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Original Research Article

Cisplatin-etoposide versus paclitaxel-carboplatin along with thoracic radiotherapy in unresectable locally advanced non-small cell lung cancer: a comparison of treatment response

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ABSTRACT

Background: Although many chemotherapy regimens are used concurrently with radiotherapy in locally advanced non-small cell lung cancer (NSCLC), the ideal chemotherapy regimen is yet to be determined. The purpose of this study was to compare the therapeutic response of cisplatin-etoposide (EP) and paclitaxel-carboplatin (PC) combined with thoracic radiation in unresectable locally advanced NSCLC.
Methods: A quasi-experimental study was conducted from October 2020 to September 2021 at two centers in Dhaka. Patients with unresectable, locally advanced, histology proven NSCLC were enrolled and distributed equally in two arms. Patients underwent 60 Gy in 2 Gy daily fractions, five days/week, for six weeks of thoracic radiotherapy with either cisplatin 50 mg/m^2 and etoposide 50 mg/m^2/day (arm A), or carboplatin (AUC-2) and paclitaxel (45 mg/m^2) (arm B). All the patients were evaluated before, during, and after the completion of the treatment. Follow ups were done at 6 weeks, 12 weeks and 24 weeks following the completion of treatment.
Results: Seventy-four patients were distributed equally into two arms. After 6 weeks 3 (8.11%) patients had complete response (CR), 28 (75.68%) had partial response (PR), and 6 (16.22%) had stable disease (SD) in arm-A. In arm-B, 1 (2.71%), 24 (64.86%), 12 (32.43%) patients had CR, PR and SD respectively. In either arm none had progressive disease (PD). After 24 weeks, 5 (13.51%) versus 2 (5.41%) patients had CR, 15 (40.54%) versus 16 (43.24%) had SD, and 4 (10.81%) versus 6 (16.21%) had PD in arm-A and arm-B respectively. PR was 13 (35.14%) in both arms. But these differences were not significant (p>0.05).
Conclusions: In unresectable locally advanced non-small cell lung cancer, the short-term treatment response of a cisplatin-etoposide regimen given with concurrent radiation therapy is comparable to that of a paclitaxel-carboplatin regimen.

Keywords: Carboplatin and paclitaxel, Cisplatin and etoposide, Concurrent chemoradiation, Locally advanced, Lung cancer, Non-small cell lung cancer

INTRODUCTION

Lung cancer has a global incidence of 11.4%, accounting for 18% of all cancers. This make it the second most frequent malignancy, and the main cause of cancer-related fatalities, worldwide.[1] Although such statistics are not currently available for Bangladesh, according to the Hospital Based Cancer Registry report (2015-2017) by the NICR and H, lung was the leading site of cancers (5887, 16.6%).[2]

The most important modifiable risk factor for lung cancer is cigarette smoking. According to estimates, heavy smokers had at least a 20-times risk of developing lung cancer, whereas the average male smoker had a 9-10 fold risk.[3] Other risk factors include poor diet, genetic predisposition, and occupational/environmental exposure to carcinogens like arsenic, asbestos, beryllium, cadmium, chromium, nickel, radon, and vinyl chloride; asbestos being the most common.[4,5] For example, low serum concentrations of antioxidants, such as vitamins A, C, and E, have been associated with the development of lung cancer.[6]

When there is invasion of adjacent structures and/or lymph node metastases it is not amenable for potentially curative resection.[7] Around 35% of NSCLC present with a locally advanced disease and which in most cases are unresectable.[8] Patients with locally advanced NSCLC include those with unresectable stage II to III disease and selected patients with stage II to III disease who are surgical candidates.[9]

Regarding management of locally advanced NSCLC, chemoradiation has survival benefit over radiotherapy alone but the optimal chemoradiation regimen is a work in progress.[10,11] Studies have shown better local response and survival benefit in concurrent administrations of the chemotherapy and radiotherapy compared to the sequential administration of these therapies although at the expense of greater acute toxicities.[8] Various combination regimens have been trialed with concurrent RT in phase III randomized studies including mitomycin, vindesine, and cisplatin, etoposide and cisplatin (PE), vinblastine and cisplatin, paclitaxel and carboplatin (PC), vinorelbine and cisplatin etc.[7,12-14] However, very few randomized phase III trials have directly compared the different CCRT regimens.[15]

