TURKISH JOURNAL of ONCOLOGY

Turk J Oncol 2025;40(4):257–263
doi: 10.5505/tjo.2025.4605

ORIGINAL ARTICLE

Radiation-induced Lower Cranial Palsy in Nasopharyngeal Cancer: A Retrospective Dosimetric Analysis

Meltem DAĞDELEN,[1] İsmail Fatih DEMİREL,[1] Sevda YENER,[2] Songül ÇAVDAR KARAÇAM,[3] Günay CAN,[4] Ömer Erol UZEL[1]

[1]Department of Radiation Oncology, İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, İstanbul-*Türkiye*
[2]Department of Radiation Oncology, Bağcılar Training and Research Hospital, İstanbul-*Türkiye*
[3]Vocational School of Health Services, İstanbul University-Cerrahpaşa Radiotherapy Program, İstanbul-*Türkiye*
[4]Department of Public Health, İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, İstanbul-*Türkiye*

OBJECTIVE

This study aimed to assess the incidence and dosimetric predictors of radiation-induced lower cranial nerve (CN 9–12) palsy in patients with nasopharyngeal carcinoma (NPC), treated with intensity-modulated radiotherapy (IMRT) ± chemotherapy.

METHODS

A total of 125 patients with histologically confirmed NPC who underwent radiotherapy (RT) with or without chemotherapy between 2010 and 2024 were retrospectively reviewed. Lower cranial nerves (CN 9–12) were contoured, and dose-volume parameters including D_{max}, D_{mean}, D_{2cc}, D_{1cc}, and $D_{0.5cc}$ were analyzed. Statistical analyses were conducted using the Mann-Whitney U test and Cox regression to assess associations between clinical features, radiation dose, and the incidence of nerve palsy.

RESULTS

Median follow-up was 44 months. Cranial nerve palsy was observed in 8 patients (6.4%), all involving CN 12, with concomitant CN 9–11 involvement in 5 cases. No statistically significant correlation was found between palsy and patient characteristics or dosimetric parameters.

CONCLUSION

Although our study did not demonstrate statistically significant associations, likely due to limited sample size, contouring and sparing of lower cranial nerves in radiotherapy planning are crucial. These nerves play a vital role in preserving long-term quality of life, and their protection should be integrated into routine clinical practice.

Keywords: Lower cranial nerve palsy; nasopharyngeal carcinoma; radiation-induced neuropathy; radiotherapy.

INTRODUCTION

The primary treatment for nasopharyngeal cancer (NPC) is radiotherapy (RT) ± chemotherapy (CT). Intensity-modulated radiotherapy (IMRT) has been used in NPC for the past two decades. Local control (LC) rates are over 90% with IMRT and CT.[1] In addition, IMRT reportedly decreases toxicity rates and provides better survival rates in NPC cases. Therefore chronic side effects are becoming increasingly important, espe-

Received: June 11, 2025
Revised: August 13, 2025
Accepted: September 09, 2025
Online: December 26, 2025

Accessible online at:
www.onkder.org

Dr. Meltem DAĞDELEN
İstanbul Üniversitesi-Cerrahpaşa,
Cerrahpaşa Tıp Fakültesi,
Radyasyon Onkolojisi Kliniği,
İstanbul-Türkiye
E-mail: meltem.dagdelen@iuc.edu.tr

Turk J Oncol 2025;40(4):257–263
doi: 10.5505/tjo.2025.4605

cially as overall survival has improved with IMRT.[2] Chronic injury is possible as the lower cranial nerves usually remain within the primary high-dose volume during treatment. Lower cranial nerve injury may result in a significant reduction in the patient's quality of life. There is limited evidence on the relationship between the dose to which the lower cranial nerves are exposed and nerve damage.[3] Some of the published data focus on D_{max} for nerves considered serial organs, while others focus on the mean dose.[4,5] Furthermore, lower cranial nerve contouring in treatment planning is uncommon. In this study, the dosimetric differences in the lower cranial nerves and the dose-volume relationship in cases with side effects will be evaluated by contouring the lower cranial nerves and retrospectively analyzing the radiotherapy plans.

