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Commentary

Vestibular Symptoms: An Underrecognized Extra-Sinonasal Dimension of Chronic Rhinosinusitis

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Abstract

Chronic rhinosinusitis (CRS) is a prevalent inflammatory disease traditionally defined and assessed by sinonasal symptoms such as nasal obstruction, rhinorrhea, facial pressure, and olfactory dysfunction. However, the burden of CRS extends beyond the sinonasal compartment, including a range of systemic and functional complaints that are not routinely addressed in standard rhinologic practice. Among these, vestibular symptoms, including dizziness, imbalance, and nonspecific disequilibrium, are frequently reported by patients with CRS, yet remain underrecognized and poorly integrated into current diagnostic frameworks and clinical guidelines, despite being captured as a single, psychometrically limited item within the 22-item Sinonasal Outcome Test (SNOT-22). Clinical observations and limited published data, mostly small observational studies and case reports, suggest that vestibular symptoms may fluctuate in parallel with CRS disease activity and may improve following effective medical or surgical control of sinonasal inflammation. Proposed mechanisms include Eustachian tube dysfunction, immune-mediated and neurogenic pathways, trigemino-vestibular interactions, and altered multisensory integration, although current evidence does not establish a causal relationship between CRS disease activity and measurable peripheral vestibular dysfunction. Comparative observations in allergic rhinitis and post-viral upper-airway inflammation situate CRS within a broader inflammatory upper-airway–vestibular interface. This Commentary highlights vestibular dysfunction as an underappreciated extra-sinonasal dimension of CRS with potential clinical and functional relevance. By drawing attention to this clinical blind spot, we aim to encourage more systematic symptom inquiry, interdisciplinary dialogue, and prospective research into the functional consequences of chronic upper-airway inflammation.

Keywords: chronic rhinosinusitis; vestibular symptoms; dizziness; extra-sinonasal manifestations; functional symptoms; quality of life

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1. Introduction

Chronic rhinosinusitis (CRS) is a prevalent and heterogeneous inflammatory disease of the nasal and paranasal sinuses, defined by persistent symptoms lasting longer than 12 weeks and supported by objective endoscopic or radiologic findings, according to the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS 2020) [1]. CRS affects approximately 10–11% of the adult population in Europe [2] and represents a major cause of chronic morbidity, with quality-of-life impact comparable to that of asthma and other major chronic conditions [3]. Two principal phenotypes are recognized under EPOS: CRS with nasal polyps (CRSwNP) and CRS without nasal polyps (CRSsNP) [1]. The few studies addressing vestibular involvement have included both phenotypes, with limited subgroup analyses; in this Commentary we therefore consider CRS broadly while flagging the few instances in which the available evidence specifically concerns CRSwNP or CRSsNP.

Traditional clinical assessment of CRS focuses on cardinal sinonasal symptoms, nasal obstruction, rhinorrhoea, facial pressure, and olfactory dysfunction [4], but CRS is associated with a broader range of systemic and functional symptoms that extend beyond the sinonasal compartment, including fatigue, sleep disruption, cognitive impairment, and otologic complaints [5,6]. These manifestations reflect the multisystem consequences of chronic upper-airway inflammation and its interaction with adjacent anatomical structures and central sensory processing.

Within this broader clinical phenotype, vestibular symptoms, dizziness, imbalance, and nonspecific disequilibrium are described by patients with CRS but may be underrecognised in routine rhinologic practice [7].

Although the 22-item Sinonasal Outcome Test (SNOT-22) explicitly includes a "dizziness" item [8], item-response analyses have shown that this item performs less informatively than other items in the otologic/facial-pain subdomain [9], and dizziness is rarely the focus of dedicated clinical inquiry, mechanistic discussion, or guidance [10]. In this sense, the underrecognition we describe is not absolute but relative: the symptom is captured in routine quality-of-life assessment yet is seldom interrogated in depth, mechanistically framed, or longitudinally followed.

Limited published data, mostly small observational studies, case reports, and pathophysiological hypotheses, suggest that vestibular symptoms in CRS may fluctuate with sinonasal disease activity and may improve following effective medical or surgical control of inflammation [11,12]. These observations are hypothesis-generating rather than confirmatory: objective vestibular testing does not consistently identify peripheral abnormalities [7,13], and a causal relationship between CRS disease activity and measurable vestibular dysfunction has not been established. The aim of this Commentary is therefore not to claim such a relationship, but to argue that vestibular complaints in CRS deserve systematic recognition, careful differential diagnosis, and prospective study.

