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Advanced Nasopharyngeal Carcinoma with Intracranial Extension and Bilateral Cervical Lymphadenopathy: A Case Report

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ABSTRACT

Background: Nasopharyngeal carcinoma (NPC) is an epithelial malignancy that is often found in Southeast Asia and is ranked sixth as the most common cancer in Indonesia. This disease is often diagnosed at an advanced stage due to nonspecific initial symptoms. Case Description: A 72-year-old man presented with chronic epistaxis, bilateral nasal obstruction, tinnitus, and bilateral neck node enlargement. Nasoendoscopy revealed a mass obscuring the fossa of Rosenmüller, while a contrast-enhanced CT scan revealed an infiltrative lesion in the nasopharynx extending to the skull base and intracranially with multiple cervical lymphadenopathy. Histopathology revealed nasopharyngeal nonkeratinizing squamous cell carcinoma, stage T4N3Mx. Management and Outcome: The patient was referred for concomitant chemoradiotherapy as standard therapy for advanced stage, with planned follow-up at an oncology center. Discussion: This case highlights the association between smoking and salted fish consumption with the occurrence of NPC and the challenges of early diagnosis due to symptoms resembling chronic rhinitis. Conclusion: Recognizing recurrent epistaxis and painless neck node enlargement is crucial for early detection of NPC. A multidisciplinary approach and integrated chemoradiotherapy play a significant role in improving patient prognosis.

1. INTRODUCTION

Nasopharyngeal carcinoma (NPC) is a non-lymphomatous squamous cell carcinoma arising from the epithelial lining of the nasopharynx. According to the International Agency for Research on Cancer (IARC), in 2018 there were approximately 129,000 new cases of nasopharyngeal carcinoma worldwide, accounting for only 0.7% of all cancers diagnosed in that year.[1,2] In Indonesia, nasopharyngeal carcinoma is one of the most frequently encountered malignancies, ranking sixth among the most common cancers after breast, lung, cervical, colorectal, and liver cancers. NPC is also one of the most common cancers affecting men in Indonesia, along with lymphoma. Based on GLOBOCAN 2022 data, there were 18,835 new cases of nasopharyngeal carcinoma in Indonesia, with 12,949 associated deaths. The distribution of this cancer demonstrates remarkable ethnic and geographic characteristics, with higher prevalence observed in Southeast Asia, Southern China, North Africa, Greenland, and Alaska.[1,3,9,11]

Nasopharyngeal carcinoma is associated with several risk factors, including Epstein–Barr virus (EBV) infection, genetic predisposition, and environmental factors. The incidence of NPC is higher in men than in women, with a male-to-female ratio of approximately 2.5:1.[1,3] The risk of NPC increases with age, showing a first peak in late adolescence or early adulthood (15–24 years), followed by a second peak in later life (65–79 years).[4]

From a genetic perspective, certain human leukocyte antigen (HLA) alleles, a positive family history, and polymorphisms in immune- and tumor-related genes increase individual susceptibility to NPC and influence the host response to Epstein–Barr virus infection, which plays a crucial role in NPC pathogenesis. In addition, environmental exposures such as consumption of preserved foods containing nitrosamines, tobacco smoke exposure, and exposure to wood dust or

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industrial chemicals contribute to an increased likelihood of genetic mutations in nasopharyngeal epithelial cells.[12] Carcinogens such as nitrosamines, polycyclic aromatic hydrocarbons, and aromatic amines can induce DNA damage through the formation of DNA adducts. DNA adduct formation results from interactions between cytochrome P450 enzymes and carcinogens, whereby non-reactive carcinogens are converted into DNA-binding forms. Increased levels of DNA adducts are associated with the early stages of carcinogenesis, and the resulting genetic alterations may lead to tumor formation.[13]

