

# Leukaemia Presenting with Nasal Septal Abscess: A Case Report

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## ABSTRACT

Nasal Septal Abscess (NSA) is a rare but serious condition that typically arises from trauma, infections, or surgical interventions. However, in immunocompromised patients, NSA may develop spontaneously due to underlying haematological conditions. A 28-year-old male with recently diagnosed pre-B-cell Acute Lymphoblastic Leukaemia (ALL) presented with bilateral nasal obstruction and respiratory discomfort. Examination and imaging confirmed an NSA with cartilage erosion. Initial management included aspiration and empirical antibiotic therapy. However, recurrence necessitated incision and drainage, with cultures revealing fungal elements, prompting antifungal treatment. The patient responded well to therapy and no long-term complications were observed. This case underscores the importance of recognising NSA as a potential complication in leukaemia patients, even in the absence of trauma. Timely diagnosis and appropriate management are critical in preventing complications such as nasal deformities and recurrent infections.

**Keywords:** Acute lymphoblastic leukaemia, Case report, Fungal infection, Immunocompromised host, Saddle nose deformity

## CASE REPORT

A 28-year-old male patient presented to the Ear, Nose and Throat Outpatient Department (ENT OPD) with bilateral nasal obstruction and breathing difficulties for three days [Table/Fig-1]. The patient had been diagnosed with pre-B-cell ALL, 15 fifteen days prior.



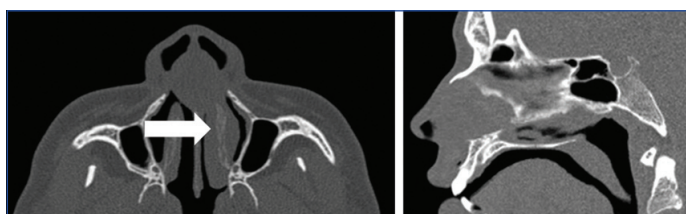
[Table/Fig-1]: Ear, Nose and Throat (ENT) examination showing a bilateral septal swelling.

The ENT examination revealed complete obstruction of the nasal cavity and bilateral bulging of the septum anteriorly. On probing, it was non tender and compressible. Subsequently, a Computed Tomography of the Paranasal Sinuses (CT PNS) scan was done, which revealed erosion of the septal cartilage, a distinct hypodense collection on both sides of the cartilaginous septum, a thin, hyperdense peripheral rim and a CT attenuation of +15, indicating a possible septal abscess [Table/Fig-2,3].

The patient was receiving injection L-asparaginase (10,000 U), injection vincristine (2 mg), tablet prednisolone (100 mg), tablet imatinib (400 mg) and tablet udiliv (300 mg) for the treatment of ALL. A wide-bore aspiration was performed, aspirated frank pus and conducted bilateral nasal packing after obtaining appropriate consent. The sample was sent for both Cartridge Based Nucleic Acid Amplification Test (CBNAAT) and culture sensitivity testing. The CBNAAT report was negative. The culture sensitivity report showed the presence of fungal septate hyphae, suggesting a fungal infection, after which the patient was started on tablet voriconazole (200 mg)

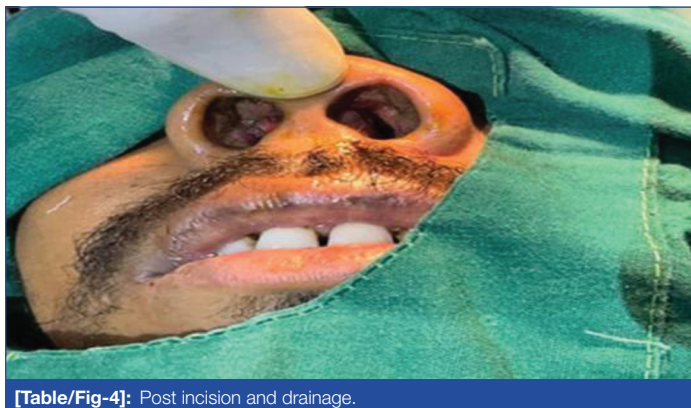


[Table/Fig-2]: Axial and sagittal soft-tissue window images showing a fairly well-defined hypodense collection (mean CT attenuation of +10 to +15 HU) on both sides of the cartilaginous nasal septum, measuring 28×12 mm with a thin peripheral hyperdense rim and obliteration of anterior nasal passages. No clear fat plane is visible between the collection and the adjacent nasal turbinates. The marked arrows reveal the areas of abscess formation.



[Table/Fig-3]: Axial and sagittal bone window images show erosion of septal cartilage.

twice daily for six weeks. Upon pack removal after 48 hours, the abscess had recurred. A 0.5 cm incision was made on the right side of the nasal septum, 2 mm away from the mucocutaneous junction. After draining approximately 5 mL of pus, the swelling subsided [Table/Fig-4]. The patient was discharged after five days of receiving intravenous broad-spectrum antibiotics. The patient was followed-up every week for the first four weeks and then monthly for three months. On follow-up, there was no evidence of recurrence or complications.



