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SPOT THE DIAGNOSIS: IMAGE CHALLENGE

A Necrotic Nonhealing Nasal Lesion in a Young Man

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CLINICAL VIGNETTE

A 25-year-old man presented with fever, purulent nasal drainage, and an enlarging nasal lesion for six months. During this period, he was evaluated at urgent care centers on multiple occasions for purulent drainage, epistaxis, and fever and completed three courses of antibiotics with transient improvement but without complete resolution of symptoms. One month before presentation, he was admitted to an outside hospital where CT imaging revealed a lesion concerning for a left nasal abscess.

The lesion was aspirated and cultures grew methicillin-sensitive *Staphylococcus aureus*. He was discharged on antibiotics and a prednisone taper, but his symptoms recurred within days of completing treatment.

On presentation, the patient denied visual changes, cough, dyspnea, urinary abnormalities, or rash. He was born in Mexico, had lived primarily in the Gulf Coast region of the United States, and reported no recent travel or illicit substance use.

Physical examination was notable for a necrotic facial lesion involving the nasal septum with ulceration, crusting, and extension to the hard palate (Figure 1). There was no appreciable rash, lymphadenopathy, or synovitis, and cardiopulmonary examination was unremarkable. His temperature was 38°C; heart rate, 118 beats per minute; blood pressure, 148/72 mmHg; respirations, 16 breaths per minute; and oxygen saturation, 99% on room air. Serum electrolytes, liver function tests, white blood cell count, erythrocyte sedimentation rate, and C-reactive protein were within normal limits. HIV testing was negative, and hemoglobin A1c was 4.0%. ANA was positive at a titer of 1:320 with a speckled pattern, anti-SSA antibodies were positive, and p-ANCA and c-ANCA testing was negative.

Which of the following best characterizes the diagnostic significance of the findings in this patient?

A. Negative ANCA testing effectively excludes granulomatosis with polyangiitis (GPA)

B. Isolation of *S. aureus* provides a sufficient explanation for the lesion

C. Positive ANA and anti-SSA identify an autoimmune disorder as the most likely etiology for this presentation

D. The reported absence of cocaine use is sufficient to exclude a drug-induced lesion

E. The discordance between the clinical course and individual test results warrants further tissue evaluation



Figure 1. Clinical presentation of the sinonasal lesion. (A) Extensive crusting and tissue destruction involving the left greater than right nasal cavity. (B) Extension of the lesion to the hard palate.

ANSWER

E. The discordance between the clinical course and individual test results warrants further tissue evaluation

DISCUSSION

A biopsy was obtained. Bacterial and fungal cultures were negative, as were fungal and acid-fast stains. Histopathologic examination demonstrated an atypical lymphoid infiltrate. Immunohistochemical staining showed expression of NK/T-cell markers (CD2, CD3, CD7, and CD56) with in situ hybridization positivity for Epstein-Barr virus-encoded RNA, consistent with extranodal natural killer/T-cell lymphoma (ENKTL). PET/CT staging showed no evidence of distant disease, and he was treated with the SMILE regimen (dexamethasone, methotrexate, ifosfamide, L-asparaginase, etoposide) followed by consolidative radiotherapy. At six-month follow-up, he remained without evidence of disease.

ENKTL is a rare EBV-associated lymphoma that most commonly involves the upper aerodigestive tract and classically presents with progressive sinonasal destruction, ulceration, and tissue necrosis. Although uncommon in North America, it occurs with substantially higher frequency in East Asia and Latin America.[1] Early symptoms often mimic rhinosinusitis, and bacterial superinfection can further obscure the underlying diagnosis.

Recurrent or persistent bacterial sinusitis, particularly when symptoms only partially respond to appropriate antimicrobial therapy or persist for weeks to months, should prompt a diagnostic pause and reconsideration of the underlying etiology. The extensive tissue necrosis, septal destruction, and visible involvement of the hard palate in this patient are atypical for uncomplicated bacterial sinusitis and should raise concern for an invasive infectious, inflammatory, or neoplastic process. The differential diagnosis for destructive sinonasal lesions is broad and includes infectious etiologies (such as mucormycosis, endemic mycoses, mycobacterial infections, and parasitic infections), vasculitides (particularly granulomatosis with polyangiitis), drug-induced injury (such as intranasal cocaine use), and malignancy.[2] Autoimmune serologies in this case illustrate a diagnostic pitfall of their own. The positive ANA and anti-SSA may have contributed to diagnostic anchoring toward an autoimmune etiology despite the lack of a fully compatible clinical syndrome. Such antibodies are not specific for systemic autoimmune disease, and EBV-associated immune activation has been implicated in autoantibody production in the absence of a defined autoimmune disorder.[3] However, the significance of these serological findings in this patient remains uncertain.

Why Not the Other Choices?

- **Why doesn't negative ANCA testing exclude GPA?** ANCA testing alone cannot exclude GPA, as an estimated 10-50% of GPA cases are ANCA-negative, with ANCA-negativity occurring more frequently in patients with localized disease.
- **Why wasn't the isolation of *S. aureus* pathologic?** Primary bacterial sinusitis would be unlikely to progress despite multiple courses of appropriate antibiotics, and the degree of tissue destruction noted in this case would be atypical. Isolation of *S. aureus* more likely reflects secondary infection or colonization rather than the underlying cause of the lesion.
- **Why isn't this autoimmune disease?** Positive ANA and anti-SSA antibodies can occur in the absence of a defined autoimmune disease and should be interpreted within the clinical context. In this patient, the absence of a compatible clinical syndrome makes the serological findings insufficient to explain the progressive nasal lesion.
- **Why couldn't we exclude cocaine use on history alone?** Cocaine-induced midline destructive lesions (CIMDL) can mimic vasculitis and malignancy, presenting with progressive destruction of the nasal septum and surrounding structures. Although the absence of reported intranasal cocaine use makes CIMDL less likely, history alone is insufficient to exclude it when the clinical presentation is otherwise compatible.

Similarly, a negative ANCA does not exclude vasculitis, particularly in patients with localized disease in whom sensitivity is lower.[4] Therefore, autoimmune serologies such as ANCA and ANA should be interpreted as data points within the broader clinical context, rather than used in isolation to establish or exclude a diagnosis.

Progressive destructive sinonasal lesions warrant prompt tissue diagnosis. When clinical findings are atypical, symptoms recur despite appropriate therapy, or the disease course remains discordant with the presumed diagnosis, clinicians should reconsider the differential rather than continue or escalate empiric treatment. No single positive culture result, serologic finding, or negative historical feature should supersede an otherwise unexplained progressive clinical phenotype. Tissue diagnosis is critical because infectious, inflammatory, and malignant causes may present similarly but require fundamentally different management strategies.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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ETHICS STATEMENT

The authors obtained verbal informed consent, witnessed and documented in accordance with institutional guidelines, for publication of medical information and images.

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