MATERIALS AND METHODS

Patient Characterictics

A total of 125 patients diagnosed with nasopharyngeal cancer who underwent definitive RT with or without CT at our institution between 2010 and 2024 were included in the study. The patient characterictics is presented in Table 1. The inclusion criteria were patients with histopathologically confirmed nasopharyngeal cancer who underwent curative RT ± CT, had at least 6 months' follow-up after treatment, had been followed-up with magnetic resonance imaging (MRI) scans, had no primary recurrence, and had no involvement of the lower cranial nerves before treatment. The exclusion criteria were patients who did not undergo treatment or follow-up at our institution; those who did not follow with MRI scans; and those with recurrence were excluded from the study. All patients underwent a standard diagnostic radiological assessment, which included nasopharyngeal and neck magnetic resonance imaging (MRI) on admission and after three courses of neoadjuvant chemotherapy. In cases diagnosed after 2010, an additional procedure was conducted as a routine part of the radiologic evaluation: 18-Fluorodeoxyglucose positron emission tomography-computed tomography (18-FDG PET-CT). Tumour-node-metastasis staging was performed under the eighth edition of the American Joint Committee on Cancer (AJCC) classification. The 5th version of the Common Terminology Criteria for Adverse Events (CTCAE) was used to classify the adverse effects. This study was approved by the Istanbul University-Cerrahpaşa Rectorate Ethics Committee and adhered to the principles of the Declaration of Helsinki.

Chemotherapy

In patients with T3, T4, and N3 tumors, chemoradiotherapy is typically initiated following three cycles of induction chemotherapy. Before 219, the induction CT regimen was TPF (5-FU 750 mg/m^2 by 24-h continuous infusion d1–5, cisplatin 75 mg/m^2 d1, and docetaxel 75 mg/m^2 d1 for three cycles every 3 weeks). Subsequently, GP (gemcitabine 1 g/m^2 d1 and d8 with cisplatin 80 mg/m^2 d1, for three cycles every 3 weeks) was used as the induction CT regimen. Cisplatin chemotherapy was administered at a dose of either 75–100 mg/m^2 every three weeks or 40 mg/m^2 every week, according to the patient's performance status, concomitantly with RT. Of the patients, 38% underwent induction chemotherapy. The proportion of patients who received cisplatin above 200 mg, either with or without induction chemotherapy, is as follows 76.7 %.

Radiotherapy

The patients were all immobilized with the use of a thermoplastic head and shoulder mask. A series of computed tomography (CT) scans were obtained at a slice thickness of 1.25–2.5 mm, extending from the cranial vertex to the bifurcation of the carina in the supine position. The planning CT scan was fused with the MRI and PET/CT images.

Target delineation, dose planning, and treatment volume definitions were performed in accordance with the methodology established in our previous study. The same contouring protocols and dose constraints were applied in a consistent approach, without any modifications.

The radiotherapy dose was reduced for pediatric patients. The dose for PTV high-risk was 61.2–63Gy, for PTV intermediate-risk was 54Gy, and PTV low-risk was 45Gy. Of these 80% underwent the simultaneous integrated boost (SIB) technique.[6]

Treatment and Follow-up

The IMRT or volumetric modulated arc therapy (VMAT) plan was generated for each patient using the Eclipse version 8.6 treatment planning system with 6-MV photon beams from a LINAC (linear accelerator; Varian Medical Systems, Palo Alto, USA). Verification of the radiotherapy volume was conducted on each fraction using either a cone beam CT scan or a kilovoltage (kV) imaging modality for image-guided radiation therapy (IGRT). A dose-volume histogram (DVH) was used to define the target dose and the dose to organs at risk (OAR). The patients were examined every week to evaluate any acute adverse effects. All patients completed the planned course of treatment. At each visit, patients underwent

