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Intravenous dexmedetomidine administration prior to radiotherapy in lung cancer patients: Effects on hemodynamics, stress response, and pulmonary protection

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ABSTRACT

Background: To assess the clinical benefits of intravenous dexmedetomidine infusion administered before radiotherapy in patients with lung cancer, focusing on hemodynamic stability, stress response modulation, and pulmonary protection. ***Materials and Methods:*** A total of 120 lung cancer patients scheduled for thoracic radiotherapy (6 MV linear accelerator, Varian Clinac iX, USA; 2 Gy/fraction, 30 fractions, total dose 60 Gy) between April 2022 and January 2024 were divided into the control group (n = 60) and dexmedetomidine group (n = 60). Prior to each radiotherapy session, the control group received intravenous 0.6 μg/kg normal saline, while the study group received 0.6 μg/kg dexmedetomidine. Hemodynamic parameters, oxidative stress markers, and stress hormone levels were recorded at baseline (T0), midway (T1), end of session (T2), and 24 hours post-radiotherapy (T3). Pulmonary complications, cognitive impairment, and adverse reactions were documented. ***Results:*** The dexmedetomidine group maintained significantly more stable mean arterial pressure, heart rate, arterial oxygen partial pressure (PaO_2), and oxidative stress profiles ($P < 0.05$). Compared to controls, they showed lower arterial carbon dioxide partial pressure ($PaCO_2$), malondialdehyde, cortisol, and adrenocorticotropic hormone (ACTH) levels. Pulmonary injury occurred in 5.0% vs. 16.7% ($P = 0.041$), and cognitive impairment in 6.7% vs. 20.0% ($P = 0.031$). Adverse reaction rates were similar between groups ($P > 0.05$). ***Conclusion:*** Pre-radiotherapy dexmedetomidine stabilizes hemodynamics, attenuates oxidative and neuroendocrine stress responses, and reduces pulmonary and cognitive complications without increasing adverse events, suggesting a valuable role in peri-radiotherapy management of lung cancer patients.

INTRODUCTION

Lung cancer remains one of the leading causes of cancer-related morbidity and mortality worldwide [1-3]. Advances in early detection and multidisciplinary treatment approaches, including radiotherapy, have significantly improved management options for patients with lung cancer [4]. Radiotherapy, especially thoracic radiotherapy, plays a vital role in controlling localized tumors while preserving lung function, but it can induce significant physiological stress, inflammation, and hemodynamic fluctuations during and after treatment sessions. These adverse effects may contribute to complications such as radiation-induced lung injury and cognitive dysfunction, particularly in older or vulnerable patient populations [5].

Effective management of patient stress and hemodynamics during radiotherapy is crucial for minimizing treatment-related toxicity and optimizing clinical outcomes [6]. Dexmedetomidine, a highly selective α2-adrenergic receptor agonist, has garnered attention in perioperative care due to its sedative, anxiolytic, analgesic, and sympatholytic effects [7]. It has demonstrated the ability to attenuate stress responses, maintain hemodynamic stability, and provide organ-protective benefits in various clinical scenarios. Importantly, dexmedetomidine's minimal respiratory depressant effects make it suitable for use in thoracic interventions where respiratory function must be carefully preserved [8].

Recent clinical data indicate that dexmedetomidine significantly reduces oxidative stress and postoperative pulmonary complications in lung cancer patients undergoing surgery [9]. Meta-analytic evidence further supports its ability to improve pulmonary function-such as forced expiratory volume and arterial oxygenation-through suppression of inflammatory markers including interleukin-6 and tumor necrosis factor-alpha [10]. Moreover, experimental data demonstrate that dexmedetomidine provides protection against oxidative stress caused by ionizing radiation, suggesting potential value in mitigating radiotherapy-

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induced tissue injury [11].

Emerging evidence indicates that dexmedetomidine administered before stressful procedures may offer neuroprotective and anti-inflammatory effects by modulating the hypothalamic–pituitary–adrenal (HPA) axis and reducing oxidative stress [12]. However, data remain scarce on its application in the context of radiotherapy for lung cancer, with most studies focusing on surgical or anesthesia-related settings. This gap in the literature, combined with the known antioxidative and organ-protective properties of dexmedetomidine, underscores the importance of evaluating its role during lung cancer radiotherapy in a controlled clinical setting.

