[8,37]

Table 2. Strength and interpretation of key otolaryngologic-cutaneous associations.

Association or condition	Evidence summarized	Interpretation
Acute bacterial rhinosinusitis-cellulitis	Established anatomic and infectious spread	Direct complication; not remote neurogenic inflammation.[17,18]
CRS-rosacea	One small cross-sectional case-control study; shared cathelicidin expression	Association only; temporality and causality are unknown.[20-22]
Allergic rhinitis-atopic dermatitis	Epidemiologic co-occurrence and shared type 2 inflammation	Atopic multimorbidity; neither condition is shown to cause the other.[23-28]
Pruritus and neuropathic-like symptoms	Strong C-fiber evidence for itch; nociceptor sensitization may produce burning, hyperalgesia, or allodynia without nerve injury	Symptoms can overlap with neuropathy, but neuropathic classification requires a somatosensory lesion or disease.[2,3,7,8,37]

Diagnostic and therapeutic approaches

Diagnostic strategies

Clinical evaluation should first identify the most likely mechanism: direct infectious extension; a primary inflammatory dermatosis or shared atopic disease; neurogenic inflammation with neuropathic-like burning, pruritus, hyperalgesia, or allodynia but no demonstrated nerve lesion; or neuropathic pain or itch caused by a somatosensory lesion or disease. Relevant features include the temporal relationship between ear, nose, or throat and cutaneous symptoms; laterality; lesion morphology; dermatomal distribution; sensory loss; allodynia; autonomic features; and reproducible triggers.[38]

Examination should compare involved and uninvolved skin, document primary and secondary lesion morphology, and assess pinprick, thermal sensation, allodynia, and hyperalgesia when neuropathy is suspected. Proptosis, ophthalmoplegia, or pain with eye movement warrants urgent evaluation for orbital cellulitis. Objective sensory loss, a dermatomal distribution, or autonomic signs should prompt consideration of neuropathic disease rather than neurogenic inflammation alone.

Routine nerve-conduction studies evaluate large myelinated fibers and are generally normal in isolated small-fiber neuropathy. When small-fiber neuropathy is suspected, diagnosis should integrate compatible clinical signs with structural or functional small-fiber abnormalities, such as reduced intraepidermal nerve-fiber density on skin biopsy or abnormal quantitative sensory or autonomic testing.[8,37,39] These tests identify small-fiber pathology; they do not diagnose neurogenic inflammation.

Therapeutic interventions

Treatment should target the established diagnosis and demonstrated mechanism. Direct infectious complications require appropriate antimicrobial and surgical evaluation; allergic rhinitis, atopic dermatitis, and rosacea should receive condition-specific therapy; and neuropathic pain or itch should be managed as neuropathic disease. Neuropathic-like symptoms associated with neurogenic inflammation do not by themselves establish a neuropathic disorder or justify treatment directed at nerve injury. No therapy has been shown to treat a remote cutaneous manifestation caused by ear, nose, or throat-origin neurogenic inflammation.

Intranasal corticosteroids reduce nasal inflammatory cells and improve selected symptoms of allergic rhinitis and rhinosinusitis.[40-43] These data support treatment of the nasal disorder; they do not show that corticosteroids prevent or treat a remote cutaneous response mediated by neurogenic inflammation. Systemic corticosteroids should be reserved for established indications rather than inferred ENT-cutaneous crosstalk.

Second-generation H1 antihistamines are evidence-based for allergic rhinitis and selected histamine-mediated dermatoses.[44] Topical antihistamines have limited evidence for pruritus and may cause local adverse effects; their use should follow the specific dermatologic diagnosis.[45] Efficacy in rhinitis or urticaria does not establish efficacy for an unproven remote neurogenic-inflammatory pathway.

Neuropeptide-directed therapies remain investigational for otolaryngologic-associated cutaneous disease. The supporting literature cited here concerns potential neuropeptide Y targets, chemotherapy-induced nausea, gastrointestinal motility, or bronchoconstriction rather than clinical treatment of an ear, nose, throat-cutaneous syndrome.[46-49] Trials with prespecified cutaneous outcomes are required before neurokinin or calcitonin gene-related peptide antagonists can be recommended for this purpose.

A small translational study of intravenous lidocaine in atopic dermatitis is preliminary and requires clinical confirmation.[50]

Future directions and research opportunities

Research priorities across otolaryngology, neurology, and dermatology

Sensory-neuropeptide signaling is well described at the cellular level, but direct human evidence linking a localized otolaryngologic disorder to a remote cutaneous manifestation remains sparse. Future studies should separate direct infection, shared inflammatory predisposition, local neurogenic inflammation with neuropathic-like symptoms, and neuropathic disease caused by a somatosensory lesion rather than combining these mechanisms under a single label.

Neurogenic switching warrants investigation as a hypothesis. Prospective human studies should establish the temporal relationship between ear, nose, or throat activity and cutaneous outcomes, define reproducible phenotypes, and include comparator groups before estimating prevalence or risk factors.

Collaborative registries involving otolaryngology, dermatology, allergy, and neurology could pair standardized clinical phenotyping with biomarkers, imaging, and longitudinal follow-up. Such designs would clarify whether observed associations reflect shared susceptibility, treatment effects, direct extension, or sensory-neuroimmune signaling.

Developing integrated treatment approaches

Integrated care is most useful when it clarifies diagnosis and directs established condition-specific treatment. Concurrent ear, nose, throat and cutaneous symptoms should prompt assessment for direct infectious spread, shared atopy, an independent dermatosis, medication effects, and neuropathic disease before a shared neuroimmune mechanism is inferred.

Targeted neuropeptide therapies should be evaluated in controlled trials before clinical use for putative downstream skin effects. Trials should distinguish improvement in local inflammatory or neuropathic-like symptoms from treatment of a verified neuropathic disorder.

Future interventional studies should prespecify both otolaryngologic and cutaneous outcomes, document disease chronology, and stratify participants by evidence of nerve injury. This approach would test whether modifying a sensory-neuropeptide pathway changes clinically meaningful outcomes beyond standard treatment of each condition.

Limitations

This review was limited to PubMed/MEDLINE and English-language publications and used purposive narrative selection; relevant evidence may therefore have been missed. No duplicate screening or formal risk-of-bias or certainty assessment was performed. Publication bias is possible. Direct human evidence linking otolaryngologic-origin neurogenic inflammation with remote cutaneous manifestations was sparse; much of the mechanistic discussion derives from animal experiments, small observational studies, case reports, or evidence extrapolated from other diseases. Heterogeneity in terminology further complicated comparison, particularly the

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inconsistent use of neurogenic inflammation, neuropathic pain, and neurogenic or neuropathic itch. The associations summarized here cannot establish causality or temporal sequence.

Conclusions

Reported otolaryngologic-cutaneous relationships fall into mechanistically distinct categories. Acute bacterial rhinosinusitis can produce periorbital or orbital cellulitis through direct infectious spread; CRS-rosacea and allergic rhinitis-eczema findings are observational associations that may reflect shared inflammatory susceptibility; and local sensory-neuropeptide signaling is best supported for pruritus and remains suggestive for rosacea. Neurogenic inflammation can produce neuropathic-like burning, pruritus, hyperalgesia, or allodynia without the somatosensory lesion required for neuropathic disease. Clinical evaluation and treatment should therefore follow the established infectious, dermatologic, atopic, neurogenic-inflammatory, or neuropathic mechanism. Prospective human studies are needed before a shared otolaryngologic-cutaneous neuroimmune pathway or targeted therapy can be established.

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