Original Article

Clinical Spectrum of Rhinogenic Contact Point Headache (RCPH): A Case Series with Diagnostic Challenges and Surgical Outcomes

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Abstract

Background: Rhinogenic contact point headache (RCPH) is a well-characterised yet frequently underdiagnosed entity arising from mucosal contact between opposing nasal structures. Its clinical manifestations often overlap with primary headache syndromes, rendering diagnosis challenging. This case series presents three anatomically and clinically distinct presentations of RCPH to highlight the spectrum of the condition and to reinforce the role of functional endoscopic sinus surgery (FESS) in its management.

Keywords: Rhinogenic contact point headache; Concha Bullosa; Sludger's neuralgia; Migraine; Functional Endoscopic sinus surgery; Sphenopalatine ganglion; Sinonasal anatomy.

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Introduction

Rhinogenic contact point headache (RCPH) represents a distinctive headache syndrome in which direct mucosal apposition between intranasal structures, most commonly the nasal septum, turbinates, or their anatomical variants generates neurally mediated facial pain. The syndrome was formally characterised by Stammberger and Wolf in 1988, who proposed that pressure- induced stimulation of trigeminal afferents at sinonasal contact points initiates a cascade of nociceptive and autonomic responses.[1]

The International Headache Society (IHS) classifies RCPH under headache attributed to disorder of the nose or paranasal sinuses (ICHD-3, 11.5.3), stipulating that pain must be ipsilateral to the contact point, resolve or significantly diminish following topical anaesthesia applied to the point of contact, and not fulfil criteria for any other headache disorder.[2] Crucially, the last criterion introduces a diagnostic dilemma when RCPH coexists with a primary headache disorder such as migraine.

Anatomical variants predisposing to RCPH include concha bullosa, paradoxical middle turbinate, deviated nasal septum, septal spurs, Haller cells, and hypertrophied inferior turbinates. The clinical presentation may resemble tension-type headache, cluster headache, or the poorly delineated entity of Sluder's neuralgia, a sphenopalatine ganglion-mediated pain syndrome. This series presents three patients managed at our institution, each representing a therapeutically and diagnostically distinct facet of RCPH. The cases are described with informed consent obtained from all patients and institutional ethical approval obtained for case series reporting.

Case 1: RCPH Associated with Bilateral Concha Bullosa

A 32-year-old man presented to the Department of Otorhinolaryngology with complaints of persistent nasal obstruction and recurrent frontal and periorbital headache for the past two years. The nasal obstruction had a gradual onset and progressively worsened over time, with symptoms being more pronounced on the left side than the right. He denied anosmia and experienced only partial symptomatic relief with the use of intranasal decongestant sprays. The headache was intermittent, ranging from moderate to severe intensity, and significantly affected his daily activities. There was no associated history of fever, purulent nasal discharge, facial trauma, visual complaints, nausea, vomiting, or focal neurological symptoms.

The patient had sought medical attention from several healthcare providers and had been treated with analgesics and antimigraine medications without significant improvement. Comprehensive neurological and ophthalmological evaluations were normal, excluding intracranial and ocular causes of headache.

Anterior rhinoscopic examination demonstrated a deviation of

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the nasal septum towards the left side. Diagnostic nasal endoscopy revealed bilateral concha bullosa involving the middle turbinates, with mucosal contact between the enlarged turbinates and the nasal septum [Figure 1,2]. No evidence of nasal polyps, mucopurulent secretions, or active sinonasal inflammation was observed.