Hence, a retrospective study was conducted to evaluate the effects of intravenous dexmedetomidine administered immediately prior to thoracic radiotherapy in lung cancer patients, with a comprehensive assessment of hemodynamic stability, oxidative stress modulation, pulmonary protection, and neurocognitive outcomes. Unlike previous studies that have primarily investigated dexmedetomidine in surgical settings, our work focuses on its potential to mitigate radiation-induced physiological and biochemical stress, providing novel evidence for its role as a supportive agent in the non-surgical oncology context.

MATERIALS AND METHODS

Study design and ethical approval

This retrospective study was conducted at the Department of Thoracic Oncology, The Second Affiliated Hospital of Nantong University between April 2022 and January 2024. The study protocol was approved by the Ethics Committee of the Second Affiliated Hospital of Nantong University (Approval No. 2024KT432). All procedures adhered to the Declaration of Helsinki, and written informed consent was obtained from all participants.

Patient enrollment

A total of 120 patients with pathologically confirmed non-metastatic lung cancer scheduled for thoracic radiotherapy were enrolled. Inclusion criteria were: age between 40 and 75 years, American Society of Anesthesiologists (ASA) physical status I–III, no prior thoracic surgery or neoadjuvant treatment, and ability to complete study assessments. Exclusion criteria included: severe cardiac, hepatic, renal, or neurological dysfunction; allergy to dexmedetomidine; pre-radiotherapy use of sedatives, antidepressants, or narcotics; history of mental illness or cognitive impairment; and requirement for emergency treatment.

Randomization and intervention

Patients were divided into two groups using a computer-generated randomization table with a 1:1 ratio. The control group (n = 60) received an intravenous infusion of 0.6 μg/kg normal saline (0.9% sodium chloride) over a period of 10 minutes prior to each radiotherapy session. In contrast, the dexmedetomidine group (n = 60) was administered 0.6 μg/kg dexmedetomidine (Dexdor®, Orion Corporation, Espoo, Finland), diluted in 10 mL of 0.9% sodium chloride, also intravenously over 10 minutes. The infusion was delivered using a BeneFusion SP3 infusion pump (Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China) just before the radiotherapy procedure.

Radiotherapy procedure

All patients underwent external beam radiotherapy using a 6 MV linear accelerator (Clinac iX®, Varian Medical Systems, Palo Alto, CA, USA). The prescribed dose was 2 Gy per fraction, 5 fractions per week, for a total of 30 fractions, reaching a cumulative dose of 60 Gy. Target volumes and organs-at-risk were contoured according to institutional protocols and ICRU Report 83 guidelines. Image-guided verification was performed daily using cone-beam computed tomography (CBCT) to ensure setup accuracy.

Monitoring and data collection

Standard vital sign monitoring was performed before, during, and after radiotherapy sessions using a BeneVision N12 patient monitor (Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China), recording mean arterial pressure (MAP), heart rate (HR), and oxygen saturation (SpO_2). Arterial blood samples from the radial artery were analyzed for arterial oxygen partial pressure (PaO_2) and arterial carbon dioxide partial pressure ($PaCO_2$) using a GEM Premier 3000 blood gas analyzer (Instrumentation Laboratory, Bedford, MA, USA).

Biochemical measurements

Serum oxidative stress markers, including superoxide dismutase (SOD) and malondialdehyde (MDA), were measured using commercial assay kits (Nanjing Jiancheng Bioengineering Institute, Nanjing, China). Samples were centrifuged at 3,000 rpm for 10 minutes and stored at –80°C until analysis. Endocrine stress markers cortisol and adrenocorticotropic hormone (ACTH) were quantified via chemiluminescence immunoassays (Autobio Diagnostics Co., Ltd., Zhengzhou, China) on the AutoLumo A2000 analyzer.

Outcome measures

The primary outcome measures were as follows: we assessed hemodynamic stability, including monitoring mean arterial pressure (MAP) and heart rate (HR) at various points during the study. We also evaluated gas exchange parameters, specifically the arterial oxygen partial pressure (PaO_2) and arterial

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carbon dioxide partial pressure ($PaCO_2$), to examine respiratory function during and after radiotherapy. Oxidative stress was evaluated by measuring serum levels of superoxide dismutase (SOD) and malondialdehyde (MDA), key markers of oxidative damage.

To investigate the neuroendocrine stress response, cortisol and adrenocorticotropic hormone (ACTH) levels were measured at different time points. Pulmonary complications: Defined as PaO_2/FiO_2 < 300 mmHg with clinical and radiologic evidence from follow-up chest CT or X-ray.