These genetic and environmental factors interact closely with Epstein–Barr virus infection, which plays a central role in oncogenesis. In individuals with genetic predisposition and exposure to environmental carcinogens, chronic EBV infection accelerates the accumulation of mutations that ultimately promote malignant cellular transformation. EBV expresses latent proteins such as latent membrane protein 1 (LMP1), latent membrane protein 2 (LMP2), and Epstein–Barr nuclear antigen 1 (EBNA1), as well as non-coding RNAs including EBER and BART. LMP1 functions similarly to a constitutively active CD40 receptor, activating signaling pathways such as NF-κB, JAK/STAT, and MAPK, which stimulate proliferation, survival, and resistance to apoptosis in infected epithelial cells. EBNA1 maintains viral episomes within host cells and regulates host gene expression. Chronic EBV infection in epithelial cells that have already sustained genetic damage due to environmental carcinogen exposure accelerates the accumulation of mutations, including inactivation of p16 and RASSF1A and activation of cyclin D1, thereby promoting malignant transformation. Furthermore, EBV induces DNA hypermethylation and epigenetic modifications that suppress tumor suppressor genes, creating a microenvironment conducive to clonal proliferation and tumor progression. The combined effects of viral gene expression, genomic instability, and dysregulation of signaling pathways such as NF-κB and PI3K/MAPK, along with mutations in TP53 or MHC class I genes, contribute to the proliferation, heterogeneity, and aggressiveness of nasopharyngeal carcinoma.[1,3]

The characteristic clinical manifestations of nasopharyngeal carcinoma can be classified into nasal symptoms (nasal obstruction, epistaxis, postnasal drip, hyponasal speech, or cacosmia), otologic symptoms (conductive hearing loss, middle ear effusion, or aural fullness), and nodal involvement (cervical lymphadenopathy).[5,10] In the early stages, signs and symptoms are often nonspecific and develop insidiously, leading to delayed diagnosis. NPC is frequently diagnosed at advanced stages (III–IV) due to its concealed anatomical location and mild early symptoms, necessitating additional diagnostic investigations such as nasoendoscopy, CT or MRI imaging, and plasma EBV DNA biomarkers. Nasoendoscopy allows direct visualization of the nasopharynx, CT scanning is useful for assessing bony invasion and destruction, MRI provides excellent soft tissue contrast for evaluating tumor extension, and plasma EBV DNA serves as a valuable diagnostic and prognostic biomarker, particularly in endemic populations.[5,7]

The mainstay of treatment for nasopharyngeal carcinoma is radiotherapy, particularly intensity-modulated radiotherapy (IMRT), with or without concurrent chemotherapy, due to the tumor's anatomical inaccessibility for surgery and its high radiosensitivity. Radiotherapy aims to achieve local and nodal control, while chemotherapy administered concurrently enhances locoregional control and reduces the risk of distant metastasis.[3,5] Prognosis depends largely on the stage at diagnosis and the response to initial therapy.

Considering the high incidence in Indonesia and its association with multifactorial risk factors, nasopharyngeal carcinoma represents a significant public health concern. In this case report, the authors present a patient diagnosed with nasopharyngeal carcinoma who exhibited typical clinical manifestations, diagnostic challenges, and therapeutic responses, highlighting the importance of early detection and a multidisciplinary management approach.

2. CASE DESCRIPTION

A 72-year-old male patient presented to the ENT outpatient clinic with a chief complaint of recurrent epistaxis for the past two years. In addition, the patient reported frequent rhinorrhea and a sensation of nasal obstruction in both nostrils almost every day. Previously, the patient had also experienced tinnitus and otalgia, which were ignored and therefore not medically evaluated.

These complaints were accompanied by masses on both the right and left sides of the neck. The neck masses were initially small, gradually increased in size, were painless, and showed no signs of redness. Approximately one year prior, the patient underwent cervical lymph node surgery performed by a general surgeon at another hospital; however, the excised tissue was not examined histopathologically, and no further treatment was administered.

Over the past six months, the patient experienced persistent headaches, worsening tinnitus, a sensation of fullness in the left ear accompanied by pain, occasional otorrhea from the left ear, and progressive hearing difficulty. In the past few weeks, the patient also complained of visual disturbances in the form of diplopia affecting the left eye. The patient denied significant weight loss but reported decreased appetite over the past few months. There were no complaints of fever, nausea, vomiting, facial pain, dysphagia, hemoptysis, or shortness of breath.

The patient had a history of recurrent upper respiratory tract infections but denied any history of hypertension, asthma, diabetes mellitus, malignancy, nasal polyps, dental infection, tonsillar infection, or renal disease. There was no family history of cancer. The patient had a long-standing smoking habit since school age, consuming approximately one pack per day (around 12 cigarettes), sometimes more, but had quit smoking five years prior. In addition, the patient reported frequent consumption of salted fish approximately three times per week and a preference for preserved foods.