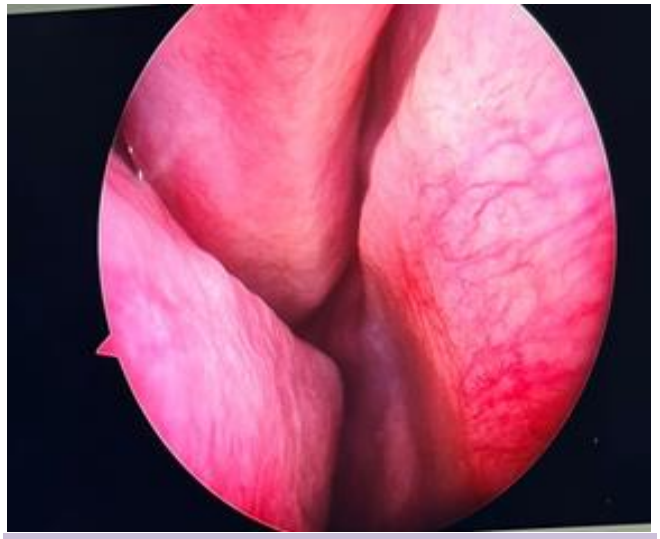

Figure 1: Right Nasal Cavity

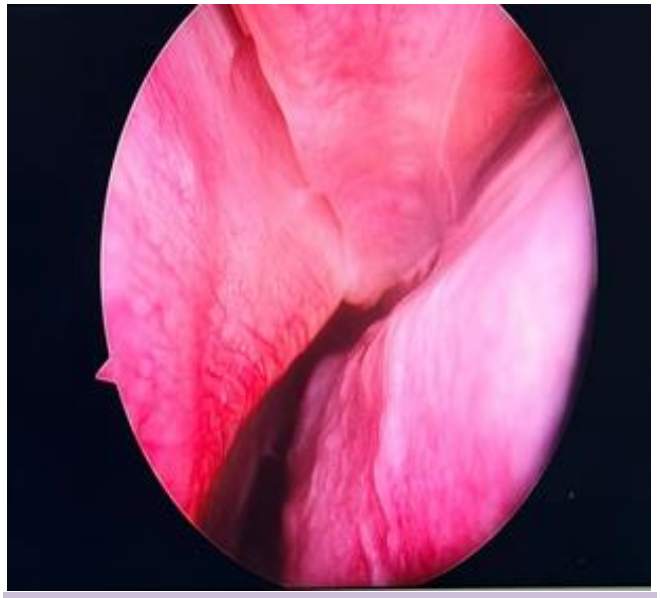

Figure 2: Left Nasal Cavity

Both Showing Septal Deviation and Contact Point

Computed tomography (CT) of the paranasal sinuses demonstrated bilateral concha bullosa of the middle turbinates along with an S-shaped deviated nasal septum associated with a left-sided bony septal spur [Figure 3]. The remaining paranasal sinuses were well aerated, with no radiological features suggestive of chronic rhinosinusitis. Correlation of the patient's clinical presentation with the endoscopic and radiological findings established the diagnosis of rhinogenic contact point headache secondary to bilateral concha bullosa and deviated nasal septum.

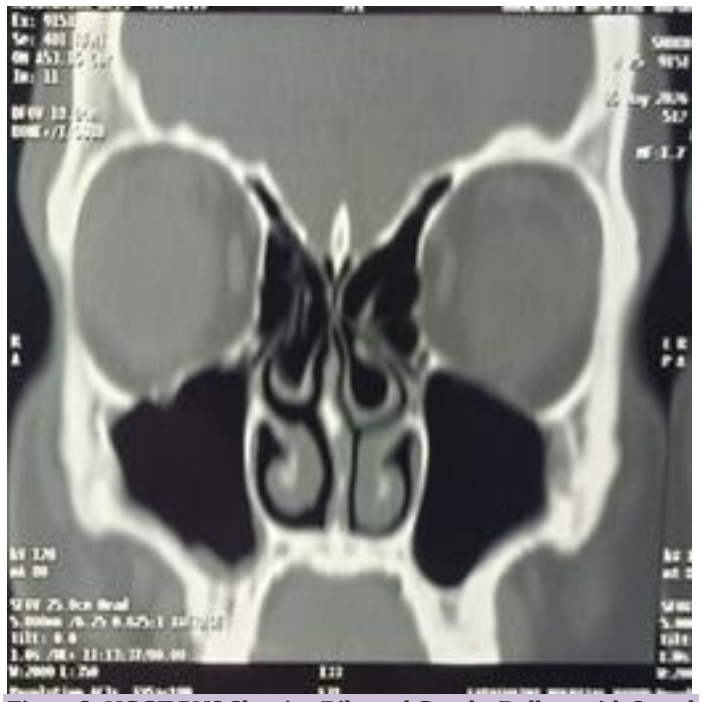

Figure 3: NCCT PNS Showing Bilateral Concha Bullosa with Septal Deviation

The patient underwent Endoscopic Septoplasty with Bilateral partial lateral resection of the concha bullosa. The postoperative recovery was uneventful. Follow-up evaluations at one, three, and six months demonstrated complete resolution of headache and a marked improvement in nasal breathing. No recurrence of symptoms was noted throughout the follow-up period. This case has been published as case report in journal of surgical radiology.[14]

