

similar appearance to those afflicted by the staphylococcal scalded skin syndrome, but the level of epidermal cleavage is much deeper in toxic epidermal necrolysis. It is a highly lethal disease with a mortality of 20–70%, even in the best centres. It has a substantial morbidity which includes corneal scarring, contractures, and oesophageal strictures. Pneumonia is a common complication of this disorder.¹

The plan of management is to keep the affected areas free from infection with applications of local antiseptics. Good nursing is of paramount importance. Systemic steroids are not of proven benefit and may increase the risk of secondary infection. Careful fluid and electrolyte monitoring is required.

Our patient developed symptoms and signs of respiratory mucosal sloughing for 48 hours

before her acute choking attacks. The bronchial obstruction was removed with a fiberoptic bronchoscope although this took up to an hour to do on two separate occasions. In the light of this experience we feel that it may be safer to use a rigid bronchoscope at the onset of bronchial mucosal sloughing to facilitate suction of secretions and slough. Tracheostomy may need to be performed to secure the airway patency despite the risks of local sepsis.

This case highlights the existence of a life-threatening complication of this disorder, and should enable clinicians to plan management of such cases on the basis of our experience.

¹ Heimback DM, Engrav LH, Marvin JA, Harnar TJ, Grube BJ. Toxic epidermal necrolysis – a step forward in treatment. *JAMA* 1987;257:2171–5.

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Pneumothorax following thoracic radiation therapy for Hodgkin's disease

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Abstract

Radiation therapy alone to the nodal drainage sites above the diaphragm, namely a "mantle" field, is often standard treatment for early stage Hodgkin's disease and may be used in combination with chemotherapy in more advanced disease. Localised pneumonitis and fibrosis are recognised treatment related sequelae; however, other pulmonary complications, including pneumothorax, have been described. Two cases of spontaneous pneumothorax following mantle radiation therapy are presented.

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Case 1

An 18 year old non-smoking male university student of medium build presented with left axillary and supraclavicular masses and was subsequently diagnosed as having clinical stage IIA nodular sclerosing Hodgkin's disease. Subtotal nodal irradiation was given, beginning with a mantle field delivering 36 Gy in 20 fractions, followed by an infradiaphragmatic field delivering the same dose to the para-aortic nodes and spleen. The treatment course was uneventful but five months after completing the chest irradiation he was found to have a

right apical pneumothorax on routine chest radiography.

As the patient was asymptomatic and the pneumothorax comprised less than 20% of the right lung volume, he was observed. Serial chest radiographs showed gradual resolution over a further five months. He continued normal activity, including sports, and has had no evidence of Hodgkin's disease one year after treatment.

Case 2

A 28 year old non-smoking woman of normal build presented with a four week history of a mass in the left side of her neck and night sweats twice a week for two months. Nodular sclerosing Hodgkin's disease was diagnosed after nodal biopsy, and computed tomographic scanning of the neck and thorax showed a mediastinal mass which was continuous with the right neck disease. She was designated pathological stage IIB following a normal staging laparotomy and proceeded to mantle irradiation to a total dose of 36 Gy.

Follow up chest radiographs indicated that the mediastinal mass had completely responded but five months after treatment bilateral apical pleural capping was noted. The apical changes progressed until a chest radiograph 17 months after treatment showed a small left pneumothorax which had then resolved three months later. Again the patient remained asymptomatic.

Discussion

The two cases reported demonstrate the complication of pneumothorax occurring after mantle radiation therapy alone for Hodgkin's disease. Both the cases received 36 Gy and this is consistent with other reported cases who also received more than 30 Gy.^{1–4} There have been too few reported cases to postulate a dose-response relation.

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Our two cases occurred amongst a group of over 60 patients with Hodgkin's disease treated with radiation therapy alone in our department during the last 10 years using modern megavoltage linear accelerators with customised lung shielding. Pezner *et al* reported a frequency of spontaneous pneumothorax of 2.2% in patients with Hodgkin's disease without concurrent pulmonary disease treated with mantle radiation therapy.² As the finding is often in asymptomatic patients the frequency will depend on the vigor of patient follow up, but exceeds figures generally quoted as the incidence of spontaneous pneumothorax in the general community.⁵ The true incidence of asymptomatic pneumothorax in the general population is unknown. The temporal relation of the pneumothoraces in these two cases, in relation to radiation therapy, raises questions as to their specific aetiology.

Spontaneous pneumothorax arises most commonly in previously healthy 20–40 year olds, usually the result of rupture of apical blebs on the visceral pleura.⁶ Subpleural apical blebs and fibrosis were found at thoracotomy in three patients from a series of nine pneumothoraces in patients with Hodgkin's disease.² Although these cases received mantle radiation therapy they had documented interstitial fibrosis associated with protracted courses of BCNU or cyclophosphamide, and the contribution of the radiation therapy is difficult to determine.

Apical fibrosis as the result of radiation therapy, occurring prior to the diagnosis of pneumothorax, has been reported previously.³ The radiation dose to the lung apex is higher than at other points in the chest due to the smaller thoracic diameter superiorly. This increases late radiation effects, particularly fibrosis. Patients with unusual degrees of fibrosis, either as a result of chemotherapy or radiation therapy, may be at a higher risk of pneumothorax.²

The pathogenesis of pneumothorax in cases treated with radiation therapy alone remains undefined, although apical fibrosis seems to be the initiating process. It is unclear whether the frequency of this complication will change as follow up time increases in these patients cured of their Hodgkin's disease.

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