

Primary Atrophic Rhinitis in Three Adults: A Case Series

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Abstract

Background: Primary atrophic rhinitis, or ozaena, is an uncommon chronic inflammatory-degenerative disorder of the nasal cavity. It is characterized by mucosal and turbinate atrophy, widened nasal cavities, adherent crusts, fetor, and variable olfactory dysfunction. Diagnosis is mainly clinical and is strengthened by nasal endoscopy, microbiological assessment, and computed tomography of the paranasal sinuses in selected patients. **Case presentation:** Three adults presented with long-standing bilateral nasal obstruction and reduced smell perception. Recurrent crust expulsion was present in two patients. Endoscopic findings included dry pale mucosa, roomy nasal cavities, inferior turbinate atrophy, adherent crusts, and fetor. One patient had a small anterior septal perforation. Nasal swab culture isolated *Klebsiella ozaenae* in two cases, while the third case showed mixed commensal nasal flora without *Klebsiella ozaenae* growth. Computed tomography supported the presence of widened nasal cavities and turbinate volume loss without destructive sinonasal disease. **Management and outcome:** All patients were managed conservatively with endoscopic crust removal, regular alkaline nasal douching, saline irrigation, and topical lubrication. Culture-positive cases received sensitivity-guided oral ciprofloxacin and topical mupirocin. Follow-up showed reduction in fetor, crust burden, mucosal dryness, and nasal obstruction. Hyposmia persisted mildly in two patients despite clinical improvement. **Conclusion:** Primary atrophic rhinitis remains a clinically recognizable but underreported nasal disorder. Complete reporting requires symptom duration, nasal endoscopic findings, microbiological status, imaging findings when available, treatment details, and follow-up response.

Keywords: Atrophic rhinitis; Ozaena; Nasal crusting; Merciful anosmia; *Klebsiella ozaenae*; Nasal endoscopy; Case series.

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INTRODUCTION

Atrophic rhinitis is a chronic nasal disorder in which progressive mucosal, glandular, vascular, neural, and turbinate atrophy produces a paradoxical clinical picture. Patients commonly complain of nasal obstruction even though the nasal cavities are abnormally wide. This apparent contradiction is explained by crusting, mucosal dryness, impaired mucociliary clearance, altered airflow sensation, and secondary bacterial colonization.^[1-3]

Primary atrophic rhinitis has been associated with chronic bacterial colonization, particularly *Klebsiella ozaenae*, nutritional deficiency, endocrine influence, hereditary susceptibility, autonomic dysfunction, and environmental factors. Secondary atrophic rhinitis may follow extensive sinonasal surgery, radiotherapy, trauma, granulomatous infection, autoimmune disease, or chronic destructive inflammation.^[2-4] Distinguishing primary from secondary disease is important because the diagnostic evaluation and long-term management differ.

The classical clinical profile includes roomy nasal cavities, dry pale mucosa, atrophic inferior and middle turbinates, thick foul-smelling crusts, cacosmia perceived by others, hyposmia or merciful anosmia, and bleeding after crust removal. Septal perforation and external nasal deformity are not universal; however, their presence requires careful documentation and exclusion of destructive inflammatory,

infective, traumatic, or neoplastic causes.^[4-6]

Although the diagnosis is largely clinical, nasal swab culture and sensitivity are useful when fetor, purulent discharge, recurrent crusting, or active infection is present. *Klebsiella ozaenae* isolation supports the microbiological diagnosis and can guide antimicrobial therapy.^[4,7] Computed tomography of the paranasal sinuses is valuable in severe, recurrent, atypical, perforating, or diagnostically uncertain cases because it documents turbinate atrophy and excludes bony destruction, granulomatous disease, invasive fungal disease, and sinonasal malignancy.^[4,11]

Management is usually prolonged and symptom-directed. Conservative measures include repeated crust removal, alkaline nasal douching, saline irrigation, topical emollients, and topical or systemic antibiotics when clinically indicated. Surgical procedures such as cavity-narrowing techniques or nostril-closure procedures are reserved for refractory disease with

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persistent crusting, severe fetor, and impaired quality of life despite adequate conservative treatment.^[8,9,12-14] This case series describes the clinical profile, diagnostic evaluation, treatment, and short-term outcomes of three adults with primary atrophic rhinitis.