Case 2: RCPH Presenting as Sluder's Neuralgia

A 18-year-old female was referred from neurology with an 18-month history of paroxysmal, unilateral right-sided nasal obstruction ,nasal discharge lacrimation and headache . The pain was localised to the right periorbital region, the root of the nose, and the right malar eminence. He described attacks as intense, burning-aching in character (NRS 8/10), lasting 20–90 minutes, and occurring two to three times per week, predominantly in the afternoon. There was no scalp tenderness, jaw claudication, or aural fullness.

A neurological diagnosis of Sluder's neuralgia (sphenopalatine ganglion neuralgia) had been entertained based on the autonomic accompaniments and the distribution of pain. Verapamil and sumatriptan had been initiated with only marginal improvement in attack frequency. The referring neurologist requested otolaryngological evaluation to exclude a structural sinonasal contributor.

Rigid nasal endoscopy was performed after thorough decongestion with oxymetazoline. On the right side, a prominent septal spur was identified at the junction of the perpendicular plate and the vomer, projecting laterally and making firm contact with the anteroinferior aspect of the right middle turbinate. The contact point appeared erythematous and mildly oedematous [Figure 4]. The left sinonasal cavity was unremarkable.

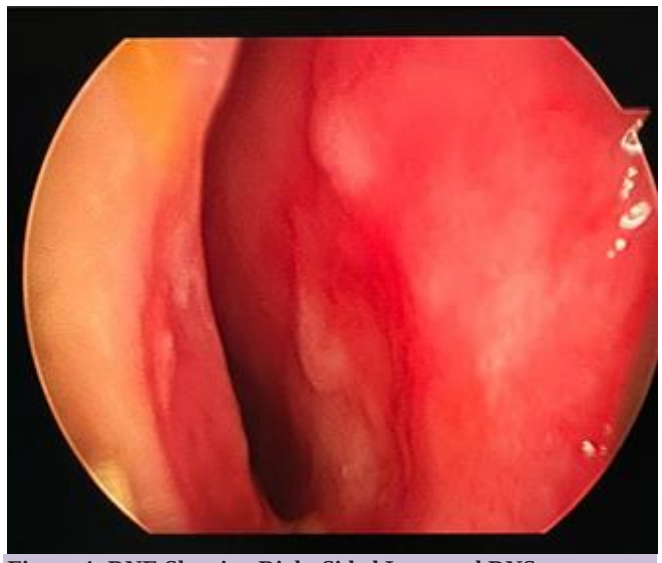

Figure 4: DNE Showing Right Sided Impacted DNS

NCCT of the paranasal sinuses confirmed a right-sided septal spur at the osseocartilaginous junction, indenting and displacing the right middle turbinate laterally [Figure 5]. There was no mucosal thickening of the paranasal sinuses, no concha bullosa, and no significant septal deviation proximal to the spur. MRI brain with gadolinium, performed by the neurology team prior to referral, was unremarkable, excluding secondary causes of facial pain including trigeminal neuroma and cavernous sinus pathology.