Table 1 Patient characteristics

	n (125)	%
Age (years)		
≤50	64	51.2
>50	61	48.8
Gender		
Male	97	77.6
Female	28	22.4
Smoking status		
Non-smoker	60	48
Smoker/Ex-smoker	56	44.8
Unknown	9	7.2
Diabetes mellitus		
No	102	81.6
Yes	14	11.2
Unknown	9	7.2
Hypertension		
No	87	69.6
Yes	29	23.2
Unknown	9	7.2
Pathology		
Keratinized	3	2.4
Non-keratinized undiferentiated	122	97.6
T-stage		
T0	3	2.4
T1	42	33.6
T2	37	29.6
T3	17	13.6
T4	26	20.8
N-stage		
N0	23	18.4
N1	23	18.4
N2	60	48
N3	19	15.2
Treatmen Modality		
CRT	58	46.4
IC+CRT	50	40
RT	17	13.6
Total Cisplatin Dose		
≤200 mg/m^2	29	23.2
>200 mg/m^2	96	76.8

ICRT: Chemoradiotherapy; IC: Induction chemotherapy; RT: Radiotherapy

both endoscopic examination and MRI of the nasopharynx and neck. This follow-up schedule was conducted every 3 months for the first two years, every 6 months for the next three years, and annually thereafter.

Dosimetric Data and Statistical Analysis

A total of 125 patients with histologically confirmed NPC who underwent radiotherapy with or without chemotherapy between 2010 and 2024 were retrospectively reviewed. For a detailed dosimetric analysis, we selected a subset of 96 patients from this cohort. The remaining 29 patients were excluded due to technical limitations, such as the unavailability of complete treatment planning for accurate contouring, thereby minimizing the potential for selection bias. For these 96 patients, the lower cranial nerves (CN9, CN10, CN11, and CN12) were separately and bilaterally delineated on treatment planning CT scans that were fused with MRI and PET/CT images (Fig. 1). This contouring was performed by a single experienced radiation oncologist using a published CT-based atlas as an anatomical reference by Mourad et al.[7] to ensure consistency. The relationship between several dosimetric parameters (D_{max}, D_{mean}, D_{2cc}, D_{1cc}, and $D_{0.5cc}$) and lower cranial nerve palsy was then analyzed. The diagnosis was primarily based on clinical findings, as documented in patient records. To ensure diagnostic accuracy, all patient files were reviewed for physical examination findings, and all MRI reports interpreted by a neuroradiologist were carefully examined to identify patients with nerve palsy. The findings in the nerve-palsied patients were then confirmed by a physical examination performed by a physician experienced in neuro-oncology during a follow-up appointment. Regarding electromyography (EMG), we confirmed that routine EMG was not performed. The trapezius muscle was examined for the presence of multiple cranial palsy on magnetic resonance imaging (MRI) in patients presenting cranial nerve (CN) 12 palsy. The patient characteristics were determined using descriptive statistical methods. The Mann-Whitney U test was performed for the purpose of evaluating the data associated with the nerve palsy and the exposed doses. Cox regression analysis was performed using univariate analyses for diabetes mellitus (DM), hypertension (HT) and smoking. A p-value of 0.05 was used to determine the statistical significance of the results. All analyses were conducted using SPSS version 22.0 (IBM Corp., Armonk, NY, USA).

RESULTS

The patient cohort was 77.6% male and 22.4% female. Upon analysis of the comorbidities of the patients, it was observed that the percentage of smokers, diabetes, and hypertension were 44.8%, 11.2%, 23.2% respectively. The percentage of patients with T3-T4 tumors and tumors with positive lymph nodes is as follows: 34.4%, 81.6%. The number of patients presenting with disease-