MATERIALS AND METHODS

Study design and setting: This descriptive case series includes three adult patients diagnosed with primary atrophic rhinitis in the otorhinolaryngology outpatient setting of a tertiary care teaching hospital of KIMS, Narketpally, Telangana, India. The study follows standard case-series reporting principles and incorporates key CARE-style elements, including patient information, diagnostic assessment, therapeutic intervention, and follow-up.^[1]

Diagnostic approach: Primary atrophic rhinitis was diagnosed on the basis of chronic bilateral nasal obstruction with reduced smell perception or anosmia, recurrent nasal crusting or dryness, and characteristic anterior rhinoscopy or nasal endoscopy findings. These findings included roomy nasal cavities, pale dry mucosa, turbinate atrophy, adherent crusts, and fetor. Secondary causes were assessed clinically through history, examination, microbiology, and imaging where indicated.

Investigations and treatment: All patients underwent anterior rhinoscopy and diagnostic nasal endoscopy. Nasal swab culture and sensitivity were performed from crusted or mucoid material. Computed tomography of the paranasal sinuses was used to document turbinate atrophy, nasal cavity widening, sinus involvement, septal defect, and absence of destructive sinonasal disease. Treatment was individualized and included endoscopic debridement, alkaline nasal douching, saline irrigation, topical lubrication, topical antibiotics when indicated, and culture-guided systemic antibiotic therapy in culture-positive cases.

Outcome assessment: Follow-up response was assessed clinically using reduction in crust burden, fetor, dryness, nasal obstruction, ease of crust removal, endoscopic appearance, olfactory symptoms, and stability of septal perforation when present.

Case Presentations

Case 1

A 45-year-old woman presented with decreased perception of smell, bilateral nasal obstruction, and recurrent expulsion of crusts from both nasal cavities for four years. She also reported right-sided mucoid ear discharge for six months. There was no history of excessive nasal surgery, nasal trauma, radiation exposure, corrosive exposure, or known granulomatous disease.

Anterior rhinoscopy and diagnostic nasal endoscopy showed bilateral roomy nasal cavities filled with thick greenish-brown adherent crusts. The nasal mucosa was dry, pale, and insensitive over crusted areas. The inferior turbinates were markedly atrophic, and the middle turbinates were mildly atrophic. Fetor was clearly present and noticed by the examiner. Minimal contact bleeding occurred after removal of adherent crusts. No septal perforation, saddle-nose deformity, mass lesion, or active epistaxis was seen.

Nasal swab culture from crusted discharge showed heavy growth of *Klebsiella ozaenae*. The isolate was sensitive to ciprofloxacin, ceftriaxone, gentamicin, and amikacin, and resistant to ampicillin. Fungal elements were not detected on routine testing. Computed tomography of the paranasal sinuses showed bilateral widening of the nasal cavities, reduced inferior turbinate volume, crusting along the nasal floor and lateral nasal wall, mild bilateral maxillary sinus mucosal thickening, and no bony destruction or sinonasal mass.

The patient underwent outpatient endoscopic crust removal and nasal toileting. She was advised alkaline nasal douching three times daily, isotonic saline irrigation, and topical liquid paraffin or glycerine-based nasal lubricant twice daily. Topical mupirocin was applied over crusted vestibular areas for seven days. Based on culture sensitivity, oral ciprofloxacin 500 mg twice daily was prescribed for 10 days. The right ear discharge was managed with aural toilet and appropriate topical therapy after otoscopic evaluation.

At two-week review, fetor and crust burden had reduced. At six weeks, nasal obstruction improved and crust removal became easier. At three months, she continued regular nasal douching with persistent but milder hyposmia. No septal perforation or external nasal deformity was observed during follow-up.

Case 2

A 28-year-old woman presented with decreased perception of smell and bilateral nasal obstruction for two years. She had mild recurrent dryness and anterior nasal crusting. There was no history of previous nasal surgery, trauma, radiotherapy, systemic granulomatous disease, or destructive nasal symptoms.

Anterior rhinoscopy and nasal endoscopy showed dry pale mucosa, moderate widening of both nasal cavities, and mild-to-moderate inferior turbinate atrophy. Small dry crusts were present over the anterior nasal septum and floor of both nasal cavities. Fetor was mild. The middle turbinates were visualized and were not grossly destroyed. The nasal septum was intact, and no septal perforation, granulation tissue, mass, or saddle-nose deformity was noted.

Nasal swab culture showed mixed commensal nasal flora without *Klebsiella ozaenae* growth. Computed tomography of the paranasal sinuses showed mild bilateral nasal cavity widening with reduced inferior turbinate bulk, without significant sinus opacification, bony erosion, septal perforation, or sinonasal mass.