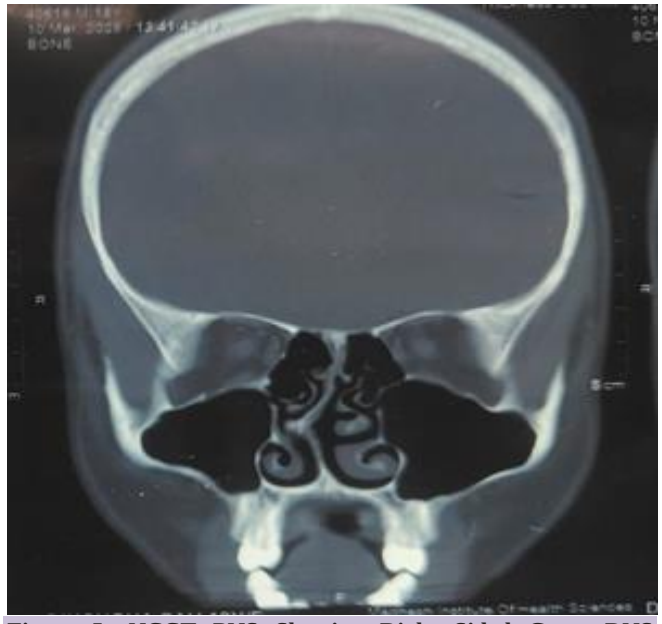

Figure 5: NCCT PNS Showing Right Sided Gross DNS Impinging On Right Lateral Wall

A sphenopalatine ganglion block using 4% lignocaine applied transnasally to the posterior middle meatus under endoscopic guidance was performed. The patient reported near-complete resolution of his ongoing facial discomfort within ten minutes (NRS 1/10). Importantly, a cotton pledget soaked in 4% lignocaine was also placed directly at the septal spur–middle turbinate contact point. This application alone, performed on a separate occasion, similarly reduced his pain by approximately eighty percent, confirming a mucosal contact mechanism as the primary driver of sphenopalatine ganglion irritation.

The patient underwent right endoscopic septoplasty with targeted resection of the septal spur under general anaesthesia. Endoscopic inspection confirmed complete release of the contact point [Figure 6]. No turbinate surgery was required as the middle turbinate returned to its natural position once the spur was excised.

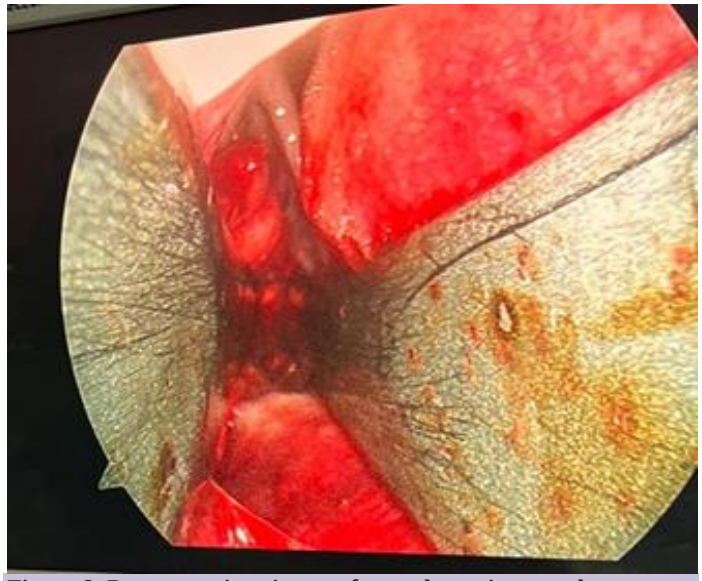

Figure 6: Post operative picture after endoscopic septoplasty

The patient was reviewed at four weeks, twelve weeks, and six months postoperatively. By four weeks, his attack frequency had reduced from two to three per week to one per fortnight. By twelve weeks, he reported complete freedom from the characteristic periorbital and facial pain. The autonomic accompaniments — lacrimation and rhinorrhoea — resolved in parallel, consistent with cessation of sphenopalatine ganglion irritation. Verapamil was tapered under neurological supervision and discontinued at three months without headache recurrence. At six months, endoscopy confirmed a smooth right nasal septum with a well-healed surgical site and no scarring or synaechiae.

Case 3: RCPH Coexisting with Migraine — Diagnostic Challenges And Surgical Outcome

A 57-year-old female with a diagnosis of migraine without aura presented for otolaryngological assessment following a two-year period of headache escalation refractory to optimised migraine pharmacotherapy. Her established migraine attacks — characterised by right-sided pulsatile hemicranial pain, photophobia, nausea, and phonophobia, typically lasting 12–48 hours — had previously been well controlled on topiramate and sumatriptan as an acute abortive agent.