Etoposide-cisplatin (EP) and paclitaxel-carboplatin (PC) are two very widely used regimens. A recent randomized trial showed better response rate as well as a significant benefit in overall survival with EP arm (28.0% vs 19.7%).[15] On the other hand, there have been some meta-analyses and systematic reviews which showed comparable outcomes in terms of response and survival in both while the toxicity profile favored the PC regimen.[16]

Although every oncology institution has standard protocols, particularly in this region of the world, there is a lack of conclusive evidence to back up their choices. Therefore, the aim of this study was to compare clinical outcome of these two drug regimens along with radiotherapy which may help in future treatment decisions.

METHODS

This quasi-experimental study was conducted from October 2020 to September 2021 in the department of clinical oncology, BSMMU and department of radiation oncology, NICR and H, Dhaka. Total 74 patients were enrolled in the study with the inclusion criteria of having histologically proven unresectable locally advanced NSCLC of stage IIIA, IIIB and IIIC (AJCC 8th edition). Patients with ECOG (eastern co-operative oncology group) performance status score of more than 2, prior radiotherapy to chest, or serious concomitant medical illness were excluded. Ethical standards outlined in the Helsinki declaration were strictly followed. Approval was secured from respective institutions. Before collecting data informed written consent was obtained from each patient. A structured data collection form was used to collect data by face-to-face interviews with patients, and from their history and investigation reports.

Patients were distributed equally into two arms using purposive sampling. All patients received thoracic radiotherapy of 60Gy in 2Gy daily fractions, 5 fractions a week, for 6 weeks using 6 MV photon from LINAC machine with concurrent chemotherapy. Patients in arm-A received intravenous cisplatin 50 mg/m^2 on days 1, 8, 29, and 36, plus etoposide 50 mg/m^2/day on days 1-5 and 29-33. Arm-B patients received intravenous carboplatin (AUC-2) and paclitaxel administered on day 1, weekly, over a 6-week period concurrently with radiotherapy.[15,17,18]

All the patients were evaluated before, during, and after the completion of the treatment. Follow ups were done at 6 weeks, 12 weeks and 24 weeks following the completion of treatment. Treatment response was measured during follow up by clinical examination (general physical examination including regional lymph node examination and chest examination) and relevant investigations like chest radiogram and CT scan. Treatment response was evaluated according to the RECIST criteria version 1.1. ECOG (Eastern Cooperative Oncology Group) scale was used to evaluate performance status of patients before and after completion of therapy. The data were analyzed using the SPSS software program (North Castle, NY, USA) for Windows, version 25. A p value of <0.05 was considered as statistically significant.

RESULTS

A total of 74 patients were enrolled in this study. Table 1 summarizes the baseline characteristics of patients in the two arms.

The patients were aged 45 to 70 years and were mostly men (77%). Their mean age at diagnosis was 61.43 years in arm A and 61.32 years in arm-B. Histologically both adenocarcinomas and squamous cell carcinomas were almost equally distributed in both arms. Arm-A patients had more adenocarcinomas than arm-B. The most prevalent disease stage was IIIB (52.7%). There were no statistically significant differences in terms of age, gender, body surface area (BSA), performance status, or stage between the two arms (p value >0.05). Most

patients in both arms had an ECOG performance rating of 0 to 1 (64.9% in arm-A and 56.8% in arm-B). As per inclusion criteria no patients with metastasis (M1) or ECOG performance status 3 were included in the study.

Table 1: Distribution of patients according to the baseline character.