The patient underwent gentle debridement of anterior crusts and was advised alkaline nasal douching three times daily for six weeks. She also received topical saline gel or glycerine-based lubricant twice daily and counselling regarding long-term nasal hygiene. Systemic antibiotics were not used because *Klebsiella ozaenae* was not isolated and purulent discharge was absent.

At four-week follow-up, she reported reduced nasal obstruction and dryness. Endoscopy showed fewer crusts and better mucosal hydration. At three months, she remained on maintenance saline irrigation once or twice daily. Smell perception improved slightly but did not return completely to normal.

Case 3

A 34-year-old man presented with bilateral nasal obstruction, decreased perception of smell, and recurrent expulsion of crusts from both nasal cavities for three years. There was no history of previous turbinectomy, radiotherapy, cocaine exposure, palatal

involvement, or known systemic granulomatous disease. Anterior rhinoscopy and nasal endoscopy showed extensive bilateral foul-smelling greenish-brown crusts occupying the floor, septum, and lateral nasal wall. After crust removal, both nasal cavities appeared markedly roomy. The mucosa was dry, pale, and friable, with contact bleeding at multiple crusted points. Inferior turbinate atrophy was severe bilaterally, and middle turbinate atrophy was moderate. Fetor was marked. A small anterior septal perforation measuring approximately 4 × 3 mm with crusted margins was present. There was no saddle-nose deformity, visible mass, or palatal involvement.

Nasal swab culture from crusted material showed *Klebsiella ozaenae* with mixed secondary bacterial flora. The *Klebsiella* isolate was sensitive to ciprofloxacin, ceftriaxone, amikacin, and gentamicin, and resistant to ampicillin. Computed tomography of the paranasal sinuses showed bilateral roomy nasal cavities, marked inferior turbinate volume loss, patchy

mucosal thickening in the bilateral maxillary and anterior ethmoid sinuses, crust-density material within the nasal cavities, and a small anterior septal defect. No destructive bony erosion, sinonasal mass, orbital extension, or skull-base involvement was present.

Treatment consisted of weekly outpatient endoscopic debridement for four weeks, alkaline nasal douching three times daily, saline irrigation, topical mupirocin for 10 days, and regular liquid paraffin or saline gel application to reduce crust adherence. Based on culture sensitivity, oral ciprofloxacin 500 mg twice daily was prescribed for 14 days. Surgery was reserved as a future option only if conservative treatment failed.

At one month, fetor reduced substantially and crusting became less extensive. At three months, nasal obstruction improved, but mild crusting persisted and required maintenance douching. The septal perforation remained stable without enlargement, and no external nasal deformity developed.

Table 1: Demographic, symptom, and nasal examination profile of the three cases

Variable	Case 1	Case 2	Case 3
Age/sex	45-year-old female	28-year-old female	34-year-old male
Main symptoms	Hyposmia, bilateral obstruction, recurrent crust expulsion, and right mucoid ear discharge	Hyposmia, bilateral obstruction, mild dryness, and anterior crusting	Bilateral obstruction, hyposmia, and recurrent crust expulsion
Duration	Nasal symptoms for 4 years; right ear discharge for 6 months	2 years	3 years
Nasal cavity	Bilateral roomy cavities	Moderately roomy cavities	Markedly roomy cavities
Crusts	Thick greenish-brown adherent crusts bilaterally	Small dry crusts over anterior septum and nasal floor	Extensive foul-smelling greenish-brown crusts bilaterally
Mucosa and turbinates	Dry pale mucosa; marked inferior turbinate atrophy; mild middle turbinate atrophy	Dry pale mucosa; mild-to-moderate inferior turbinate atrophy	Dry friable mucosa; severe inferior and moderate middle turbinate atrophy
Fetor	Present	Mild	Marked
Septal perforation	Absent	Absent	Small anterior perforation, approximately 4 × 3 mm
Other findings	No saddle-nose deformity, mass, or active epistaxis	No granulation tissue, mass, perforation, or saddle-nose deformity	No saddle-nose deformity, mass, palatal involvement, or skull-base/orbital extension

Table 2: Investigation profile including nasal culture and computed tomography findings