Physical examination revealed proptosis. Otoscopic examination of the left ear showed cerumen and discharge, with a yellowish tympanic membrane. Tuning fork tests revealed Rinne positive on the right ear and negative on the left ear, Weber lateralized to the left, and Schwabach test equal to the examiner on the right and prolonged on the left, consistent with left-sided conductive hearing loss. Nasal examination showed hyperemic mucosa in both nasal cavities with serous secretions. Palpation revealed enlarged cervical lymph nodes at levels III and IV bilaterally; the nodes were rubbery in consistency, immobile, and non-tender. Nasoendoscopic examination demonstrated narrowing of both nasal cavities, bilateral inferior turbinate edema, obliteration of the Eustachian tube orifice, torus tubarius, and fossa of Rosenmüller by a mass, with a visible mass occupying the nasopharynx.

Laboratory, radiological, and histopathological investigations were performed. Laboratory results showed hemoglobin 10.9 g/dL, leukocytes $10.68 \times 10^3/\mu L$, prothrombin time 10.7 seconds, activated partial thromboplastin time 25.6 seconds, AST 20 U/L, ALT 10 U/L, blood urea 37 mg/dL, serum creatinine 0.92 mg/dL, and estimated glomerular filtration rate (eGFR) 89 mL/min/1.73 m^2. Contrast-enhanced CT scan of the nasopharynx revealed a heterogeneously enhancing mass on the posterior wall of the nasopharynx extending inferiorly to the oropharynx, involving the choanae, bilateral parapharyngeal spaces, left masticator space, infiltrating the left pterygoid muscles, with destruction of the medial and lateral pterygoid plates, destruction of the left sphenoid sinus wall and vomer bone, extension into the left sphenoid sinus, and intracranial extension to the left parasellar and temporal regions. Multiple enlarged lymph nodes were observed in the parapharyngeal region and bilateral cervical levels II–III and right level IV, with necrotic components; the largest measured 2.6 × 1.9 × 2.5 cm. Opacification of the left frontal sinus, ethmoid sinuses, and bilateral maxillary sinuses was also noted. These findings were consistent with a malignant nasopharyngeal mass with intracranial extension, classified as T4, and multiple bilateral cervical lymphadenopathies consistent with N3 disease, along with left frontal sinusitis, bilateral ethmoid and maxillary sinusitis, and bilateral mastoiditis. The lesion demonstrated extensive local infiltration involving the skull base and paranasal sinuses, consistent with advanced-stage nasopharyngeal carcinoma (T4N3Mx).

Histopathological examination of bilateral nasopharyngeal biopsy specimens showed tissue fragments lined by pseudostratified ciliated columnar epithelium with scattered goblet cells. The subepithelial tissue appeared edematous with seromucinous glands, some showing cystic dilatation. A malignant epithelial tumor composed of infiltrative nests within adipose tissue was identified. Tumor cells were arranged in syncytial patterns with round to oval nuclei, pleomorphism, hyperchromasia, vesicular nuclei with prominent nucleoli, and eosinophilic cytoplasm. Mitotic figures were present, along with infiltration of chronic inflammatory cells. No lymphovascular invasion was identified. These histological findings were consistent with non-

keratinizing squamous cell carcinoma of the nasopharynx, with no evidence of lymphovascular invasion.

The patient was diagnosed with nasopharyngeal carcinoma staged as T4N3Mx, according to the AJCC 8th edition, where T4 indicates intracranial extension, N3 indicates bilateral lymphadenopathy larger than 6 cm, and Mx indicates that distant metastasis could not be assessed due to limited information. Differential diagnoses included nasopharyngeal lymphoma, adenocarcinoma, and metastasis from another primary tumor. Advanced-stage disease (T4N3) is commonly encountered due to nonspecific early symptoms, resulting in delayed diagnosis. This case illustrates advanced presentation secondary to delayed diagnosis and low patient awareness.

Management at the hospital included nasopharyngeal biopsy followed by referral to RSUP Fatmawati for access to IMRT radiotherapy and medical oncology services. The planned treatment consisted of concurrent chemotherapy (cisplatin 100 mg/m^2 every three weeks), radiotherapy with a total dose of 70 Gy in 35 fractions, and response evaluation using MRI or CT scan after three months. This case highlights the importance of early detection of nasopharyngeal carcinoma in patients presenting with chronic epistaxis, nasal obstruction, and bilateral cervical lymphadenopathy. Accurate diagnosis and prompt referral to centers equipped with integrated chemoradiotherapy facilities are crucial to improving patient prognosis.