Variable	Arm A (n=37) (%)	Arm B (n=37) (%)	Total (n=74) (%)	P value*
Age (years)				
Mean (SD)	61.43 (±5.87)	61.32 (±5.84)	61.38 (±5.82)	0.937
Range	25 (45-70)	20 (49-69)	25 (45-70)	
Gender				
Male	30 (81.1)	27 (73)	57 (77)	0.581
Female	7 (18.9)	10 (27)	17 (23)	
Body surface area (m^2)				
Mean (SD)	1.615 (±0.1185)	1.603 (±0.1138)	1.604 (±0.1154)	0.944
Range	0.46 (1.35-1.81)	0.44 (1.36-1.80)	0.44 (1.36-1.80)	
Histology				
Adenocarcinoma	22 (59.46)	18 (48.64)	40 (54.05)	0.351
Squamous cell carcinoma	15 (40.54)	19 (51.36)	34 (45.95)	
Stage (AJCC 8th edition)				
IIIA	19 (51.35)	10 (27.03)	29 (39.19)	0.095
IIIB	16 (43.24)	23 (62.16)	39 (52.70)	
IIIC	2 (5.41)	4 (10.81)	6 (8.11)	
ECOG performance status				
0-1	24 (64.86)	21 (56.76)	45 (60.8)	0.634
2	13 (35.1)	16 (43.2)	29 (39.2)	
3	0	0	0	

*Calculated using Student's t test or chi-square (χ^2) test
AJCC = American Joint Committee on Cancer, ECOG = Eastern Cooperative Oncology Group

Table 2: Clinical response at 1st follow-up (at week 6) after completion of treatment.

Response	Arm A (n=37) (%)	Arm B (n=37) (%)	Overall (n=74) (%)	P value*
Complete response	3 (8.11)	1 (2.71)	4 (5.41)	0.223
Partial response	28 (75.68)	24 (64.86)	52 (70.27)	
Stable disease	6 (16.22)	12 (32.43)	18 (24.32)	
Progressive disease	0	0	0	

*Fisher's exact test

Table 3: Clinical response at 2nd follow-up (at week 12) after completion of treatment.

Response	Arm A (n=37) (%)	Arm B (n=37) (%)	Overall (n=74) (%)	P value*
Complete response	5 (13.51)	2 (5.41)	7 (9.46)	0.076
Partial response	25 (67.57)	18 (48.65)	43 (58.11)	
Stable disease	5 (13.51)	14 (37.84)	19 (25.68)	
Progressive disease	2 (5.41)	3 (8.11)	5 (6.76)	

*Fisher's exact test

Table 4: Clinical response at 3rd follow-up (at week 24) after completion of treatment.

Response	Arm A (n=37) (%)	Arm B (n=37) (%)	Overall (n=74) (%)	P value*
Complete response	5 (13.51)	2 (5.41)	7 (9.46)	0.669
Partial response	13 (35.14)	13 (35.14)	26 (47.30)	
Stable disease	15 (40.54)	16 (43.24)	31 (29.73)	
Progressive disease	4 (10.81)	6 (16.21)	10 (13.51)	

*Fisher's exact test

Tobacco smoking was quite common among the patients (50 or 67.57%). It is shown in Figure 1 that 26 patients (70.27%) in arm-A and 24 (64.86%) in arm-B were smokers. This was followed by occupational exposure in overall 10 (13.52%) patients; 16% in arm-A and 11% in arm-B. Others risk factors like indoor air pollution (e.g., firewood user) or history of chronic obstructive lung disease (COPD) were not very frequently observed. Observed differences between the arms were not significant (p>0.05).

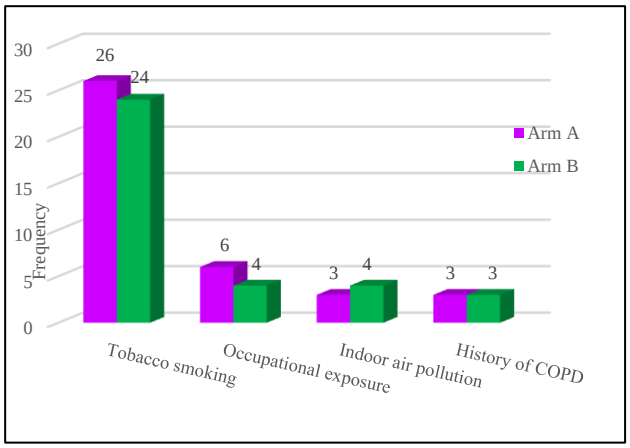

Figure 1: Distribution of the patients according to risk factors.