Variable	Case 1	Case 2	Case 3
Nasal swab culture	Heavy growth of <i>Klebsiella ozaenae</i>	Mixed commensal nasal flora; <i>Klebsiella ozaenae</i> not isolated	<i>Klebsiella ozaenae</i> with mixed secondary bacterial flora
Antibiotic sensitivity	Sensitive to ciprofloxacin, ceftriaxone, gentamicin, and amikacin; resistant to ampicillin	Systemic antibiotic sensitivity not applicable because <i>Klebsiella ozaenae</i> was not isolated	Sensitive to ciprofloxacin, ceftriaxone, amikacin, and gentamicin; resistant to ampicillin
CT paranasal sinuses	Roomy nasal cavities, inferior turbinate atrophy, crusting, mild maxillary mucosal thickening, and no bony destruction	Mild roomy nasal cavities with reduced inferior turbinate bulk; no significant sinus disease, bony erosion, septal defect, or mass	Roomy cavities, turbinate atrophy, maxillary/anterior ethmoid mucosal thickening, small anterior septal defect, and no destructive lesion
Additional evaluation	No fungal elements detected on routine testing; otologic evaluation performed for right ear discharge	No clinical evidence of destructive granulomatous or neoplastic disease	No CT evidence of destructive bony erosion, sinonasal mass, orbital extension, or skull-base involvement

Table 3: Therapeutic intervention and follow-up response

Case	Initial treatment	Antibiotic/topical therapy	Follow-up and outcome
Case 1	Outpatient endoscopic debridement, nasal toileting, alkaline nasal douche three times daily, saline irrigation, and topical lubricant	Culture-guided oral ciprofloxacin for 10 days; topical mupirocin for vestibular crusting; topical therapy for ear discharge after otoscopy	Reviewed at 2 weeks, 6 weeks, and 3 months. Fetor and crusting reduced, obstruction improved, mild hyposmia persisted, and no septal perforation developed.
Case 2	Gentle crust removal, alkaline nasal douche, saline gel or glycerine-based lubricant, and counselling for long-term nasal hygiene	No systemic antibiotic because <i>Klebsiella ozaenae</i> was not isolated and purulence was absent	Reviewed at 4 weeks and 3 months. Dryness and obstruction improved, crusts reduced, mucosal hydration improved, and olfactory recovery was partial.

Case 3	Weekly debridement for 4 weeks, alkaline nasal douche three times daily, saline irrigation, and topical emollient	Culture-guided oral ciprofloxacin for 14 days; topical mupirocin for 10 days	Reviewed at 1 month and 3 months. Fetor and crusting reduced markedly, obstruction improved, mild residual crusting persisted, and septal perforation remained stable.
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Representative nasal endoscopic images demonstrating the characteristic mucosal dryness, roomy nasal cavities, crusting, and turbinate atrophy are presented in [Figure 1-3].

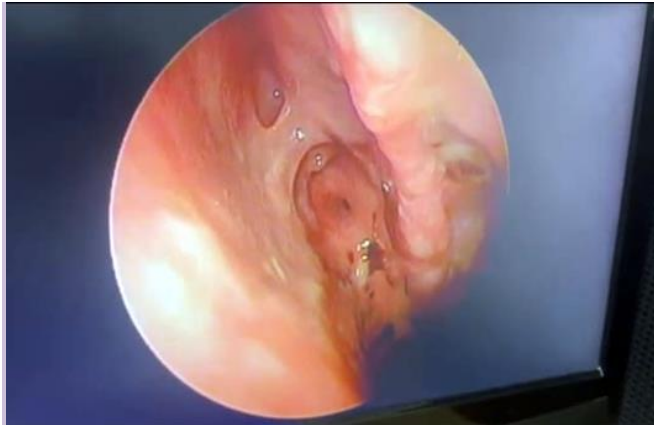


Figure 1. Diagnostic nasal endoscopic image showing a roomy nasal cavity with dry pale mucosa, mucosal atrophy, and adherent crusting, consistent with primary atrophic rhinitis.



Figure 2. Nasal endoscopic view demonstrating widened nasal cavity with crusting over the septal and lateral nasal wall regions, along with mucosal dryness and turbinate atrophy.



Figure 3. Nasal endoscopic image showing extensive greenish-brown crusting, friable mucosa, and marked turbinate atrophy in a severe presentation of primary atrophic rhinitis.