Turk J Oncol 2025;40(4):257–263
doi: 10.5505/tjo.2025.4605

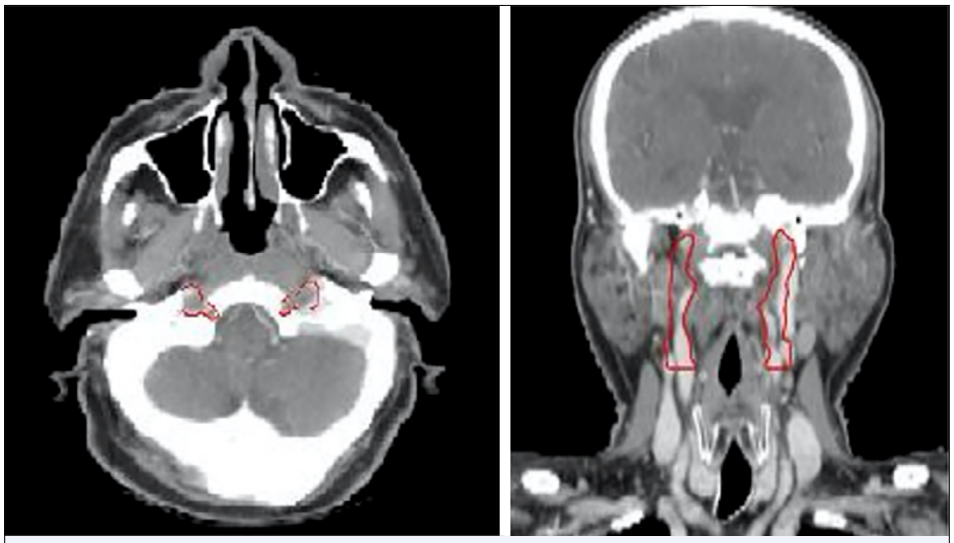

Fig. 1. Bilateral lower cranial nerves' volume on axial and coronal slices

related nerve palsies (with the exception of the CN 9–12 involvement) at the initiation of therapy was 12.

No patients developed grade 3–4 toxic effects associated with the therapeutic regimens. The overall median follow-up period for the sample was 44 months. The median follow-up period was 58 months (range 6–151) among the surviving patients. All patients evaluated for nerve palsy showed no evidence of locoregional recurrence. The number of patients who developed distant metastasis is as follows: 20 (16%). The median overall survival was observed to be 53 months. Thirty-eight patients (30.4%) died during the follow-up period. Of these patients,17 died of disease-related causes, while 21 died of non-disease-related causes. Nerve palsy was observed in 8 out of the 125 patients who underwent definitive IMRT. Five patients exhibited CN9–11 palsy while all patients who developed 9–11. nerve palsy also had CN12 palsy. The median time to the onset of nerve palsy was 75 months (range 14–133)

When the patients were evaluated in terms of clinical characteristics such as smoking, DM, HT, age and stage no statistically significance was found with lower cranial nerve palsy including CN12 and CN 9–11. The impact of smoking, DM, HT and age on the time interval following RT and the occurrence of lower cranial nerve palsy was evaluated. However, the findings revealed that these factors were not statistically significant.

A total of 250 lower cranial nerves were evaluated from a dosimetric perspective. The median CN 12 volume was 9.4 cc (range 4.2–17.4) while 9.9 cc (range 4.2–17.8) was for CN 9–11 unilaterally. There was no statistically significant with nerve palsy and dosimetric data. The dosimetric data of the patients and the incidence of nerve palsy are presented in Table 2.

The evaluation of the dosimetric characteristics of the patients in terms of time following RT and nerve palsy revealed no significant findings.

DISCUSSION

The prevalence of severe adverse effects that lead to a reduction in the quality of life of survivors has increased with the enhanced efficacy of cancer treatments. In the majority of cases, clinically asymptomatic conditions do not have an negative impact on the quality of life of patients.[8] However, depending on the irradiated tissue and administered dose, serious adverse effects, such as damage to the cranial nerves, may occur in the long term. As is widely acknowledged, RT carries out its effects on tissue through the generation of free oxygen radicals as a consequence of ionization. Radicals typically participate in physiological processes such as cell differentiation, proliferation, and inflammation. However, excessive production can result in physical and chemical damage, as well as pathological stress, when antioxidant defences are inadequate. As the concentration of radicals in the tissue increases with RT, direct toxicity for cells and a fibrotic process are initiated.[9] The short-term damage becomes chronic by overcoming the repair mechanisms of the tissue via the regenerative processes of stem cells. The delayed local damage initially attributable to microvascular damage in mature nerve tissue is defined as radiotherapy-induced neuropathy (RIN). This phenomenon is char-