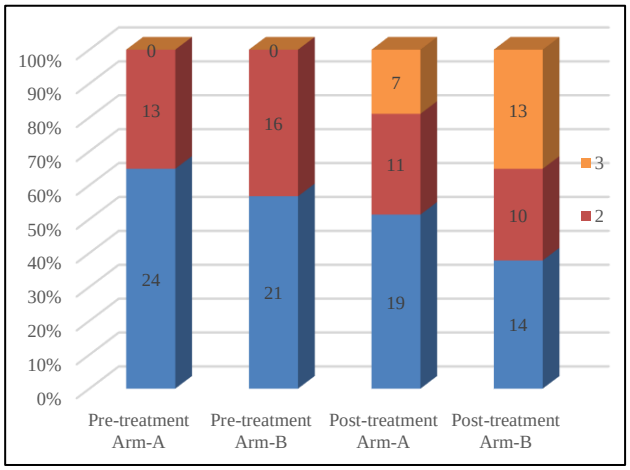

Figure 2: Comparison of ECOG performance status before and after treatment between two arms.

Figure 2 compares both pre-treatment and post-treatment ECOG performance status between the arms. After completion of CCRT, performance status deteriorated in both arms as expected. More patients in arm-B deteriorated. After completion of the treatment, 7 (18.92%) patients in arm-A and 13 (35.13%) patients in arm-B had ECOG score 3. Patients with ECOG status 2 reduced from 13 (35.1%) to 11 (29.73%) in arm-A and from 16 (43.2%) to 10 (27.03%) in arm-B. While no patients had an ECOG 3 score, after treatment 7 (18.92%) in arm-A and 13 (35.13%) in arm-B had ECOG score 3. However, the difference was not statistically significant (p=0.272).

Treatment response

Treatment response was the main end point of the study. According to RECIST criteria four types responses were recorded, namely, complete response (CR), partial response (PR), stable disease (SD), and progressive disease (PD). Figure 3 depicts the responses as were found during the three follow ups over a 24-week period following treatment completion.

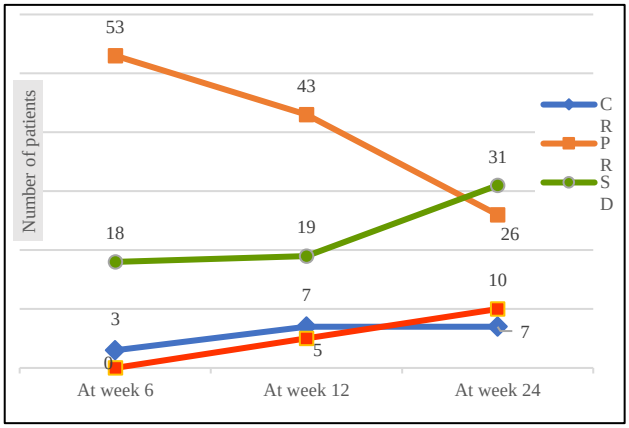

Figure 3: Overall treatment response among patients.

Tables 2, 3, and 4 summarize treatment response as was recorded after 6 weeks, 12 weeks, and 24 weeks of treatment completion respectively. Patients mostly had partial response in the first follow up. But by the third follow up, number of patients with partial response decreased whereas that with stable disease increased. Number of patients with complete response remained somewhat similar during the follow up period. On the other hand, number of progressive diseases increased. Although arm-A showed better responses in the first two follow ups, observed differences in the two arms were not significant (p>0.05 for all).

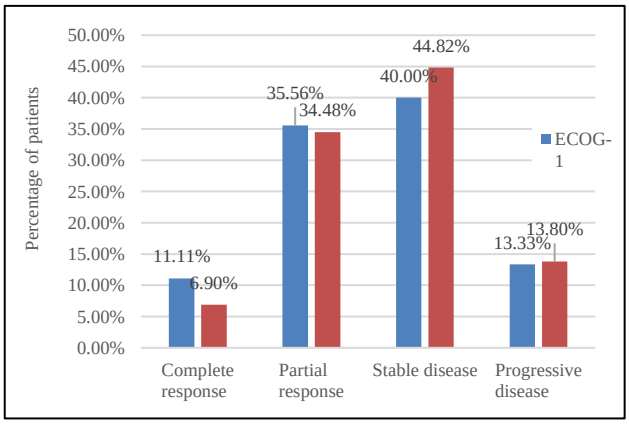

Figure 4: Clinical response according to pre-treatment ECOG performance status.

