

Effect of Radiation Therapy on Atelectasis from Lung Cancer

Seong Eon Hong, M.D. and Young Ki Hong, M.D.

Department of Therapeutic Radiology, College of Medicine, Kyung Hee University, Seoul, Korea

From January 1981 to December 1989, total 42 patients with atelectasis from lung cancer were treated with radiation therapy at the Department of Therapeutic Radiology in Kyung Hee University Hospital. The reexpansion of atelectasis after radiotherapy of the lung was evaluated retrospectively, utilizing treatment records and follow-up chest radiographs. Of the patients with non-small cell carcinoma of the lung, the response rate was 62% (21/34). Patient with small cell carcinoma showed a 75% (6/8) response rate. There appears to be some evidence of a relationship of total tumor dose versus response of atelectasis; radiation dose over 40 Gy (1337 ret), had a favorable effect on the rate of response compared with that below 40 Gy (1297 ret), 70% (21/30) and 50% (6/12), respectively ($p < 0.01$). Total response rate (partial and complete responses) of all patients was 64% (27/42). Fraction size was not contributed to the difference of response rates between small fraction (180~200 cGy) and large fraction (300 cGy), 53% (14/22) and 65% (13/20), respectively. The results of this study suggest that radiation therapy has a definite positive role in management of atelectasis caused by lung cancer, especially in inoperable non-small cell carcinoma.

Key Words: Radiation therapy, Lung cancer, Atelectasis

INTRODUCTION

Carcinoma of the lung, which is steadily increasing, is the third leading cause of death from cancer in Korea¹. One of the symptomatic manifestations of lung cancer is from the occurrence of atelectasis. Whether collapse of single lobe or of a complete lung, the resulting symptoms of dyspnea and associated decrease in lung function may cause respiratory embarrassment, therefore it must be treated immediately before pneumonia or irreversible obstruction occurs². Most cases who have obstructing a major bronchus can not be adequately treated surgically and radiation therapy remains the only potential cure for these patients³.

There are few reports relating symptoms to histology, either by frequency or response of atelectasis caused by lung cancer. Atelectasis from tumor obstruction is a common problem that is often difficult to relieve, responding to radiation in less than 25% of cases⁴. However, recent data indicate that more than 60% of patients with non-small cell lung cancer presenting with atelectasis can have this problem relieved by appropriate radiotherapy⁵.

The purpose of following retrospective study was to evaluate the efficacy of radiation therapy for

the patients with atelectasis from lung cancer.

MATERIALS AND METHODS

Two hundred and eighty-six patients with lung cancer were irradiated for palliative or definitive treatment at the Department of Therapeutic Radiology in Kyung Hee University Hospital from January 1981 through December 1989. Patients selected from this group for the study included only those who had biopsy proven lung cancers and evidence of atelectasis of single lobe or of a complete lung on chest radiographs. Patients with prior pneumonectomy or lobectomy, and those who received previous chemotherapy and/or radiation therapy, were excluded from this study. Forty-two patients fit the above criteria (Table 1).

The reexpansion of atelectasis by radiation therapy of the lung was evaluated retrospectively, utilizing treatment records and chest X-ray films. Chest radiographs of these patients taken before and after irradiation were compared, noting only the change in atelectasis. Complete response (reexpansion) was defined when post-irradiation chest X-rays showed a complete expansion of the atelectatic segment. A partial response was defined when chest X-ray evidence of less than a optimal complete reexpansion of the atelectatic

segment was observed. All other lesser levels of reexpansion observed were determined as no re-

Table 1. Patients Characteristics

Characteristics	No. of Patients (%)
Age	
Range	30–86 years
Median	60 years
Sex	
Male	36 (86)
Female	6 (14)
Histology	
Squamous cell ca.	28 (67)
Small cell ca.	8 (19)
Adenocarcinoma	4 (9)
Large cell ca.	2 (5)
Location of Atelectasis	
Right Lung	24 (57)
RUL	10
RLL	10
RUL + RLL	4
Left Lung	18 (43)
LUL	6
LLL	6
LUL + LLL	6
Radiation Dose	
less 40 Gy	12 (29)
over 40 Gy	30 (71)
Fraction Size	
180 – 200 cGy	22 (52)
300 cGy	20 (48)

sponse. The radiation response was defined to include X-ray evidence of complete or partial reexpansion of the atelectatic lobe or lung, up to one month post-irradiation. No attempt was made to evaluate the effect of therapy on survival rate.