2. Pathophysiological Considerations Linking CRS and Vestibular Symptoms

The relationship between CRS and vestibular symptoms is likely multifactorial. In a subset of patients, dizziness and imbalance may arise from chronic upper-airway inflammation interacting with peripheral sensory input, neurogenic pathways, and central multisensory processing, rather than from a primary vestibular lesion. The mechanistic framework outlined below is drawn from preclinical and clinical work on related conditions and from a small literature directly addressing CRS; we present it as a plausible, hypothesis-generating model rather than as established physiology.

2.1. Eustachian Tube Dysfunction and Middle-Ear Homeostasis

Eustachian tube dysfunction (ETD) is commonly observed in CRS [14] and is the most anatomically straightforward candidate mechanism for vestibular complaints. Chronic inflammation of the shared respiratory mucosa can impair pressure regulation within the middle ear, and a recent systematic review and meta-analysis demonstrated that endoscopic sinus surgery significantly reduces ETD-related symptoms in patients with CRS [15]. Even subtle or intermittent alterations in middle-ear homeostasis may influence vestibular perception and contribute to nonspecific dizziness or imbalance, although the consensus diagnostic framework for ETD emphasizes that such effects are more likely to alter sensory input than to produce true vestibular end-organ dysfunction [16].

2.2. Inflammatory and Neurogenic Mechanisms

CRS is characterized by persistent inflammatory activity, frequently associated with type-2 immune responses and sustained cytokine release [17]. A recent narrative review of vestibular dysfunction in immune-mediated disease has summarized converging evidence that systemic and local inflammatory mediators can modulate neural excitability and sensory thresholds at peripheral and central levels, potentially increasing susceptibility to vestibular symptoms [18]. In parallel, the nasal mucosa is densely innervated by trigeminal afferents that project to brainstem and cortical networks shared with vestibular processing; persistent trigeminal input from chronic sinonasal inflammation has been proposed to modulate vestibular sensory gain through these connections [19,20]. Intranasal trigeminal function has been shown to be altered in CRS [21], providing a mechanistic anchor for this hypothesis.

This neuro-inflammatory framework parallels mechanisms proposed in functional vestibular disorders such as vestibular migraine and persistent postural-perceptual dizziness (PPPD), in which heightened sensory sensitivity and altered central processing, rather than structural vestibular damage, are thought to underlie symptom generation [22–24]. Similar centrally mediated mechanisms may contribute to vestibular complaints in a subset of patients with CRS and may help to explain the well-documented mismatch between patient-reported balance disturbances and normal findings on standard vestibular testing [25]. These parallels are intended as analogies for hypothesis generation, not as established equivalence between CRS-related dizziness and PPPD.

2.3. Altered Multisensory Integration

Postural stability depends on the central integration of vestibular, visual, and somatosensory inputs into a coherent estimate of self-motion. When one of these channels delivers persistently atypical or fluctuating information, central re-calibration mechanisms must compensate; when compensation is incomplete, subjective disequilibrium can emerge even without a measurable peripheral deficit. We use the term atypical sensory input in this sense, conceptually preferable to "aberrant", which carries a stronger pathological connotation than the available data support, to denote the chronically altered nasal, mucosal, trigeminal, and middle-ear afferent traffic that may accompany active CRS.

This conceptual model has direct empirical anchors. Convergence of nasal trigeminal and vestibular pathways has been described in anatomical and functional studies [19,20], and posturographic abnormalities have been reported in patients with chronic sinonasal disease and after endoscopic sinus surgery, suggesting that sinonasal afferent input contributes to postural control [11]. The model is consistent with the broader concept that central vestibular networks integrate inputs well beyond the labyrinth and that disorders of central integration can generate clinically meaningful imbalance with normal peripheral testing [26], but it remains a hypothesis: prospective studies linking CRS disease activity to objective measures of multisensory integration are not yet available.

3. Comparative Context: Vestibular Symptoms in Other Upper-Airway Conditions

Vestibular complaints are not unique to CRS. Situating CRS within the broader landscape of upper-airway inflammatory conditions helps to calibrate the strength of the proposed association and to clarify what, if anything, is distinctive about the CRS phenotype.

Allergic rhinitis (AR). A large population-based case–control study using the Taiwanese National Health Insurance database (78,503 cases of peripheral vestibular disorder and 235,509 propensity-matched controls) reported a higher prevalence of prior AR diagnosis among patients with peripheral vestibular disorder than among controls (32.2% vs. 22.8%; $p < 0.001$) [27]. An accompanying commentary highlighted the role of detection bias and the "sick-patient" effect in interpreting such associations [28], underscoring that observational evidence of an AR–vestibular link, although stronger numerically than the CRS evidence, also requires cautious interpretation. Mechanistically, AR shares with CRS several plausible routes to vestibular involvement, including ETD secondary to mucosal oedema, mast-cell-mediated inflammatory mediators reaching the inner ear, and migraine-related pathways [29].