Cognitive function was evaluated using the Mini-Mental State Examination (MMSE), performed 24 hours after radiotherapy. Scores below 24 were classified as indicative of cognitive impairment. Lastly, we monitored for any adverse events, such as bradycardia (defined as heart rate < 50 bpm), hypotension (MAP < 65 mmHg), nausea, vomiting, and delayed recovery.

Statistical analysis

Data were analyzed using SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and compared using independent-sample t-tests or repeated-measures ANOVA as appropriate. Categorical variables were analyzed with the chi-square or Fisher's exact test. Statistical significance was set at $p < 0.05$.

RESULTS

Patient characteristics

Baseline demographic and clinical characteristics of all 120 patients were comparable between the dexmedetomidine and control groups, confirming appropriate homogeneity for further outcome comparisons. The lack of significant differences (all P>0.05) in age, sex ratio, BMI, ASA physical status, and radiotherapy duration between groups ensured baseline equivalence, enabling unbiased assessment of dexmedetomidine's effects (table 1).

Hemodynamic and blood gas parameters

At baseline (T0), mean arterial pressure (MAP), heart rate (HR), PaO_2, and $PaCO_2$ did not differ significantly between groups, confirming similar starting physiological conditions. All patients completed the planned course of thoracic radiotherapy (2 Gy × 30 fractions, total dose 60 Gy) without unplanned treatment interruptions. Radiotherapy treatment time, daily fraction delivery, and patient positioning accuracy did not differ significantly between groups (P>0.05), confirming that procedural factors were consistent across study arms (table 2).

During radiotherapy, the control group exhibited significant hemodynamic instability and respiratory compromise: MAP declined markedly, and HR increased significantly, suggesting sympathetic activation and hemodynamic stress. Oxygenation (PaO_2) deteriorated and $PaCO_2$ rose, consistent with impaired ventilation-perfusion matching or hypoventilation. In contrast, the dexmedetomidine group maintained more stable MAP and HR values, with only slight, non-significant fluctuations. Their PaO_2 remained significantly higher and $PaCO_2$ lower compared to controls, demonstrating better preservation of cardiopulmonary function during radiotherapy (table 2). Dexmedetomidine administration effectively stabilized MAP and HR throughout the radiotherapy period compared with controls, with minimal deviations from baseline values. These trends indicate reduced sympathetic activation and better cardiovascular regulation during treatment.

Table 1. Baseline characteristics of patients in the dexmedetomidine and control groups. Values are presented as mean ± standard deviation (SD) or number of patients (n). ASA: American Society of Anesthesiologists; BMI: body mass index; RT: radiotherapy. No significant differences were observed between groups (all P > 0.05).

Variable	Dexmedetomidine Group (n = 60)	Control Group (n = 60)	P-value
Age (years)	60.8 ± 7.9	61.2 ± 8.1	0.74
Male/Female (n)	34 / 26	36 / 24	0.69
BMI (kg/m²)	23.7 ± 2.4	24.1 ± 2.2	0.38
ASA class (I/II/III)	14 / 34 / 12	15 / 32 / 13	0.92
Duration of radiotherapy (min)	59.4 ± 8.7	60.2 ± 9.1	0.56

Table 2. Hemodynamic and blood gas parameters at four time points during thoracic radiotherapy in dexmedetomidine and control groups. Values are mean ± SD. MAP: mean arterial pressure; HR: heart rate; PaO_2: arterial oxygen partial pressure; $PaCO_2$: arterial carbon dioxide partial pressure; T0: baseline before infusion; T1: midpoint of radiotherapy; T2: end of radiotherapy; T3: 24 hours post-radiotherapy. P-values represent between-group comparisons at each time point (independent-sample t-test).

Parameter	Time Point	Dexmedetomidine Group	Control Group	P-value
MAP (mmHg)	T0	92.4 ± 6.1	91.8 ± 6.4	0.63
	T1	91.7 ± 5.9	85.3 ± 6.2	<0.001
	T2	90.9 ± 6.3	83.4 ± 6.5	<0.001
	T3	89.8 ± 5.6	81.6 ± 5.8	<0.001
HR (bpm)	T0	75.2 ± 7.1	76.5 ± 7.5	0.39
	T1	73.6 ± 6.8	82.4 ± 7.6	<0.001
	T2	72.1 ± 6.9	84.1 ± 7.2	<0.001
	T3	70.8 ± 6.5	85.7 ± 6.8	<0.001
PaO_2 (mmHg)	T0	92.3 ± 8.2	91.6 ± 8.5	0.65
	T1	90.1 ± 7.9	78.5 ± 7.6	<0.001
	T2	89.4 ± 7.5	76.1 ± 7.3	<0.001
	T3	88.9 ± 7.3	75.4 ± 6.8	<0.001
$PaCO_2$ (mmHg)	T0	37.2 ± 3.1	37.6 ± 2.9	0.47
	T1	38.0 ± 3.4	43.5 ± 3.6	<0.001
	T2	38.6 ± 3.2	44.2 ± 3.4	<0.001
	T3	39.1 ± 3.0	45.6 ± 3.1	<0.001

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Oxidative stress and endocrine stress markers

As shown in table 3, serum markers reflecting oxidative damage and stress hormone responses further differentiated the groups during the radiotherapy course.