However, she had begun experiencing a superimposed, daily right-sided facial pressure and perinasal ache (NRS 4–5/10), distinct in character from her migrainous pain, which she described as constant, non-pulsatile, lacking autonomic

accompaniments, and refractory to sumatriptan.

She was referred by her neurologist specifically to exclude a rhinogenic contributor to her increasingly complex headache burden. Rigid nasal endoscopy under decongestion demonstrated a right-sided paradoxical middle turbinate with its convexity directed medially, creating a broad mucosal contact zone with the nasal septum across the right middle meatus. A coincident mild rightward septal deviation at the junction of the cartilaginous and bony septum augmented the contact. The left nasal cavity was within normal limits. NCCT of the paranasal sinuses confirmed a right-sided septal spur at the bonycartilaginous junction, indenting and displacing the right middle turbinate laterally [Figure 7].

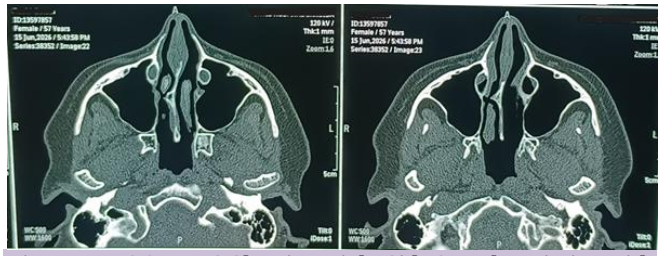

Figure 7: NCCT PNS Showing Right Side Septal Deviation with Contact Point

After multidisciplinary discussion with the treating neurologist, the decision was made to proceed with right endoscopic partial middle turbinectomy to correct the paradoxical turbinate configuration and relieve the contact point. The rationale was that surgical correction of the rhinogenic component carried low morbidity and offered the prospect of reducing the patients overall headache burden without interfering with ongoing migraine pharmacotherapy. Under general anaesthesia, the convex medial surface of the right middle turbinate was resected using straight through-cutting forceps and a microdebrider. The contact zone with the septum was fully released. Haemostasis was secured with bipolar diathermy, and a small absorbable haemostatic sponge was placed in the right middle meatus.

Postoperative follow-up at six weeks, three months, and six months demonstrated a stepwise improvement in the patient's headache profile. By six weeks, the daily right-sided facial pressure ,her rhinogenic headache had resolved entirely, confirmed by the patient's prospective headache diary which showed absence of the constant perinasal aching component. Her classic migrainous attacks continued at their baseline frequency and character, confirming that the surgical intervention had not altered the primary headache disorder.

By three months, however, the patient and her neurologist noted a twenty-five percent reduction in the frequency of migraine attacks (from an average of eight to six per month). This secondary reduction was hypothesised to reflect reduced peripheral trigeminal sensitisation from the eliminated contact point, which had previously contributed to a lower migraine threshold through convergent trigeminal activation. At six months, endoscopy confirmed a patent right middle meatus with well-healed turbinate remnants.

Discussion

The three cases presented in this series collectively illustrate the breadth of clinical phenotypes encompassed by RCPH and highlight three recurring challenges in its diagnosis and management: (i) the attribution error arising from pre-existing or concurrent neurological diagnoses; (ii) the mimicry of autonomic cephalalgias — specifically Sluder's neuralgia — by posteriorly located rhinogenic contact points irritating the sphenopalatine ganglion; and (iii) the mechanistic interaction between peripheral sinonasal sensitisation and central migraine susceptibility.

Concha bullosa is among the most frequently encountered anatomical variations of the sinonasal cavity. Although it is commonly detected as an incidental finding on computed tomography and remains asymptomatic in most individuals, excessive pneumatization of the middle turbinate may narrow the nasal passage and create mucosal contact with adjacent structures. The coexistence of bilateral concha bullosa with a deviated nasal septum further increases the likelihood of contact point formation, which has been implicated as a potential source of rhinogenic contact point headache.[3,4]

The diagnosis of rhinogenic contact point headache should be established only after a comprehensive evaluation and exclusion of other primary and secondary causes of headache. Disorders such as migraine, tension-type headache, cluster headache, trigeminal neuralgia, temporomandibular joint dysfunction, dental pathology, and intracranial lesions should be carefully considered during the diagnostic workup. In the present case, the patient's persistent symptoms despite prolonged treatment with analgesics and antimigraine medications, together with normal neurological and ophthalmological examinations, prompted a detailed otorhinolaryngological assessment that identified the underlying anatomical abnormalities.