Figure 4 shows clinical response according to pre-treatment ECOG performance status for arms. Here, overall number of patients with ECOG PS 0-1 was 45 and ECOG PS-2 was 29. CR was seen in 5 (11.11%) patients with ECOG PS-1 and in 2 (6.9%) patients with ECOG PS-2. PR was seen in 16 (35.56%) patients with ECOG PS-1 and 10 (34.48%) patients with ECOG PS-2. More percentage of patients with ECOG PS-2 showed stable disease (44.82% versus 40.0%) and progressive disease (13.82% versus 13.33%) compared to patients with ECOG PS-1. The difference was not significant (p=0.932).

Overall, there were 50 smokers and 24 non-smokers, and responses were compared between them. In smokers 4 (8.0%) patients showed CR while 16 (32.0%) patients showed PR. And in non-smokers, 3 (12.5%) patients showed CR, while 10 (41.67%) patients showed PR. Eight (16.0%) smokers ultimately had progressive disease compared to non-smokers [2 (8.33%)]. The difference was, however, not statistically significant (p=0.663).

DISCUSSION

Total 74 patients with histologically proven locally advanced NSCLC were enrolled in this study. Their mean age was 61.38±5.82 years. Among them 77% were male and 23% were female. These demographics were similar to the Hospital Cancer Registry Report 2015-2017, NICR and H where male and female was 85% and 15% respectively.[2] This finding was also similar to that of Bi et al.[19]

Smoking, which is established as a major independent risk factor for lung cancer, was commonly observed in this study. Halperin et al highlighted that around 80-90% lung cancer cases are attributable to voluntary/ involuntary exposure to cigarette smoking.[11] We also found that most of the patients in both the arms were smokers (67.57%). Arm-A had 26 (70.27%) patients and arm-B had 24 (64.86%) patients, who were smokers, and this mainly comprised of male patients. This finding is also supported by Senan et al where they found 75.75% of patients who presented with NSCLC were smokers.[20] Various other risk factors were also observed such as tobacco smoking, occupational exposure, indoor air pollution and, chronic obstructive lung disease. But those were much less common.

At presentation, most of the patients that were included in the study had ECOG performance status of 0-1 (60.81%). Fewer patients were attributed to ECOG 2. In arm-A, 24 (64.86%) patients and in arm-B, 21 (56.75%) patients had ECOG 0-1. Patients with ECOG>2 were not included in this study owing to their decreased capacity to tolerate the treatment. After completion of treatment, performance status deteriorated in both arms as expected. More patients in arm-B deteriorated. ECOG 0 to 2 reduced while ECOG 3 increased. However, the differences were not statistically significant. This deterioration was due to various toxicities like nausea/vomiting, esophagitis causing dysphagia and ultimately inadequate nutrition, and radiation pneumonitis.

The main end-point of our study was clinical response. Six weeks following completion of treatment, 1st Follow up was done. In arm-A, 3 (8.11%) patients had complete response (CR), 28 (75.68%) patients had partial response (PR), and 6 (16.22%) patients had stable disease (SD). Whereas, in arm-B, 1 (2.71%) patient showed CR, 24 (64.86%) patients showed PR and, 12 (32.43%) patients showed SD. Overall, 4 (5.41%) patients showed CR, 52 (70.27%) showed PR and, 18 (24.32%) showed SD. None showed progressive disease (PD). Although, arm-A showed arithmetically better response compared to arm-B, it was not statistically significant (p value =0.223).