RESULTS

Of the 42 patients evaluated, there were 34 patients with non-small cell carcinoma, who received tumor doses ranging from 1600 to 6600 cGy (753~1876 ret). Twenty-three (23/34) of the patient with non-small cell carcinomas received 4000~6600 cGy (1337~1876 ret) in 4 to 7 weeks. In this category 70% (16/23) showed a response, ten complete and six partial. In those who received less than 4000 cGy (1297 ret) the response rate was 45.5% (5/11). Twenty-one of these 34 patients (62%) responded (complete and partial responses) to irradiation. The data are shown by histology in Table 2. Sixty-eight percent of patients (19/28) with atelectasis secondary to squamous cell carcinoma responded, with the atelectasis completely clearing in 42.8% (12/28). Patients with small cell carcinoma showed a 75% (6/8) response rate, and patients with adenocarcinoma showed a 50% response rate (2/4). But patients with undifferentiated large cell carcinoma were not responded (0/2). There appears to be some evidence of a relationship of total tumor dose versus response of atelectasis: radiation dose over 40 Gy (1337 ret) had a favorable effect on the rate of response compared with that below 40 Gy (1297 ret), 70% (21/30) and 50% (6/12), respectively ($p < 0.01$). Total response rate (complete and partial responses) of overall patients was 64% (27/42).

Table 2. Response of Atelectasis according to Cell Type and Radiation Dose

	Complete response	Partial response	No change	Progression	Total
Less 40 Gy (12)	Squamous cell 4	1	2	1	8
	Small cell	1			1
	Adenoca.		2		2
	Large cell		1		1
Over 40 Gy (30)	Squamous cell 8	6	4	2	20
	Small cell 4	1	1	1	7
	Adenoca. 2				2
	Large cell			1	1
Total patients	18 (43%)	9 (21%)	10 (24%)	5 (12%)	42

Table 3. Response of Atelectasis according to Location of Lesion

		Complete response	Partial response	No change	Progression	Total
Right (24)	RUL	5	2	2	1	10
	RLL	4	2	3	1	10
	RUL + RLL		2	2		4
	Total	9 (38%)	6 (25%)	7	2	15/24 (63%)
Left (18)	LUL	3	1		2	6
	LLL	3	1	1	1	6
	LUL + LLL	3	1	2		6
	Total	9 (50%)	3 (17%)	3	3	12/18 (67%)

Table 3 presents response rates of atelectasis according to location of lesion. Total response rates of right and left lung were 62.5% (15/24) and 66.7% (12/18), respectively. Fraction size was not contributed in difference of response rate between small fraction (180~200 cGy) and large fraction (300 cGy), 63% (14/22) and 65% (13/20), respectively.

DISCUSSION

Atelectasis of the lung is usually caused by bronchial obstruction from lung carcinoma. At presentation, signs of bronchial obstruction are evident on chest radiographs in 53% of patients with squamous cell lung carcinoma, 25% with adenocarcinoma, 33% with large cell carcinoma, and 38% with small cell carcinoma⁶. Twenty percent had an endobronchial lesion in a mainstem bronchus and 50% in a lobar bronchus³. The incidence of significant endobronchial metastasis ranges from 25 to 50%^{7,8}. Breast, colon, and renal cell carcinomas are the most common histologies associated with endobronchial metastases, although endobronchial spread also had been reported with sarcomas, melanomas, and ovarian cancers⁷. Tracheal and bronchial tumors that occlude a significant portion of the airway lumen must be treated promptly before infection, respiratory distress, or irreversible obstruction occurs⁹.