Diagnostic Assessment

The diagnosis of primary atrophic rhinitis was supported by chronic bilateral nasal obstruction, reduced smell perception, nasal crusting, widened nasal cavities, mucosal dryness, turbinate atrophy, and fetor. Culture positivity for *Klebsiella ozaenae* strengthened the diagnosis in Cases 1 and 3 but was not mandatory for diagnosis because Case 2 had classical clinical and endoscopic features despite culture-negative findings.^[4,7]

Cases 1 and 3 represented culture-positive disease with prominent fetor and crusting. Case 2 represented a milder culture-negative phenotype. Computed tomography supported the diagnosis by demonstrating nasal cavity widening and turbinate volume loss while excluding destructive sinonasal disease, granulomatous inflammation, invasive fungal disease, and neoplasm.^[4,11]

Differential Diagnosis: The differential diagnosis included chronic rhinosinusitis with crusting, rhinoscleroma, leprosy, tuberculosis, syphilis, invasive fungal disease, granulomatosis with polyangiitis, sarcoidosis, sinonasal malignancy, cocaine-related midline destructive lesion, radiation injury, and secondary atrophic rhinitis after excessive turbinate surgery. The absence of destructive bony erosion, sinonasal mass, palatal involvement, orbital extension, and rapidly progressive septal destruction supported primary atrophic rhinitis in these cases.

DISCUSSION

This case series highlights the core clinical pattern of primary atrophic rhinitis in adults: chronic bilateral nasal obstruction with reduced smell perception, recurrent crusting, widened nasal cavities, turbinate atrophy, and fetor. The finding of obstruction despite a roomy nasal cavity reflects altered airflow sensation, mucosal dryness, crust load, sensory impairment, and impaired mucociliary clearance rather than true anatomical narrowing.^[2-5]

Nasal endoscopy remains central to diagnosis. The combination of roomy nasal cavities, dry pale mucosa, inferior turbinate atrophy, adherent crusts, and fetor provides stronger diagnostic evidence than symptoms alone. Documentation of septal perforation is clinically important because a stable small perforation can occur with chronic crusting, while progressive perforation should prompt evaluation for vasculitis, infection, drug-related injury, malignancy, or other destructive causes.^[4-6]

Microbiology has practical value because *Klebsiella ozaenae* has a recognized association with ozaena. Culture results help guide antibiotic therapy in patients with marked fetor, purulent discharge, recurrent crusting, or active secondary infection. In the present series, two patients had culture-positive disease and received sensitivity-guided ciprofloxacin. The culture-negative case improved without systemic antibiotics, supporting a restrained and clinically guided approach to antimicrobial use.^[4,7] Computed tomography is not required for every mild classical case, but it is useful when disease is severe, recurrent, perforating, or diagnostically uncertain. Imaging can demonstrate widened nasal spaces, turbinate volume loss, crust-density material, sinus mucosal thickening, and absence of

aggressive bony destruction. In this series, CT-PNS helped document disease extent and excluded destructive sinonasal pathology in all three patients.^[4,11]

Conservative treatment remains the foundation of management. Regular alkaline nasal douching, saline irrigation, debridement, and topical lubrication reduce crust adherence, improve mucosal hydration, and decrease fetor. Antibiotics are best reserved for culture-positive or clinically infected cases and should follow local antimicrobial policy. Surgical options, including modified Young procedures or other cavity-narrowing techniques, are considered for refractory disease with persistent fetor, crusting, and impaired quality of life despite adequate conservative therapy.^[8,9,12-14]

The outcomes in this series were consistent with the chronic nature of the disease. Fetor, crusting, dryness, and obstruction improved with conservative therapy, but olfactory recovery was incomplete in two patients. This pattern is clinically relevant because patients require counselling that long-term nasal hygiene is essential and that smell perception may recover slowly or remain partially impaired even after crusting and fetor improve.^[10]

Learning Points

- Primary atrophic rhinitis should be suspected when chronic bilateral nasal obstruction coexists with reduced smell perception and recurrent nasal crusting.
- Roomy nasal cavities, turbinate atrophy, dry pale mucosa, adherent crusts, and fetor should be documented clearly in every case.
- Nasal swab culture and sensitivity, especially *Klebsiella ozaenae* status, help guide antibiotic therapy when infection is suspected.
- Computed tomography of the paranasal sinuses is useful in severe, recurrent, perforating, atypical, or diagnostically uncertain cases.
- Treatment response should be reported using symptom improvement and repeat endoscopic findings at defined follow-up points.

CONCLUSION

Primary atrophic rhinitis remains a clinically recognizable but underreported chronic nasal disorder. In this three-case series, the diagnosis was supported by bilateral nasal obstruction, reduced smell perception, recurrent crusting, roomy nasal cavities, mucosal dryness, turbinate atrophy, and fetor. Culture-guided antibiotics were useful in *Klebsiella ozaenae*-positive cases, while conservative therapy with debridement, alkaline douching, saline irrigation, and lubrication improved symptoms across all cases. Complete case-series reporting should include symptom duration, endoscopic findings, culture results, imaging when performed, treatment details, and follow-up response.

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Conflicts of interest

There are no conflicts of interest.

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