After 12 weeks of treatment completion, 2nd follow up was conducted. CR was observed in 5 (13.51%) and 2 (5.41%) patients in arm-A and arm-B respectively. Likewise, 25 (67.57%) patients in arm-A and 18 (48.65%) patients in arm-B showed PR. We observed SD in 5 (13.51%) patients in arm-A and 14 (37.84%) in arm-B. Two (5.40%) patients in arm-A and 3 (8.11%) in arm-B developed PD. Here, 2 patients in arm-A and 1 patient in arm-B, previously thought to have PR in 1st follow up, showed CR later in 2nd follow up. The reason for this may be that the imaging findings had post-radiation inflammatory changes which were misinterpreted as residual tumor. A patient with progressive disease had loco-regional progression while rest 4 had distant metastases; 3 had bone (vertebral) metastases and 1 (from Arm B) had disease spread to the brain. No statistical difference was found (p value =0.066).

On 3rd and final follow up, after 24 weeks of treatment completion, 5 (13.51%) patients in arm A and 2 (5.41%) in arm B maintained CR; 13 (35.14%) patients showed PR in both arms; 15 (40.54%) patients in arm A and 16 (43.24%) patients in arm B had stable disease. Arm A had 4 (10.81%) and arm B had 6 (16.21%) patients with progressive disease. The differences were not statistically significant (p=0.669). Similar observation was found in a randomized trial by Liang et al where they observed overall response rate of 73.7% in the EP arm (arm A) and 64.5% in the PC arm (arm B).[15] In this study, the response rate in the EP arm was higher than that of the PC arm but not statistically significant. Liang et al, however, showed a significant survival benefit in terms of 3-year overall survival (OS) rate with EP arm [41.1% (95% CI 31.1%-50.7%)] versus PC arm [26% (95% CI 17.8%-35.1%)] with a median follow up time of 73 months (range 41-88 months). Some studies, on the other hand, showed different findings. Steuer et al performed a systematic review where they found no significant difference in response rates between cisplatin-etoposide and paclitaxel-carboplatin (58% versus 56%; p 0.26).[16] Nevertheless, these studies were retrospective and

possibly have more bias, as there is significant heterogeneity between the studies analysed affecting the results. Examples of this include radiation and chemotherapy dosing, experimental agents, overall treatment protocol, etc. In addition to that, more studies in the paclitaxel-carboplatin arm included induction and/or consolidation chemotherapy suggesting that several cycles of full dose paclitaxel and carboplatin were required for adequate response in this arm. This could be the reason, that in our study, we got sub-optimal response in PC arm as no induction or consolidation therapy was allowed in the treatment protocol.

Those patients, who showed progression during follow up, were planned for local palliative measures like palliative radiation to involved bones when necessary, and/or to whole brain. Side by side, they were planned for molecular studies and palliative systemic therapy.

There were multiple limitations of this study. As it was a non-randomized quasi-experimental study selection bias was present. The time period was short to evaluate the outcomes like progression free survival or overall survival. This study doesn't reflect the nationwide scenario owing to a small sample size and to the fact that it was conducted in two centers of Dhaka city only.

CONCLUSION

As per this study, it can be concluded that the treatment response of cisplatin-etoposide regimen is similar to that of paclitaxel-carboplatin regimen when given with concurrent radiotherapy in unresectable locally advanced non-small cell lung cancer. Choice between them may be dictated by their respective toxicity profile.