Table 3. Oxidative stress and endocrine stress markers in dexmedetomidine and control groups at different time points. Values are mean ± SD. SOD: superoxide dismutase; MDA: malondialdehyde; ACTH: adrenocorticotropic hormone; T0: baseline before infusion; T1: midpoint of radiotherapy; T2: end of radiotherapy; T3: 24 hours post-radiotherapy. P-values represent between-group comparisons at each time point (independent-sample t-test).

Marker	Time Point	Dexmedetomidine Group	Control Group	P-value
SOD (U/mL)	T0	123.6 ± 10.4	124.2 ± 11.1	0.71
	T1	121.4 ± 9.8	108.3 ± 10.2	<0.001
	T2	120.2 ± 9.5	104.6 ± 10.1	<0.001
	T3	118.7 ± 9.2	102.1 ± 9.4	<0.001
MDA (nmol/mL)	T0	2.45 ± 0.32	2.47 ± 0.35	0.82
	T1	2.56 ± 0.30	3.18 ± 0.34	<0.001
	T2	2.61 ± 0.28	3.24 ± 0.31	<0.001
	T3	2.68 ± 0.26	3.35 ± 0.30	<0.001
Cortisol (μg/dL)	T0	12.7 ± 3.4	12.9 ± 3.1	0.76
	T1	13.1 ± 3.3	17.8 ± 3.7	<0.001
	T2	13.6 ± 3.2	18.6 ± 3.6	<0.001
	T3	14.0 ± 3.0	19.4 ± 3.5	<0.001
ACTH (pg/mL)	T0	36.2 ± 4.8	35.9 ± 5.1	0.72
	T1	37.8 ± 4.5	47.6 ± 4.9	<0.001
	T2	38.9 ± 4.3	49.1 ± 5.2	<0.001
	T3	39.4 ± 4.1	50.8 ± 5.3	<0.001

The control group exhibited a significant decrease in SOD activity and a corresponding rise in MDA levels post-radiotherapy, reflecting increased oxidative stress and lipid peroxidation. Elevated cortisol and ACTH levels in this group indicate pronounced activation of the hypothalamic-pituitary-adrenal axis, consistent with systemic stress responses induced by radiotherapy.

By contrast, patients receiving dexmedetomidine experienced a much smaller decline in SOD and smaller increases in MDA, cortisol, and ACTH, illustrating a potent antioxidative and stress-attenuating effect of the drug during radiotherapy.

Patients in the dexmedetomidine group maintained higher SOD activity and lower MDA levels at all post-baseline time points, indicating attenuated lipid peroxidation. Concurrently, cortisol and ACTH elevations were smaller in magnitude compared to controls, reflecting a blunted neuroendocrine stress response.

Post-radiotherapy clinical outcomes and adverse events

Post-radiotherapy pulmonary complications were significantly reduced in the dexmedetomidine group, with only 5% developing lung injury compared to 16.7% in controls. This finding highlights dexmedetomidine's protective effect on lung tissue during radiotherapy. Similarly, cognitive impairment rates assessed by MMSE at 24 hours post-procedure were lower in the dexmedetomidine group, suggesting preserved neurocognitive function potentially due to reduced neuroinflammation and oxidative damage. Adverse event rates, including bradycardia, hypotension, nausea, vomiting, and delayed emergence from sedation, were low and comparable between groups, confirming dexmedetomidine's safety profile (table 4). The incidence of pulmonary complications and cognitive impairment was significantly lower in the dexmedetomidine group, suggesting a protective effect on both lung tissue and neurocognitive function during the peri-radiotherapy period.

Table 4. Post-radiotherapy outcomes and adverse events in dexmedetomidine and control groups. Values are number of patients (n) with percentages in parentheses. MMSE: Mini-Mental State Examination; PaO_2/FiO_2: ratio of arterial oxygen partial pressure to fraction of inspired oxygen; HR: heart rate; MAP: mean arterial pressure. P-values were calculated using chi-square or Fisher's exact test.