3. DISCUSSION

This case demonstrates a delay in diagnosis resulting from the initial misinterpretation of mild symptoms such as epistaxis and chronic nasal obstruction. The performance of cervical lymph node surgery without histopathological examination reflects a low level of clinical suspicion for nasopharyngeal carcinoma (NPC) at the primary care level. Diagnostic delay may occur due to nonspecific symptoms, limited early detection at primary healthcare facilities, and restricted access to nasoendoscopic examinations in regional areas, leading to diagnosis at an advanced stage (T4N3) with intracranial invasion. Therefore, nasopharyngeal screening is essential in patients presenting with chronic epistaxis or tinnitus, particularly in endemic regions.

Nasopharyngeal carcinoma is associated with multiple risk factors, including Epstein–Barr virus (EBV) infection, genetic predisposition, lifestyle, dietary habits, and environmental exposures. The incidence of NPC is higher in men than in women, with a male-to-female ratio of approximately 2.5:1.[1,2,3] The risk of NPC increases with age, with a first peak in late adolescence or early adulthood (15–24 years), followed by a second peak in later life (65–79 years).[4] This demographic pattern is consistent with the present case, involving a 72-year-old male patient. The patient had a long history of smoking since a young age and habitual consumption of salted fish and preserved foods. Smoking (increasing risk by approximately 2–6 fold), heavy alcohol consumption, and dietary agents containing nitrosamines have been strongly implicated in the pathogenesis of this malignancy.[5]

In the early stage, the patient experienced recurrent epistaxis accompanied by blood-tinged nasal discharge, particularly in the morning. Theoretically, NPC commonly causes mild, intermittent epistaxis due to the fragility of blood vessels in the nasopharyngeal region infiltrated by tumor. Its concealed anatomical location prevents direct visualization of the lesion, leading early symptoms to be misinterpreted as simple rhinitis. These symptoms were accompanied by chronic bilateral nasal obstruction, reflecting the presence of a mass obstructing the nasopharyngeal lumen.[5,6]

A history of tinnitus, otalgia, and aural fullness indicates Eustachian tube involvement due to compression or obstruction of its nasopharyngeal orifice by the tumor mass. This condition may lead to otitis media with effusion or even mastoiditis, as confirmed by radiological findings. These findings are supported by the Rinne, Weber, and Schwabach test results, which were consistent with left-sided conductive hearing loss due to Eustachian tube dysfunction or secondary chronic inflammatory processes.[5,6,7]

Bilateral neck masses with rubbery consistency, immobility, and absence of tenderness represent metastatic cervical lymphadenopathy. NPC is well known for its tendency toward early metastasis to cervical lymph nodes, particularly levels II–IV. Chronic headache and diplopia

indicate tumor extension to the skull base and intracranial cavity, with possible involvement of cranial nerves, particularly the oculomotor (III), trochlear (IV), or abducens (VI) nerves. Such extension is characteristic of T4-stage disease in the TNM staging system for NPC.[6,7]

On nasal examination, hyperemic mucosa with serous secretion indicates chronic inflammatory or reactive processes secondary to obstruction and mucosal irritation by the nasopharyngeal tumor mass. Bilateral nasal cavity obstruction, observed as narrowing of both nasal cavities, results directly from mass effect on the lateral walls of the nasopharynx, disrupting normal airflow. Inferior turbinate edema further exacerbates nasal airflow limitation.[5,7]

Obliteration of the Eustachian tube orifice, torus tubarius, and the fossa of Rosenmüller by the mass is highly characteristic of NPC. The fossa of Rosenmüller represents the most common site of origin of nasopharyngeal tumors; therefore, endoscopic examination frequently reveals a mass occupying this region. Obstruction of the Eustachian tube orifice explains the patient's otologic symptoms, including tinnitus, aural fullness, otalgia, and hearing impairment due to middle ear ventilation dysfunction.[6,7]

Contrast-enhanced CT imaging in this patient demonstrated an extensive and invasive nasopharyngeal mass, highly consistent with advanced-stage NPC. The primary lesion appeared as a heterogeneously enhancing mass on the posterior wall of the nasopharynx. Inferior extension into the oropharynx and involvement of the choanae explained the bilateral nasal obstruction. Destruction of the medial and lateral pterygoid plates, the left sphenoid sinus wall, and the vomer bone indicated aggressive tumor infiltration with osseous erosion. Extension into the left sphenoid sinus and intracranial parasellar–temporal region indicated superior spread through the skull base, with potential cranial nerve involvement. Destruction of the left pterygoid structures correlates with headache and visual disturbance, likely due to abducens nerve involvement.[6]