Diagnostic nasal endoscopy remains the cornerstone for identifying mucosal contact points, allowing direct visualization of the relationship between the nasal septum and adjacent intranasal structures.[13,15] Computed tomography of the paranasal sinuses complements endoscopic evaluation by accurately defining the anatomical variations, determining the extent of septal deviation, and excluding coexisting inflammatory sinonasal disease. In this patient, the close correlation between clinical symptoms, endoscopic findings, and radiological evidence strongly supported the diagnosis of rhinogenic contact point headache.

The topical nasal anesthetic (lidocaine) test has also been described as a useful adjunctive investigation, particularly in equivocal cases, as temporary relief of headache following application of a local anesthetic to the contact area further supports the diagnosis.

The primary objective of surgical management is to eliminate the pathological mucosal contact while restoring normal intranasal anatomy and airflow. Endoscopic surgery has become the preferred treatment modality because it offers superior visualization, greater surgical precision, and lower morbidity compared with traditional approaches. Depending on the anatomical abnormality, procedures may include septoplasty, conchoplasty, turbinoplasty, or partial resection of the middle turbinate. Several studies have demonstrated significant reductions in headache intensity and frequency following

surgical correction, accompanied by improved patient-reported quality of life.[5-7]

Sluder's neuralgia, first described by Greenfield Sluder in 1908, is characterised by unilateral facial pain in the distribution of the sphenopalatine ganglion (SPG), accompanied by autonomic features including lacrimation, rhinorrhoea, and nasal congestion.[8] The nosological status of Sluder's neuralgia remains contentious; it is not formally included in the current ICHD-3 classification as a distinct entity and is considered by many authorities to overlap with cluster headache and paroxysmal hemicranias.[9]

The structural sinonasal pathology — specifically, a posterior septal spur creating contact with the middle turbinate in close proximity to the sphenopalatine foramen — may generate repetitive irritation of the SPG through direct pressure on its overlying mucosa. The SPG lies in the pterygopalatine fossa, separated from the nasal mucosa only by a thin layer of connective tissue, rendering it anatomically susceptible to stimulation from posterosuperior nasal contact points.

Any patient presenting with a clinical picture consistent with Sluder's neuralgia or SPG- mediated facial pain should undergo thorough sinonasal examination prior to accepting a primary neurological diagnosis.[10] Structural aetiology, when identified and corrected, offers a surgical cure rather than lifelong pharmacological suppression.

The coexistence of RCPH and migraine is not merely a diagnostic curiosity; it has direct therapeutic implications. Several lines of evidence support a mechanistic interaction between sinonasal contact point pain and migraine susceptibility. The trigeminal nucleus caudalis, which processes nociceptive input from both the meninges (migraine pathway) and the sinonasal mucosa (rhinogenic pathway), exhibits convergence of these afferents. Peripheral sensitisation from repetitive sinonasal contact point stimulation may reduce the threshold for central trigeminovascular activation, effectively lowering the migraine attack threshold. The coexistence of established migraine with a suspected rhinogenic contact point headache in this patient presented several well-recognised diagnostic pitfalls that merit detailed discussion.

ICHD-3 criteria for RCPH require that the headache does not better fit the criteria of another disorder. In a patient with confirmed migraine, every episode of headache, including those with a predominantly pressure-like, nasal quality may be ascribed to the migraine diagnosis by default. This diagnostic anchoring risks obscuring a concurrent treatable sinonasal pathology. The discriminating features in this case were the distinct pain character, absence of migrainous features during the daily facial pressure episodes, and localisation to the right perinasal region consistent with the right-sided contact point.