Recurrent and post-viral upper respiratory tract infections. Acute viral upper respiratory tract infections are an established antecedent of vestibular neuritis and viral labyrinthitis, with a documented preceding or concurrent viral upper-airway illness in 43–46% of vestibular neuritis cases [30]. In contrast to AR and CRS, the post-viral picture is one of overt peripheral vestibulopathy with abnormal head-impulse and caloric testing, rather than nonspecific disequilibrium with predominantly normal vestibular tests. Recurrent upper respiratory infections may therefore contribute episodically to vestibular morbidity through a fundamentally different mechanism.

Taken together, these comparisons suggest that vestibular symptoms occur across a spectrum of upper-airway inflammatory conditions, with mechanisms ranging from clearly peripheral (post-viral neuritis) through mixed peripheral and central (AR) to predominantly functional and centrally mediated (the proposed CRS phenotype). What is plausibly distinctive about CRS is the chronicity of the inflammatory exposure and the predominance of normal vestibular testing despite persistent symptoms, a profile that aligns with functional rather than structural mechanisms, but that still requires direct empirical demonstration.

4. Clinical Recognition and Assessment of Vestibular Symptoms in CRS

From a clinical standpoint, the principal challenge is symptom recognition rather than diagnostic complexity. Routine CRS evaluation focuses on sinonasal complaints, whereas dizziness, imbalance, and reduced balance confidence are seldom explored in a structured manner. The dizziness item of the SNOT-22 captures the symptom only at the level of self-rated severity and contributes limited discriminant information in psychometric analyses [9], so its presence in routine practice does not substitute for direct clinical inquiry.

Simple, targeted questions about dizziness, instability during daily activities, or fear of imbalance may suffice to identify clinically relevant symptoms. This low-threshold inquiry is brief, can be incorporated into existing CRS visits without additional infrastructure, and may reveal a functional dimension of disease burden that severity scores alone do not capture.

4.1. Exclusion of Primary Vestibular or Neurological Disorders

When vestibular symptoms are reported, the clinician's first responsibility remains the exclusion of primary otologic or neurological pathology. Bedside examination and, when clinically indicated, targeted vestibular testing can assist in identifying peripheral or central vestibular disorders that require specific management [31], and timely referral to oto-laryngology or neuro-otology should be the default response when red-flag features

are present (sudden onset, focal neurological signs, hearing loss, severe rotational vertigo, or recurrent positional vertigo). Active inquiry about dizziness in CRS is not a substitute for this differential diagnosis: it is a prompt to perform it.

It is equally important to recognise that normal findings on conventional vestibular testing do not exclude clinically meaningful balance disturbances. Standard tools are designed primarily to detect structural or peripheral deficits and may fail to capture functional, intermittent, or centrally mediated disturbances; in the only published cohort study comparing videonystagmography (VNG) findings between patients with CRS and the general population, VNG patterns were largely comparable and approximately half of the CRS patients had normal VNG results [7]. A separate small prospective study reported abnormal caloric responses in CRSwNP but not in CRSsNP [13], suggesting possible phenotype-related differences that require confirmation in larger cohorts.

4.2. Interpreting Vestibular Symptoms Within the CRS Disease Context

Once primary vestibular pathology has been considered and addressed: vestibular complaints in CRS may be interpreted within the broader context of disease activity. Clinical observations and case-level reports describe parallel fluctuations of sinonasal and vestibular symptoms and improvement following effective control of inflammation [11,12], but the level of evidence is low and the direction of causality is not established. Recognising this potential association can support a more integrated, patient-centred approach and help avoid fragmented diagnostic pathways, without re-defining vestibular symptoms as a sinonasal disorder or expanding CRS disease boundaries.

5. Functional Consequences and Quality of Life

CRS is consistently associated with substantial impairment in health-related quality of life (HRQoL), and EPOS 2020 endorses validated questionnaires such as the SNOT-22 to quantify this burden [1,8]. The SNOT-22 "dizziness" item, although limited in psychometric discrimination [9], indicates that vestibular-type complaints are part of the symptom spectrum reported by rhinologic populations and contribute to perceived disease impact. Domain-level analyses confirm that dizziness loads on the otologic/facial-pain subdomain of the instrument [32].