CT imaging demonstrated multiple bilateral cervical lymphadenopathies at levels II–III and right level IV, some with necrotic components. The largest node measured 2.6 × 1.9 × 2.5 cm, indicating a significant tumor burden. Bilateral nodal dissemination is consistent with the lymphatic metastatic pattern of NPC, which commonly involves the parapharyngeal nodes as first-echelon nodes. These findings correspond to N3 disease in TNM staging, reflecting advanced nodal involvement. Associated left frontal sinusitis, bilateral ethmoid and maxillary sinusitis were caused by sinus ostial obstruction and secondary inflammation. Bilateral mastoiditis was likely secondary to Eustachian tube obstruction at the torus tubarius, correlating with the patient's otologic symptoms.[3,5,6,7]

Large-scale lymphatic mapping studies strongly support that bilateral nodal involvement in NPC is common, particularly at level II, with substantial proportions of patients also showing level III and even level IV disease in advanced stages.[14] Previous studies by Xu et al. demonstrated that in advanced-stage disease, levels II and III serve as primary nodal basins, with frequent extension to level IV, particularly in cases of high nodal tumor burden or extracapsular spread. These findings support the interpretation that bilateral involvement and right-sided level IV extension in this case represent a typical progression pattern of advanced disease rather than atypical spread. Probabilistic lymphatic modeling further suggests that bilateral cervical nodal spread is more likely when the primary tumor is located near midline structures or when tumor burden is substantial, due to cross-neck lymphatic drainage.[15,16] These studies emphasize that increasing tumor volume strongly correlates with bilateral nodal involvement, consistent with the large necrotic lymphadenopathy observed radiologically in this patient. Advanced hypopharyngeal and nasopharyngeal nodal subsites frequently metastasize to levels II–IV, and nodal necrosis represents a key imaging marker of aggressive metastatic infiltration. The observed multilevel bilateral cervical lymph node metastases, particularly at levels II–IV with necrotic features, are highly consistent with advanced radiologic and anatomic dissemination patterns in advanced head and neck cancer, indicating high-stage nodal disease (N2–N3 pattern) and underscoring the need for comprehensive regional management.[17,18]

Currently, MRI, CT, and ^{18}F-fluorodeoxyglucose (^{18}F-FDG) PET/CT are the most commonly used imaging modalities for staging and radiotherapy planning in NPC. Contrast-enhanced CT is the modality of choice for assessing bony invasion, soft tissue extension, and cervical nodal basins.

MRI is superior for evaluating intracranial extension, cranial nerve involvement, and paranasal sinus invasion, and provides excellent soft tissue contrast for muscular involvement assessment. PET/CT is preferred for remission assessment and recurrence evaluation and is also routinely used in early-stage disease to detect distant metastases.[3,7,8]

Biopsy revealed non-keratinizing nasopharyngeal squamous cell carcinoma, a histological type commonly found in regions with high NPC incidence and strongly associated with EBV infection and dietary factors (e.g., salted fish consumption). The absence of lymphovascular invasion does not negate the fact that the disease has already spread extensively at both regional and local levels. Clinical staging demonstrated T4 disease (intracranial invasion, masticator space involvement, skull base invasion) and N3 disease (multiple large bilateral cervical lymphadenopathies). The overall clinical stage is likely stage IVA/IVB, depending on distant metastasis findings (which were undetermined in this case).[3,10]

Histopathologically, non-keratinizing NPC demonstrates classic lymphoepithelioma-like features, consisting of large polygonal tumor cells arranged in syncytial sheets with indistinct borders. These cells typically exhibit vesicular nuclei with prominent nucleoli, while the surrounding stroma is densely infiltrated by lymphocytes. This distinctive microscopic pattern reflects the strong etiological relationship with EBV and has been consistently observed even in non-endemic populations, where it remains a defining diagnostic feature of the disease.[19] According to the latest WHO Classification of Head and Neck Tumors, NPC is divided into three main histological subtypes: keratinizing squamous cell carcinoma (Type I), non-keratinizing differentiated carcinoma (Type II), and non-keratinizing undifferentiated carcinoma (Type III). Contemporary pathological reviews indicate that Types II and III predominate globally, with the undifferentiated subtype (Type III) demonstrating the most classical histological features and the strongest association with EBV infection.[20,21]