Table 2 The dosimetric data of the patients and the incidence of nerve palsy

CN12 Palsy	Absent (n=185)		Present (n=7)		Mean difference	95% CI of the difference		p
	Mean	SD	Mean	SD		Lower	Upper	
D_{MAX}	72.19	5.330	72.97	4.087	-0.777	-4.568	3.014	0.803
D_{MEAN}	63.93	8.028	62.06	19.546	1.871	-16.208	19.950	0.153
D_{2CC}	67.59	7.132	69.93	7.184	-2.338	-8.991	4.315	0.196
D_{1CC}	69.00	6.557	71.40	4.540	-2.401	-6.618	1.816	0.265
$D_{0.5CC}$	69.94	6.157	71.77	4.246	-1.830	-5.774	2.114	0.379
Volume (Cc)	9.70	3.198	9.99	2.368	-0.283	-2.481	1.915	0.667
CN9-11 Palsy	**Absent (n=187)**		**Present (n=5)**		**Mean difference**	**95% CI of the Difference**		**p**
	Mean	**SD**	**Mean**	**SD**		**Lower**	**Upper**	
D_{MAX}	72.20	5.371	72.70	4.964	-0.497	-6.612	5.617	0.735
D_{MEAN}	63.84	7.915	59.80	22.654	4.040	-24.062	32.142	0.255
D_{2CC}	67.70	7.096	69.58	8.450	-1.875	-12.315	8.564	0.235
D_{1CC}	69.14	6.488	71.24	5.419	-2.101	-8.766	4.563	0.269
$D_{0.5CC}$	70.03	6.071	71.60	5.063	-1.567	-7.794	4.660	0.385
Volume (Cc)	10.41	5.578	10.36	1.765	0.047	-2.094	2.189	0.726

CN: Cranial nerve; CI: Confidence interval; D_{max}: Maximum dose; D_{mean}: Mean dose; D_{2cc}: The prescribed dose of 2 cc; D_{1cc}: The prescribed dose of 1 cc; $D_{0.5cc}$: The prescribed dose of 0.5 cc

acterized by a prolonged and consistent progression over an extended period. The initial phase is marked by the presence of chronic inflammation, often without overt symptoms, and is referred to as the "prefibrotic" phase. Subsequent to this initial phase, a fibrotic phase becomes apparent, in which there is an organized deposition of extracellular matrix. The final phase is a late fibroatrophic phase, distinguished by a lack of vascularization and retractile fibrosis. The delayed effects are a consequence of several factors, including fibroblast proliferation induced by cytokines such as TGF-β1, extracellular matrix deposition, direct axonal injury and demyelination, extensive fibrosis, and irregular neovascularisation in and around nerve trunks, and ischemia in the capillary networks supplying the nerves.[3] These factors result in an irregular structure in the irradiated volume. Consequently, the symptomatic nerve damage observed in the chronic phase is no longer reversible.

Although factors influencing RIN risk and severity are unclear, several RT-related factors are currently known: High total dose (>50 Gy to plexus, >60 Gy to cranial nerves),[10] high dose per fraction,[11] RT volume with large part of nerve fibres,[12] inhomogeneous high dose distribution,[13] high dose at hot spots, salvage RT of sites previously treated, intracavitary radium source[14] or after IORT boost. Cases of lower cranial nerve damage were observed to be in the range of 1 to 11 years in our study and we found that 6% of patients had lower cranial nerve palsy after treatment, compared with 5.1–8.7% in the literature.[2] The doses administered to the nerves are at the apex of the sigmoidal dose-effect curve, where the probability of complications is subject to significant variation with even a minor alteration in radiation dose. The lower cranial nerves are subjected to exceptionally high doses during nasopharyngeal treatment, primarily due to their close proximity to the high-dose target volume. It is established that peripheral nerves are more radioresistant than other neural structures.[3] Nevertheless, it is essential to minimise hot spots in these structures in order to reduce the incidence of neuropathy, which has a significant impact on quality of life. There is a lack of data in the existing literature regarding the doses to which these nerves are exposed. Chow and colleagues demonstrated that D_{max}, D_{mean}, D_{2cc}, D_{1cc} and $D_{0.5cc}$ were significantly associated with the development of nerve palsy. Of these, D_{1cc} was identified as the most effective factor for predicting radiation-induced hypoglossal nerve palsy. Furthermore, they established that a D_{1cc} limit of EQD2 <74 Gy could reduce the risk of this toxicity to less than 5%.[4] In our study, we implemented these established constraints for the analysis of nerves however no statistical significance was found between doses and RIN.