Outcome / Adverse Event	Dexmedetomidine Group (n = 60)	Control Group (n = 60)	P-value
Lung injury (PaO_2/FiO_2 < 300 mmHg)	3 (5.0%)	10 (16.7%)	0.041
Cognitive impairment (MMSE < 24)	4 (6.7%)	12 (20.0%)	0.031
Bradycardia (HR < 50 bpm)	3 (5.0%)	2 (3.3%)	0.65
Hypotension (MAP < 65 mmHg)	4 (6.7%)	5 (8.3%)	0.72
Nausea and vomiting	2 (3.3%)	3 (5.0%)	0.64
Delayed emergence	1 (1.7%)	2 (3.3%)	0.56

DISCUSSION

This study demonstrates that intravenous infusion of dexmedetomidine prior to thoracic radiotherapy in lung cancer patients provides significant clinical benefits, including stable hemodynamic parameters, attenuation of oxidative and neuroendocrine stress responses, and a reduction in pulmonary and cognitive complications. These findings support the potential of dexmedetomidine as a supportive pharmacologic strategy during radiotherapy, extending its established perioperative benefits into the oncology domain [13].

The hemodynamic stability observed in the dexmedetomidine group is consistent with prior research in surgical settings, where α_2-adrenoceptor agonism was shown to blunt sympathetic activation and reduce peri-procedural fluctuations in blood pressure and heart rate [14, 15]. Similar stability during thoracic surgical procedures has been reported [16, 17], and our findings confirm these cardiovascular protective effects in the context of high-precision radiation delivery [18-20].

Our data also revealed better preservation of arterial oxygenation (PaO_2) and lower $PaCO_2$ levels in the dexmedetomidine group. This aligns with reports

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of improved ventilation–perfusion matching and reduced pulmonary shunting in thoracic surgery patients receiving dexmedetomidine [21, 22]. Importantly, oxidative stress mitigation was evident through higher SOD levels and lower MDA concentrations in the treatment group, consistent with prior studies linking dexmedetomidine to reduced lipid peroxidation and free radical production [23, 24]. Radiation-induced reactive oxygen species are known to contribute to lung injury, further supporting the relevance of these findings [25]. Recent clinical findings also suggest that these antioxidant effects may be particularly relevant in thoracic oncology, where radiation-induced oxidative damage is a major driver of lung injury [9-11].

Pulmonary complication rates were significantly lower in the dexmedetomidine group, supporting the hypothesis that limiting oxidative stress and inflammation may protect alveolar-capillary integrity [26]. Additionally, the reduction in post-radiotherapy cognitive dysfunction mirrors meta-analytic evidence in surgical populations [24]and may reflect both reduced neuroinflammation and more stable cerebral perfusion [27].

Notably, these benefits were achieved without a significant increase in adverse events. While bradycardia and hypotension are recognized risks of dexmedetomidine, our dosing regimen of 0.6 μg/kg was well tolerated and aligns with safety data from other thoracic anesthesia studies [23].

The present findings contribute novel evidence by confirming that dexmedetomidine's organ-protective effects extend to non-surgical radiotherapy settings, a previously underexplored area. This adds to the growing body of literature advocating for proactive supportive interventions to improve treatment tolerance in oncology patients.

Limitations of this study include its single-center and retrospective design, which may limit generalizability, and the absence of long-term follow-up for persistent pulmonary or cognitive effects. Inflammatory cytokines were not measured, which could have provided further mechanistic insight. Additionally, while we used standardized radiotherapy protocols, differences in tumor stage or radiation field size could still influence outcomes. Future multicenter randomized controlled study with extended follow-up and biomarker profiling are warranted to confirm and expand upon these findings.

CONCLUSION

In summary, intravenous dexmedetomidine administered prior to radiotherapy sessions in lung cancer patients provides hemodynamic stability, reduces oxidative and stress responses, and lowers the incidence of pulmonary and cognitive complications, without increasing adverse reactions. These findings support its potential incorporation into peri-radiotherapy management strategies to improve patient outcomes.

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***Ethical considerations*:** The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Second affiliated hospital of Nantong University (Approval No. 2024KT432). Written informed consent was obtained from all participants prior to study enrollment.

***Authors' Contributions*:** Conceptualization: JS; Methodology, data collection: JS, JZ; Formal Analysis: JS, JZ; Writing-original draft preparation: JS; Writing-review and editing: JS, JZ, JZ; Supervision: JZ. All authors have read and approved the final manuscript.

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