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REFERENCES

1. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2021;71(3):209-49.
2. National Institute of Cancer Research and Hospital. Hospital Cancer Registry Report 2015-2017. Dhaka; 2020.
3. Warner KE, Mendez D. Tobacco control policy in developed countries: yesterday, today, and tomorrow. Nicotine Tobacco Res. 2010;12(9):876-87.
4. Malhotra J, Malvezzi M, Negri E, La Vecchia C, Boffetta P. Risk factors for lung cancer worldwide. Eur Respir J. 2016;48(3):889-902.
5. Trichopoulos D, Lagiou P, Trichopoulou, A. Evidence-based nutrition. Asia Pac J Clin Nutr. 2000;9(Suppl.):S4-9.
6. Woodson K, Tangrea JA, Barrett MJ, Virtamo J, Taylor PR, Albanes D. Serum α-tocopherol and subsequent risk of lung cancer among male smokers. J Nat Cancer Inst. 1999;91(20):1738-43.
7. Curran Jr WJ, Paulus R, Langer CJ, Komaki R, Lee JS, Hauser S, et al. Sequential versus concurrent chemoradiation for stage III non-small cell lung cancer: randomized phase III trial RTOG 9410. J Nat Cancer Inst. 2011;103(19):1452-60.
8. Aupérin A, Le Péchoux C, Rolland E, Curran WJ, Furuse K, Fournel P, et al. Meta-analysis of concomitant versus sequential radiochemotherapy in locally advanced non-small-cell lung cancer. J Clin Oncol. 2010;28(13):2181-90.
9. Bezjak A, Temin S, Franklin G, Giaccone G, Govindan R, Johnson ML, et al. Definitive and adjuvant radiotherapy in locally advanced non-small-cell lung cancer: American Society of Clinical Oncology clinical practice guideline endorsement of the American Society for Radiation Oncology evidence-based clinical practice guideline. J Clin Oncol. 2015;33(18):2100-5.
10. Sause W, Kolesar P, Taylor IV S, Johnson D, Livingston R, Komaki R, et al. Final results of phase III trial in regionally advanced unresectable non-small cell lung cancer: Radiation Therapy Oncology Group, Eastern Cooperative Oncology Group, and Southwest Oncology Group. Chest. 2000;117(2):358-64.
11. Halperin EC, Wazer DE, Perez CA, Brady LW. Perez and Brady's principles and practice of radiation oncology. 7th edn. Wolters Kluwer; 2019.
12. Furuse K, Fukuoka M, Kawahara M, Nishikawa H, Takada Y, Kudoh S, et al. Phase III study of concurrent versus sequential thoracic radiotherapy in combination with mitomycin, vindesine, and cisplatin in unresectable stage III non-small-cell lung cancer. J Clin Oncol. 1999;17(9):2692.
13. Bradley JD, Paulus R, Komaki R, Masters GA, Forster K, Schild SE, et al. A randomized phase III comparison of standard-dose (60 Gy) versus high-dose (74 Gy) conformal chemoradiotherapy with or without cetuximab for stage III non-small cell lung cancer: Results on radiation dose in RTOG 0617. J Clin Oncol. 2013;31(15_suppl):7501.
14. Flentje M, Huber RM, Engel-Riedel W, Andreas S, Kollmeier J, Staar S, et al. GILT- A randomised

phase III study of oral vinorelbine and cisplatin with concomitant radiotherapy followed by either consolidation therapy with oral vinorelbine and cisplatin or best supportive care alone in stage III non-small cell lung cancer. Strahlenther Onkol. 2016;192(4):216.
15. Liang J, Bi N, Wu S, Chen M, Lv C, Zhao L, et al. Etoposide and cisplatin versus paclitaxel and carboplatin with concurrent thoracic radiotherapy in unresectable stage III non-small cell lung cancer: a multicenter randomized phase III trial. Ann Oncol. 2017;28(4):777-83.
16. Steuer CE, Behera M, Ernani V, Higgins KA, Saba NF, Shin DM, et al. Comparison of concurrent use of thoracic radiation with either carboplatin-paclitaxel or cisplatin-etoposide for patients with stage iii non-small-cell lung cancer: a systematic review. JAMA Oncol. 2017;3(8):1120-9.
17. Chu E, DeVita Jr VT. Physicians' cancer chemotherapy drug manual 2020. Jones and Bartlett Learning; 2019.
18. Dobbs J, Barrett A, Morris S, Roques T. Practical radiotherapy planning. 4th edn. Hodder Arnold; 2009.
19. Bi N, Liu L, Liang J, Wu S, Chen M, Lv C, et al. Efficacy and safety of concurrent chemoradiotherapy in ECOG 2 patients with locally advanced non-small-cell lung cancer: a subgroup analysis of a randomized phase III trial. BMC Cancer. 2020;20:1-10.
20. Senan S, Brade A, Wang LH, Vansteenkiste J, Dakhil S, Biesma B, et al. PROCLAIM: randomized phase III trial of pemetrexed-cisplatin or etoposide-cisplatin plus thoracic radiation therapy followed by consolidation chemotherapy in locally advanced nonsquamous non-small-cell lung cancer. J Clin Oncol. 2016;34(9):953-62.

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