Turk J Oncol 2025;40(4):257–263
doi: 10.5505/tjo.2025.4605

A possible explanation for this discrepancy could be differences in treatment techniques and patient populations between studies. For instance, our cohort was treated exclusively with modern Intensity-Modulated Radiotherapy (IMRT) techniques, which are designed to deliver a highly conformal dose to the target volume while sparing surrounding critical structures, including the cranial nerves. This contrasts with some earlier studies that may have included patients treated with older, less conformal techniques, which could have resulted in higher and more heterogeneous doses to the nerves. Furthermore, differences in patient populations, such as varying tumor stages, comorbidities (e.g., diabetes mellitus and hypertension), or follow-up durations, could also influence the observed outcomes. While our study assessed the impact of these clinical factors, the limited number of palsy events prevented us from performing a robust statistical analysis to fully account for these confounding variables. This highlights the complexity of radiation-induced neuropathy and the need for larger, multi-institutional studies to identify consistent predictive factors.

In this study, T stage, N stage, age, gender, smoking, DM, and HT were assessed as predictive factors. The incidence of nerve damage increased in the advanced stages of the disease due to the increased RT volume irradiated. In literature, hypoglossal nerve damage was related to advanced T and N stages however in our study we did not find statistical significance.[4,15] In one of our patients, unilateral CN 12 damage was observed due to the retropharyngeal lymph node metastasis, although it was in the early stage (T1N1). Side effects of radiotherapy increased due to microvascular damage. When other predictive factors were evaluated HT, DM, and smoking were related to microvascular damage.[3] Although some studies demonstrated that this relations between radiotherapy side effects and microvascular damage for NPC patients, we did not show statistical significance.

Radiation-related neuropathy was an irreversible chronic side effect. Also, high dose steroids, hyperbaric oxygen, and pentoxifylline-tocopherol clodronate were recommended for the treatment of this complication. Pentoxifylline-tocopherol reduced radiotherapy-induced fibrosis whereas clodronate reduced inflammatory effects. In our clinical procedure, clodronate combined with pentoxifylline-tocopherol was prescribed for patients with RIN. However, we did not observe any recovery in our patients, only stabilization was observed. On the other hand, physical therapy and surgical treatment might be recommended for maintaining function.[3,15,16]

Limitation and Strengths

This study has several limitations, including its retrospective design and small sample size of only 8 palsy events. The small sample size likely rendered our study underpowered and precluded a meaningful multivariate analysis, which limits our ability to identify independent predictors of cranial nerve palsy. Furthermore, the lack of a standardized neurological evaluation protocol and a formal interobserver variability assessment for contouring are acknowledged limitations.

Despite these limitations, our study has key strengths: All treatments were administered with modern IMRT techniques, and patients with primary or lymph node recurrence were excluded, which ensures a more homogeneous cohort. Additionally, all patients were consistently followed up with MRI scans and physical examinations, providing robust clinical data for evaluation.

CONCLUSION

In this study, there was no correlation between radiotherapy-induced neuropathy in the lower cranial nerves and doses or other factors. However, the lower cranial nerves are vital for quality of life, and the nerves should be contoured.

Ethics Committee Approval: The study was approved by the Istanbul University-Cerrahpaşa Rectorate Ethics Committee (no: 1069261, date: 21/08/2024).

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: No AI technologies utilized.

Author Contributions: Concept – O.E.U.; Design – M.D., O.E.U.; Supervision – M.D.; Materials – I.F.D.; Data collection and/or processing – I.F.D., S.Y.; Data analysis and/or interpretation – M.D., I.F.D., G.C.; Literature search – S.C.K., O.E.U.; Writing – I.F.D., M.D.; Critical review – O.E.U.