[Table/Fig-4]: Post incision and drainage.

## DISCUSSION

The NSA is a rare but potentially serious condition characterised by the collection of pus between the nasal septal cartilage and its overlying mucoperichondrium [1]. Abd Rahim NN et al., reported a case of a 46-year-old man with no prior co-morbidities who presented with nasal tip pain, progressive bilateral nasal obstruction and upper lip swelling. Imaging confirmed an NSA with septal erosion. The patient was treated with intravenous antibiotics and underwent surgical drainage, with culture revealing *Staphylococcus aureus* sensitive to ceftriaxone [1]. Beck AL, introduced a classification system for NSA, dividing them into three main categories: primary causes, which include trauma; secondary causes, typically resulting from infections; and idiopathic cases, where the underlying cause is unknown [2]. NSA can also develop as a result of dental infections, paranasal sinus infections, nasal septal surgeries, nasal vestibulitis and furunculosis. The most frequently identified causative organisms are aerobic bacteria, including *Staphylococcus aureus*, *Haemophilus influenzae* and *Streptococcus* species. Less commonly, *Klebsiella pneumoniae*, Enterobacteriaceae, *Streptococcus milleri* and various anaerobic bacteria have been isolated from NSA cases [3]. In immunocompromised patients, including those undergoing chemotherapy, NSA may develop due to opportunistic infections, including fungal pathogens, as seen in this case. NSA is uncommon, with an incidence of 0.8-1.6% in patients presenting with nasal infections or trauma; however, the incidence may be higher in immunocompromised populations due to their increased susceptibility to infections [4].

Facial trauma is the predominant cause of septal haematoma, followed by iatrogenic factors like septal surgery, ethmoid or sphenoid sinusitis, nasal furuncle and tobacco snuffing [5,6]. A septal abscess may arise as a complication of an untreated septal haematoma. Infrequently, immunocompromised individuals, such as those with diabetes [7] and Human Immunodeficiency Viruses (HIV) [8,9], have documented instances of septal abscess. Nasal septal haematoma occurs when an injury detaches the mucoperichondrium from the avascular septal cartilage. This disrupts submucosal arteries and causes blood to accumulate between the cartilage and perichondrium, resulting in ischaemia and cartilage necrosis [10]. If left untreated, the haematoma can serve as a breeding ground for bacteria and may progress to a septal abscess within 72 hours [10,11]. Debnam JM et al., documented two cases of NSA: one involving an elderly patient diagnosed with acute myelogenous leukaemia who developed the condition five months after the leukaemia diagnosis and another case of a teenager

with T-cell lymphoblastic lymphoma who developed a septal abscess following minor nasal trauma [11].

Even though nasal septal haematomas are extremely uncommon, they should never be disregarded as a clinical entity, particularly in immunocompromised patients. The following are clinical signs of nasal septal haematoma and abscess: nasal blockage (95%), pain (50%), fever and rhinorrhoea (25%), nasal bone fracture (15%) and bleeding (10%) [12]. Although a less experienced clinician may occasionally misdiagnose a septal haematoma or abscess as inferior turbinate hypertrophy or a deviated nasal septum, the diagnosis can be verified with needle aspiration [12]. Nasal septal haematomas frequently manifest as painless nasal swelling, in contrast to NSA, which are frequently painful [13]. Clinical examination, especially nasal endoscopic examination, frequently shows nasal septal swelling or smooth boggy swelling [14]. Additionally, Avci D reported a rare instance of spontaneous nasal septal haematoma in a patient from Turkey with idiopathic thrombocytopenia and chronic renal failure [15]. Hong CX et al., reported a case of a 51-year-old male with schizophrenia who presented with a two-week history of bilateral nasal obstruction and nasal cavity painful swellings. Initial management involved incision and drainage of an NSA, with subsequent improvement. Four months later, the patient was diagnosed with acute myelogenous leukaemia and experienced recurrent nasal septal haematomas during chemotherapy, which resolved after treatment [16].

Nasal obstruction as a presenting or accompanying symptom of leukaemia is uncommon. Love AA, reported two instances of nasal obstruction out of 152 well-documented cases of leukaemia over a ten-year period [17]. Haffly GN Schipfer LA, presented a case in which the sole complaint for 12 months preceding the diagnosis of leukaemia was nasal obstruction [18]. It is possible that leukaemic infiltration of the nasal mucosa occurs more often than previously thought but is simply overlooked because the symptom of nasal obstruction is overshadowed by other, more severe and dramatic manifestations of the disease.

## CONCLUSION(S)

This case highlights the potential for spontaneous NSA formation in immunocompromised patients, particularly those with haematological malignancies. The combination of thrombocytopenia and immunosuppression increases the risk of recurrent septal haematomas and abscess formation, which can lead to severe complications if not promptly managed. Clinicians should maintain a high index of suspicion for nasal septal infections in leukaemia patients, as early intervention is crucial to prevent structural deformities like saddle nose.

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