Rhinogenic Nasal Symptoms as Migraine Premonitory Features

Nasal congestion and facial pressure are recognised premonitory and allodynic features of migraine mediated by trigeminovascular activation and central sensitisation. Distinguishing these migraine-attributable nasal symptoms from those directly caused by sinonasal mucosal contact requires careful history-taking: rhinogenic symptoms typically persist interictally, are present in the absence of any headache phase, and respond to topical decongestants or nasal endoscopic examination-precipitated manipulation.

Anaesthetic Testing in the Context of Migraine

Application of 4% topical lignocaine to the right paradoxical turbinate contact zone was performed on two occasions outside of migraine attack windows, verified by the patient's headache diary. On both occasions, the patient reported resolution of her daily facial pressure component within fifteen to twenty minutes (NRS 0–1/10), without any alteration in the frequency or character of her classic migrainous attacks occurring in subsequent weeks. This selective response confirmed that the two headache phenotypes were mechanistically dissociable, validating the dual diagnosis.

Prior surgical series of migraine patients with intranasal contact points have similarly reported a reduction in migraine frequency following correction of the contact point, a finding echoed by our outcome.[11,12] This interaction implies that in migraine patients with inadequate pharmacological control, a meticulous rhinoscopic evaluation to exclude a concurrent RCPH is clinically warranted rather than attributing all headache burden to the primary diagnosis.

The diagnostic algorithm we recommend for suspected dual-diagnosis cases includes: (1) characterisation of headache phenotypes through prospective diary, (2) rigid nasal endoscopy and HRCT paranasal sinuses, (3) anaesthetic testing performed outside migraine attack windows, (4) correlation of anaesthetic response with the non-migrainous headache component specifically, and (5) multidisciplinary consensus before surgical intervention.

Common to all three cases was the pivotal diagnostic role of topical intranasal anaesthesia. Application of 4% lignocaine to the relevant contact point, with quantitative documentation of the pain response using a numerical rating scale, provided the functional confirmation required by both ICHD-3 criteria and clinical logic. We advocate for its systematic use in any patient presenting with facial pain or headache in whom sinonasal examination reveals a structural contact point. The test is inexpensive, well-tolerated, and carries a high degree of diagnostic specificity when performed under endoscopic guidance.

Computed tomography of the paranasal sinuses served as the primary imaging modality across all three cases, providing the anatomical detail required for preoperative surgical planning. MRI was reserved for where the autonomic pain phenotype necessitated exclusion of intracranial and cavernous sinus pathology. It is important to note that CT-identified anatomical variants do not in themselves confirm RCPH; the clinical and functional correlation — mucosal contact on endoscopy, and pain response to anaesthesia — remains indispensable.

The surgical strategies employed across the three cases — bilateral concha bullosa resection, endoscopic septoplasty with spur excision, and paradoxical turbinate partial resection — each represent targeted, minimally invasive endoscopic techniques with well-documented safety profiles. The principle governing surgical selection was not anatomical correction per se, but the elimination of the specific contact point confirmed as the pain generator. This targeted philosophy minimises operative

morbidity and preserves nasal physiological function.

From a medicolegal and ethical perspective, all three patients provided informed consent

inclusive of a clear explanation that surgery was being performed for headache — an indication that required explicit discussion, as patients and primary care clinicians may associate nasal surgery exclusively with obstruction or sinus disease. Preoperative documentation of the diagnostic anaesthetic response provided an objective basis for the surgical indication.

Conclusion

Rhinogenic contact point headache encompasses a clinically diverse range of presentations, from the straightforward bilateral anatomical variant to the neuralgiform mimicry and the diagnostically complex dual-diagnosis scenario. Accurate diagnosis hinges upon a synthesis of detailed headache characterisation, meticulous rigid nasal endoscopy, corroborative CT imaging, and objective anaesthetic testing. Surgical correction of the relevant contact point — guided by these criteria — provides durable and often complete headache relief, including secondary reduction of migraine frequency in susceptible individuals.

These cases emphasise that RCPH should be considered in the differential diagnosis of any patient presenting with refractory facial pain or headache, irrespective of a prior neurological label. Collaboration between otolaryngologists and neurologists is integral to the management of complex or dual-diagnosis cases and should be embedded in institutional headache referral pathways.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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