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Extranodal NK/T-Cell Lymphoma, Nasal Type, with Multiorgan Failure Requiring Therapeutic Adaptation

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Abstract

Extranodal natural killer/T-cell lymphoma, nasal type, is a rare and highly aggressive malignancy, often associated with rapid local destruction and a poor clinical course. We report the case of a 45-year-old man who developed rapidly progressive oronasal disease complicated by massive epistaxis, respiratory failure, and multiorgan dysfunction. His clinical course was further marked by dialysis-dependent acute kidney injury, multidrug-resistant infections, and a prolonged intensive care unit stay. Because of severe clinical instability, cyclophosphamide, doxorubicin, vincristine, and prednisone chemotherapy was selected as a feasible alternative to standard approaches but was followed by severe bone marrow aplasia. This case highlights the major challenges involved in managing this disease in critically ill patients, in whom treatment decisions must be carefully individualized according to organ failure, infectious complications, and overall clinical instability.

Keywords

Extranodal NK/T-Cell Lymphoma, Antineoplastic Combined Chemotherapy Protocols, Bone Marrow Suppression

Introduction

Extranodal NK/T-cell lymphoma, nasal type (ENKTCL), is a rare and aggressive lymphoma that typically arises in the nasal cavity and upper aerodigestive tract. It often presents with destructive midline lesions, necrosis, nasal obstruction, and epistaxis, and it is more frequent in Asia and Latin America than in Western populations [1].

Treatment strategies for ENKTCL have evolved substantially over time. Anthracycline-based regimens such as CHOP have historically been used in the management of this disease. However, their efficacy may be limited, particularly in advanced-stage ENKTCL. Consequently, non-anthracycline-based regimens have increasingly become preferred therapeutic approaches, although treatment selection remains dependent on disease characteristics, clinical presentation, and treatment availability [2].

We report a case of nasal ENKTCL with rapidly progressive disease, epistaxis, respiratory failure, dialysis-requiring kidney injury, recurrent multidrug-resistant respiratory infections, and severe post-chemotherapy marrow aplasia.

Case Presentation

A 45-year-old man with type 2 diabetes mellitus and

hypertension developed an infiltrative lesion involving the nasal and oral region. At the beginning of the course, he underwent sinonasal surgery with biopsy. Within the first postoperative week, he developed progressive facial infection with abscess formation and worsening infiltrative oronasal disease (**Fig-1**). Microbiological evaluation of the lesion later showed *Pluralibacter gergoviae*, sensitive only to amikacin, and *Candida albicans*, with Gram staining demonstrating numerous Gram-negative bacilli and fungal elements.

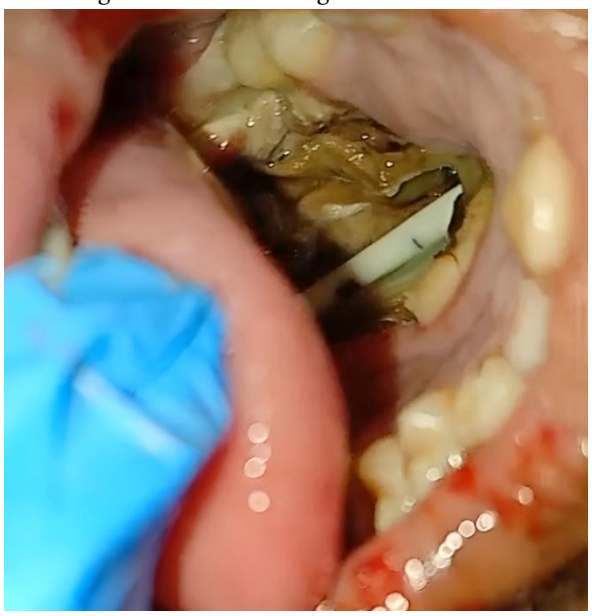

Fig-1:

Extensive necrotic and ulcerated lesion with friable, devitalized tissue in the posterior oral cavity.

During the second week of illness, the patient developed recurrent upper airway bleeding, followed by respiratory deterioration with pulmonary congestion and hemodynamic instability requiring orotracheal intubation. Histopathologic and immunohistochemical evaluation then established the diagnosis of extranodal NK/T-cell lymphoma, nasal type.

Over the following days, the patient had massive epistaxis requiring airway protection, right external carotid artery ligation, and tracheostomy. Cranial imaging showed pansinusopathy. Chest computed tomography (CT) demonstrated bronchial wall thickening, ground-glass opacities, small foci of consolidation, and small bilateral pleural effusions. Because of melena and nutritional difficulty related to the extensive oral lesion, upper gastrointestinal endoscopy was performed, and post-pyloric enteral access was established.