Peer-review: Externally peer-reviewed.

REFERENCES

1. Lee N, Harris J, Garden AS, Straube W, Glisson B, Xia P, et al. Intensity-modulated radiation therapy with or without chemotherapy for nasopharyngeal carcinoma: Radiation therapy oncology group phase II trial 0225. J Clin Oncol 2009;27(22):3684–90.

2. Au K, Ngan RK, Ng AW, Poon DM, Ng W, Yuen K, et al. Treatment outcomes of nasopharyngeal carcinoma in modern era after intensity modulated radiotherapy (IMRT) in Hong Kong: A report of 3328 patients (HKNPCSG 1301 study). Oral Oncol 2018;77:16–21.
3. Delanian S, Lefaix JL, Pradat PF. Radiation-induced neuropathy in cancer survivors. Radiother Oncol 2012;105(3):273–82.
4. Chow JC, Cheung KM, Au KH, Zee BC, Lee J, Ngan RK, et al. Radiation-induced hypoglossal nerve palsy after definitive radiotherapy for nasopharyngeal carcinoma: Clinical predictors and dose–toxicity relationship. Radiother Oncol 2019;138:93–8.
5. Janssen S, Glanzmann C, Yousefi B, Loewenich K, Huber G, Schmid S, et al. Radiation-induced lower cranial nerve palsy in patients with head and neck carcinoma. Mol Clin Oncol 2015;3(4):811–6.
6. Dağdelen M, Çatal TK, Karaçam SÇ, Akovalı ES, Kanat S, Yıldırım HC, et al. Is a total dose of 54 Gy with radiochemotherapy sufficient for treatment of intermediate-risk volumes in nasopharyngeal cancer? Strahlenther Onkol 2024;200(5):409–17.
7. Mourad WF, Young BM, Young R, Blakaj DM, Ohri N, Shourbaji RA, et al. Clinical validation and applications for CT-based atlas for contouring the lower cranial nerves for head and neck cancer radiation therapy. Oral Oncol 2013;49(9):956–63.
8. Denham JW, Hauer-Jensen M. The radiotherapeutic injury–a complex 'wound'. Radiother Oncol 2002;63(2):129–45.
9. Delanian S, Lefaix JL. The radiation-induced fibroatrophic process: Therapeutic perspective via the antioxidant pathway. Radiother Oncol 2004;73(2):119–31.
10. Parsons JT, Bova FJ, Fitzgerald CR, Mendenhall WM, Million RR. Radiation optic neuropathy after megavoltage external-beam irradiation: Analysis of time-dose factors. Int J Radiat Oncol Biol Phys 1994;30(4):755–63.
11. Johansson S, Svensson H, Denekamp J. Dose response and latency for radiation-induced fibrosis, edema, and neuropathy in breast cancer patients. Int J Radiat Oncol Biol Phys 2002;52(5):1207–19.
12. Thomas JE, Cascino TL, Earle JD. Differential diagnosis between radiation and tumor plexopathy of the pelvis. Neurology 1985;35(1):1–.
13. Delanian S, Pradat PF. Posteriori conformal radiotherapy using three-dimensional dosimetric reconstitution in a survivor of adult-onset Hodgkin's disease for definitive diagnosis of lower motor neuron disease. J Clin Oncol 2010;28(30):e599–601.
14. Ashenhurst E, Quartey G, Starreveld A. Lumbo-sacral radiculopathy induced by radiation. Can J Neurol Sci 1977;4(4):259–63.
15. Luk YS, Shum JS, Sze HC, Chan LL, Ng W, Lee AW. Predictive factors and radiological features of radiation-induced cranial nerve palsy in patients with nasopharyngeal carcinoma following radical radiotherapy. Oral Oncol 2013;49(1):49–54.
16. McDowell LJ, Jacobson MC, Levin W. High-dose intravenous steroid regimen for radiation-induced hypoglossal nerve palsy. Head Neck 2017;39(2):E23–8.