The symptoms, signs, and radiographic findings in the chest are related to central or peripheral location of primary tumor, in addition to whether regional spread has occurred, both of which are related to histologic type¹⁰. In general, epidermoid cancers have a central location, with atelectasis, pneumonia (from bronchial obstruction), hilar

adenopathy, and a tendency to cavitate; adenocarcinomas have a defined nodule in peripheral location, with pleural and chest wall involvement; large cell carcinomas have a large mass in peripheral location with pneumonitis and hilar adenopathy; and small cell carcinomas present as a central lesion with atelectasis-pneumonitis and hilar and mediastinal adenopathy. Symptoms of centrally located tumors include cough, wheezing, stridor, deep chest pain, hemoptysis, and dyspnea caused by obstruction with or without postobstructive pneumonitis. Peripheral lesions present with pain and cough from pleural or chest wall involvement, pleural effusion, and dyspnea on restrictive basis¹¹.

Bronchial obstruction is associated with radiographic abnormalities, including atelectasis, segmental consolidation, or a triangular pleural density. Routine chest films demonstrate signs of obstructive pneumonitis in 75% of patients with significant bronchial obstruction. Previous chest films should be reviewed to determine the duration of obstructive pneumonitis. A definitive diagnosis is made with fiberoptic bronchoscopy, biopsy, and sputum cytology⁹.

Patients presenting with symptoms of impending upper airway obstruction must be treated immediately to avoid severe respiratory insufficiency and death. In addition, response in symptomatic patients suggest that one should be able to delay appearance of symptoms in asymptomatic patients as inferred by Phillips and Miller¹². Patients with primary lung cancer obstructing a major bronchus are rarely curable with surgery³. Saunders et al² reported that all 6 of their patients with central airway obstruction and dyspnea showed significant improvement in the latter, although little measurable change in pulmonary function. If curative

radiotherapy is planned for inoperable, localized obstructing non-small cell carcinoma, the tumor volume should receive a midline dose of 5500 to 6000 cGy. Smaller radiation doses (from 2000 in one week to 3500 cGy in 3.5 weeks) are employed when the goal is palliation¹³⁾.

Recent data indicated that more than 60% of patients with non-small cell carcinoma were relieved by appropriate radiotherapy⁵⁾. Kang et al¹⁴⁾ reported that the rates of complete and partial response in less than 59 Gy group were 42.9% (6/14) and 21.4% (3/14), but in higher dose (above 60 Gy) the response rates were 55.6% (5/9) and 33.3% (3/9), respectively. In our cases 68% of patients (19/28) with atelectasis secondary to squamous cell carcinoma responded, with complete clearing in 42.8% (12/28). But the patients with small cell carcinoma showed a 75% (6/8) response rate. Total response rate (complete and partial response) of all patients with atelectasis was 64% (27/42). There appears to be some evidence of a relationship of tumor dose versus response of atelectasis; radiation dose over 40 Gy (1337 ret), had a favorable effect on the rate of response compared with that below 40 Gy (1297 ret), 70% (21/30) and 50% (6/12), respectively ($p < 0.01$). The response of atelectasis according to location of lesion was slight different; right lung was 62.5% (15/24), left side was 66.7% (12/18). However there was no difference between the response rates of small fraction size (180~200 cGy) and of large one (300 cGy), 63% (14/22) and 65% (13/20), respectively. External irradiation offers prompt palliation if the obstruction is of recent onset. Bronchial obstruction from small cell anaplastic lung cancer is promptly relieved with either combination chemotherapy or a combination of radiotherapy and chemotherapy¹⁵⁾.

Although the sample size is too small and varied to be statistically analyzed effectively, the previous data indicate that atelectasis due to lung neoplasms responds to therapeutic doses of radiation in varying degrees, depending on the histology of the disease. A small decrease in the size of this obstructive endobronchial lesion is sufficient to reestablish patency of the bronchial lumen and aeration of the lung. The average survival time is very short, and therapy is often aimed at reducing symptoms and improving the quality of life. If the patient was refractory to prior radiotherapy, palliation may be obtained with implantation of iridium seeds¹⁶⁾, laser beam therapy¹⁷⁾, or cryosurgery¹⁸⁾.