Radiotherapy and chemotherapy constitute the standard treatment approach for advanced-stage NPC due to the tumor's anatomical inaccessibility for surgery and high radiosensitivity. Radiotherapy aims to control both primary and nodal disease, while chemotherapy is administered concurrently to enhance locoregional control and prevent distant metastasis. Surgical intervention is limited to very small primary lesions or limited recurrences, as the morbidity associated with extensive surgical resection often exceeds that associated with radiotherapy in this region. Recent innovations in radiation delivery include intensity-modulated radiotherapy (IMRT), which utilizes CT-based imaging to define target volumes and modulate beam intensity precisely. Radiotherapy is also used for recurrent NPC, although re-irradiation with curative intent depends on the time interval since initial treatment and may require enrollment in clinical trials. Radiotherapy is additionally used for palliative purposes.[3,5,9]

In advanced-stage NPC, the preference for concurrent chemoradiotherapy (CCRT) over radiotherapy alone is driven by strong evidence demonstrating improved overall survival and enhanced locoregional control. Consensus guidelines emphasize that platinum-based chemotherapy administered during radiotherapy acts as a radiosensitizer, intensifying tumoricidal effects and significantly reducing treatment failure in locally advanced disease. This therapeutic rationale has become a global standard, particularly for stage III–IV NPC, where tumor burden and nodal involvement necessitate simultaneous systemic and local control.[22]

Effective NPC management increasingly relies on a multidisciplinary approach, involving coordinated contributions from radiation oncologists, medical oncologists, ENT specialists, radiologists, nutritionists, and supportive care teams. Evidence from Indonesia demonstrates that structured multidisciplinary workflows significantly improve survival in stage III–IV NPC by optimizing treatment delivery, reducing delays, and ensuring continuity of supportive care. In addition to oncologic decision-making, multidisciplinary nutritional management plays a crucial role, as intensive treatment often leads to toxicities that impair oral intake. Dedicated nutrition teams have been shown to reduce severe treatment-related toxicity, improve caloric adequacy, and mitigate weight loss during chemoradiotherapy.[23,24]

Treatment-related adverse effects—particularly mucositis, xerostomia, dysphagia, and treatment-induced malnutrition—are especially relevant in elderly patients, who often have physical frailty, comorbidities, and limited physiological reserve. Studies emphasize the need for

early nutritional intervention, swallowing preservation strategies, and proactive mucosal injury management to maintain treatment adherence and minimize unplanned interruptions. By integrating evidence-based oncologic therapy with strong multidisciplinary and nutritional support, clinicians can achieve not only improved survival outcomes but also better functional recovery and quality of life during and after treatment.[23,24]

Prognostic factors for NPC include age, sex, tumor stage, and EBV-DNA levels. Overall prognosis and 5-year survival rates have improved with advancements in radiotherapy techniques, with reported historical 5-year survival rates increasing from approximately 25–40% to around 70% in the modern treatment era.[5,6] In this case, several factors worsen prognosis, including advanced age, advanced stage, and cranial nerve involvement. Post-treatment monitoring of plasma EBV DNA levels is also recommended as a molecular biomarker for detecting residual disease or relapse. Following therapy, patients should undergo follow-up every three months during the first year to evaluate treatment response and to prevent and manage early post-treatment complications.

Initial differential diagnoses included nasopharyngeal lymphoma, adenocarcinoma, and metastasis from other head and neck carcinomas. However, the histopathological findings of non-keratinizing squamous cell carcinoma and CT imaging demonstrating characteristic local bone destruction typical of NPC effectively excluded these possibilities. This case emphasizes the importance of histopathological evaluation of any painless cervical lymphadenopathy, particularly in EBV-endemic regions. Early detection through nasoendoscopic screening can prevent late-stage diagnosis and significantly improve prognosis.

4. CONCLUSION

Nasopharyngeal carcinoma (NPC) is a malignant epithelial tumor of the nasopharynx that is frequently diagnosed at an advanced stage due to its nonspecific early symptoms. This case describes an elderly male patient with risk factors including smoking and habitual consumption of salted fish, who presented with chronic epistaxis, bilateral nasal obstruction, tinnitus, and bilateral cervical lymphadenopathy. The diagnosis was established through nasoendoscopic examination, contrast-enhanced CT imaging demonstrating an extensive infiltrative mass with intracranial extension, and histopathological findings consistent with non-keratinizing squamous cell carcinoma. Management was carried out using concurrent chemoradiotherapy as the standard treatment for advanced-stage disease. This case underscores the importance of early detection and comprehensive evaluation of any cervical lymphadenopathy and chronic epistaxis to improve prognosis in patients with nasopharyngeal carcinoma.

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