As hospitalization progressed, the patient developed severe acute kidney injury with marked azotemia, electrolyte abnormalities, and progressive creatinine elevation, eventually requiring hemodialysis. He first received methylprednisolone for cytoreduction, and chemotherapy with cyclophosphamide, doxorubicin, vincristine, and prednisone (CHOP) was then initiated. He remains on oncologic treatment, with care being continuously adjusted according to his clinical status.

The subsequent course was marked by prolonged invasive mechanical ventilation and recurrent respiratory infections. Sequential tracheal cultures yielded multidrug-resistant *Klebsiella aerogenes*, *Stenotrophomonas maltophilia* resistant to trimethoprim-sulfamethoxazole, and later *Pseudomonas aeruginosa*. Antimicrobial therapy was repeatedly modified according to microbiological findings and renal function, and prophylactic antifungal and anti-*Pneumocystis* therapy was added during immunosuppression.

After chemotherapy, the patient developed severe marrow aplasia with profound leukopenia, anemia, and thrombocytopenia, requiring filgrastim, transfusion support, and close hematologic monitoring. At the latest documented evaluation, he remained in the intensive care unit on invasive mechanical ventilation through tracheostomy and on dialysis support, with ongoing management for infection, cytopenias, and hemodynamic instability.

Discussion

This case reflects the biological aggressiveness of nasal ENKTCL and, more importantly, the difficulty of applying ideal lymphoma-directed therapy once the disease becomes intertwined with airway compromise, hemorrhage, organ failure, and infection. Anthracycline-based therapy has historically produced disappointing results in ENKTCL, and one proposed explanation is the frequent expression of multidrug-resistance pathways such as P-glycoprotein, which can reduce sensitivity to anthracyclines and vinca alkaloids [3]. In this patient, however, the practical therapeutic window was very different from that usually assumed

in guideline-based treatment algorithms. Radiotherapy was judged not feasible because the locally extensive disease would have required extensive treatment volumes with excessive exposure of adjacent critical structures [4]. At the same time, although asparaginase-containing regimens are more active in ENKTCL, they are also associated with clinically important toxicities, including hepatotoxicity, pancreatitis, thrombosis, encephalopathy, and hypersensitivity. In the present case, the patient already had severe infection, active bleeding, renal failure requiring dialysis, marked inflammatory activity, and later laboratory evidence of pancreatic and coagulation disturbances. Based on that clinical context, the choice of CHOP appears best understood as a pragmatic bridge strategy in a critically ill patient.

The hematologic evolution after chemotherapy is also clinically relevant and deserves a more critical interpretation than simply labeling it as an adverse event. Hematologic toxicities, including leukopenia, neutropenia, anemia, and thrombocytopenia, are documented after CHOP-based therapy [5]. In this case, severe marrow aplasia developed in a patient who was already exposed to a prolonged intensive care unit stay, invasive mechanical ventilation, multidrug-resistant infection, renal replacement therapy, and recent major bleeding. The severity of the cytopenias therefore likely reflects not only regimen toxicity but also the interaction between chemotherapy and a profoundly compromised physiologic reserve.

In prolonged intensive care unit admissions, particularly after repeated broad-spectrum antibiotic exposure and mechanical ventilation, multidrug-resistant Gram-negative colonization and infection become increasingly likely, including *Stenotrophomonas maltophilia* [6]. In the present case, recurrent isolation of multidrug-resistant *Klebsiella aerogenes*, *S. maltophilia*, and *Pseudomonas aeruginosa* was part of the expected ecological shift in a patient with prolonged critical illness and repeated antimicrobial pressure. Another point worth emphasizing is that the early coexistence of infection and tumor necrosis may obscure recognition of ENKTCL. Early tissue diagnosis remains essential when local progression, necrosis, and bleeding are disproportionate to an apparently straightforward infectious process.

This report is limited by its single-patient design and by the fact that the patient is still undergoing treatment, preventing assessment of long-term oncologic response. Its relevance lies in showing how management may need to be individualized when clinical instability limits the use of standard therapeutic strategies.

Conclusion

This case illustrates a severe presentation of nasal ENKTCL marked by airway-threatening local progression, major hemorrhage, renal failure, ventilator-associated infection, and marked post-chemotherapy marrow suppression. It also highlights that, in critically ill patients, disease trajectory may be shaped not only by lymphoma aggressiveness itself but also by the interplay between infectious complications, treatment-related cytopenias, and multiorgan dysfunction.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors declare no conflicts of interest.

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