5.1. Interpreting Functional Impact Without Overclaiming Causality

Although patients with CRS may attribute dizziness or imbalance to "sinus disease", available objective vestibular data do not currently establish a clear causal link between CRS disease activity and measurable vestibular dysfunction. Beyond Brody-Camp and colleagues' VNG study [7], few cohorts have systematically applied modern vestibular testing batteries to well-phenotyped CRS populations, and small observational studies have produced heterogeneous results [13]. The mismatch between symptom reporting and standard vestibular testing supports a cautious interpretation: vestibular complaints in CRS may be clinically relevant for quality of life even when conventional vestibular assessments are unremarkable, and the underlying pathophysiology remains uncertain.

5.2. Practical Clinical Message

From a pragmatic standpoint, the clinical priority is not to reclassify dizziness as a CRS manifestation, but to ensure that it is systematically elicited and appropriately framed during CRS assessment, alongside established CRS diagnostic and control criteria [1,33]. When dizziness is present, clinicians should avoid automatic attribution to CRS, perform a structured evaluation that considers alternative otologic and neurological explanations, and recognise that a normal vestibular test does not necessarily invalidate the patient's experience of disequilibrium.

6. Limitations of This Commentary

Several limitations of this Commentary should be acknowledged. First, the strength of the conclusions reflects the nature of the available evidence. The literature on vestibular symptoms in CRS is mainly composed of small observational studies, case reports, and hypothesis-generating reports, often with limited methodological robustness. Accordingly, the concepts presented in this manuscript should be interpreted as exploratory and hypothesis-driven rather than definitive. Second, this is not a systematic review. The literature was selected using a narrative approach, and therefore the possi-bility of selection bias cannot be excluded. Third, this work is based on a multidisciplinary clinical and research background, with limited direct specialisation in otolaryngology and vestibular disorders. While this perspective allows for a broader interpretation of the available evidence, it may not fully capture specialty-specific nuances that could be rele-vant to the topic. Future dedicated reviews or empirical studies would benefit from the involvement of otolaryngology and vestibular medicine specialists. Fourth, no original clinical data, registry datasets, or prospective patient cohorts are included. The clinical considerations regarding the routine assessment of vestibular symptoms in CRS are derived exclusively from interpretation of the published literature and expert opinion, and should therefore be regarded as preliminary and hypothesis-generating.

7. Conclusions

Vestibular symptoms are a clinically relevant but incompletely characterised component of the CRS symptom spectrum. Although CRS is traditionally regarded as a localised inflammatory disorder of the sinonasal mucosa, its impact frequently extends into functional and sensory domains that are captured only superficially by current assessment tools. A small body of observational evidence and several plausible mechanisms, Eustachian tube dysfunction, immune-mediated and neurogenic pathways, trigemino-vestibular interactions, and altered multisensory integration, suggest that dizziness, imbalance, and disequilibrium may parallel CRS disease activity in a subset of patients, but a causal relationship has not been demonstrated and the available data do not support reclassifying these complaints as a primary feature of CRS.

From a clinical perspective, vestibular symptoms in CRS represent a low-cost, high-yield domain of inquiry. We propose three pragmatic principles. First, ask: active, brief inquiry about dizziness and balance disturbances should become routine in CRS evaluation. Second, do not overlook: when dizziness is reported, primary otologic and neurological causes, including peripheral vestibular disorders, vestibular migraine, PPPD, and the post-viral and AR-related syndromes summarised above, must be considered and investigated, with referral to otolaryngology or neuro-otology where indi-cated. Third, interpret in context: once primary vestibular pathology has been excluded or addressed, optimising control of sinonasal inflammation is reasonable as part of the overall management plan, alongside vestibular care, rather than as a substitute for it.

Recognising vestibular dysfunction as a potential extra-sinonasal dimension of CRS does not redefine dizziness as a sinonasal disease; it broadens clinical awareness of the functional consequences of chronic upper-airway inflammation and identifies a clear need for adequately powered, multidisciplinary, prospective studies, ideally co-led with otolaryngology and neuro-otology, to clarify the prevalence, mechanisms, and treatment-responsiveness of vestibular complaints in this population.

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Abbreviations

The following abbreviations are used in this manuscript:

CRS	Chronic rhinosinusitis
EPOS	European Position Paper on Rhinosinusitis and Nasal Polyps
ETD	Eustachian tube dysfunction
HRQoL	Health-related quality of life
PPPD	Persistent postural-perceptual dizziness
SNOT-22	22-item Sinonasal Outcome Test
VNG	Videonystagmography

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