REFERENCES

1. 보건사회부 : 한국인 암등록 자료분석 보고서, 대한 암학회지 21:153-216, 1989
2. Saunders KB, Rudolf M, Banks RA, et al: Central airway obstruction of the bronchus treated by radiotherapy: a study of pulmonary function. Br J Radiol 51:286-290, 1978
3. Brewer LA: Patterns of survival in lung cancer. Chest 71:644-650, 1977
4. Slawsson RG, Scott RM: Radiation Therapy in Bronchogenic Carcinoma. Radiology 132:175-176, 1979
5. Majid OA, Lee S, Khushalani S, et al: The response of atelectasis from lung cancer to radiation therapy. Int J Radiat Oncol Biol Phys 12:231-232, 1986
6. Miller WE: Roentgenographic manifestations of lung cancer. In Straus MJ (ed): Lung Cancer; Clinical Diagnosis and Treatment. New York, Grune & Stratton, 1983, pp 175-184
7. Braman SS, Whitcomb ME: Endobronchial metastasis. Arch Intern Med 135:543-547, 1975
8. Baumgartner WA, Mark JBD: Metastatic malignancies from distant sites to the tracheobronchial tree. J Thorac Cardiovasc Surg 79:499-503, 1980
9. Sise JG, Crichlow RW: Obstruction due to malignant tumors. Semin Ocol 5:213-224, 1978
10. Byrd RB, Carr DT, Miller WE: Radiographic abnormalities in carcinoma of the lung as related to histologic cell type. Thorax 24:573-575, 1969
11. Cohen MH: Signs and symptoms of bronchogenic carcinoma. In Straus MJ (ed): Lung Cancer, Clinical Diagnosis and Treatment. New York, Grune & Stratton, 1983, pp 97-112
12. Phillips TL, Miller RJ: Should asymptomatic patients with inoperable bronchogenic carcinoma receive immediate radiotherapy? yes. editorial. Am Rev Respir Dis 117:405-410, 1978
13. Lee RE: Radiotherapy for lung cancer. In Straus MJ (ed): Lung Cancer; Clinical Diagnosis and Treatment, pp 213-244, Grune & Stratton, New York, 1983
14. 강철훈, 신세원, 김명세: 단순 흉부 X-선 사진상 폐암소견에 대한 방사선치료의 효과-단기 추적 조사를 중심으로-, 대한치료방사선과학회지 7:227-233, 1989
15. Cox JD, Byhardt R, Komaki R, et al: Interaction of thoracic irradiation and chemotherapy on local control and survival in small cell carcinoma of the lung. Cancer Treat Rep. 63:1251-1255, 1979
16. Rostrom AY, Morgan RL: Results of treating primary tumor of the trachea by irradiation. Thorax 33:387-393, 1978
17. Laforet E, Berger R, Vaughan C: Carcinoma obstructing the trachea. Treatment by laser resec-

tion. N Engl J Med 295:1579-1584, 1976
18. Saunderson DR, Neel HB, Fontana RS: Bronchos-

copic cryotherapy. Ann Oto Rhinol Laryngol 90:
354-358, 1981

== 국문초록 ==

무기폐를 동반한 폐암의 방사선 치료

경희대학교 의과대학 치료방사선과학교실

홍 성 언 · 홍 영 기

1981년부터 1989년까지 경희대학 부속병원 치료방사선과에서 무기폐를 동반한 폐암으로 방사선 치료를 받은 환자중, 과거력상 폐절제술, 항암요법, 방사선치료등의 기왕력이 있는 환자를 제외한 42명을 대상으로 치료성적을 분석하였다.

1. 비소세포성 폐암은 62% (21/34), 소세포성 폐암은 75% (6/8)에서 각각 무기폐가 방사선 치료 후 재팽창 되었으며, 전체 환자의 64% (27/42)에서 부분 또는 완전반응 (reexpansion)을 나타내었다.

2. 방사선 총선량이 40 Gy (1297 ret) 이하와 40 Gy (1337 ret) 이상인 군에서 반응은 각각 50% (6/12)와 70% (21/30)이었으며, 총선량과 무기폐 반응사이에는 유의한 관계가 있었다 ($p < 0.01$).

3. 분할조사선량에 따른 반응율은 소선량 (180~200 cGy)에서는 53% (14/22), 대선량 (300 cGy)에서는 65% (13/20)이었다.

4. 이상의 성적으로 보아 무기폐를 동반한 폐암 환자중에서, 특히 수술과 항암요법으로 치료가 곤란하고 조속한 처치를 요하는 비소세포성 폐암에서 방사선 치료의 중요한 역할이 확인되었다.