

18.14.12 Radiation pneumonitis 4271 S.J. Bourke

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18.14.12 Radiation pneumonitis 4271 in the early detection of subacute disease such as bronchiolitis and limiting its adverse consequences. FURTHER READING Cowl CT (2019). Assessment and treatment of acute toxic inhalations. *Curr Opin Pulm Med*, 25, 211–16. de Lange DW, Meulenbelt J (2011). Do corticosteroids have a role in preventing or reducing acute toxic lung injury caused by inhalation of chemical agents? *Clinical Toxicology*, 49, 61–71. King MS, et al. (2011). Constrictive bronchiolitis in soldiers returning from Iraq and Afghanistan. *N Engl J Med*, 365, 222–30. Kreiss K (2013). Occupational causes of constrictive bronchiolitis. *Curr Opin Allergy Clin Immunol*, 13, 167–72. Sauler M, Gulati M (2012). Newly recognized occupational and environmental causes of chronic terminal airways and parenchymal lung disease. *Clin Chest Med*, 33, 667–80. 18.14.12 Radiation pneumonitis S. J. Bourke ESSENTIALS The lungs can be injured by radiation used in the treatment of cancer, with the rapidly dividing endothelial cells and type II pneumocytes most affected. Immediate injury is followed by an inflammatory response and at a later stage by fibrosis. Chest radiography detects asymptomatic changes in about 50% of patients after radiotherapy. Acute radiation pneumonitis presents with cough, breathlessness, and fever about 2 months after exposure; corticosteroids are usually effective in relieving symptoms but do not prevent the subsequent development of fibrosis. Fibrosis typically develops about 6 months later, may progress for 6 to 24 months, but has usually stabilized by 2 years. Prevention depends on refining techniques for giving radiotherapy. Introduction The lungs are vulnerable to injury from radiation used in the treatment of cancers of the lung, breast, oesophagus, spine, thymus, and lymph glands, and when whole-body irradiation is given in preparation for bone marrow transplantation. Radiation pneumonitis continues to cause significant morbidity and rarely mortality, and remains a limiting factor for the intensity of radiation treatment of patients with

inoperable lung cancer. It is an important adverse effect which needs to be considered when assessing new treatment strategies using high intensity radiation and concurrent chemotherapy and radiation treatment, particularly in an ageing population with pre-existing lung disease, reduced lung reserve, and comorbidities. Radiation causes direct injury to cells and DNA within the field of radiotherapy, giving rise to pneumonitis and fibrosis. The induction of reactive oxygen species and the initiation of cytokine-mediated inflammatory responses result sporadically in more diffuse radiation lung injury involving areas of the lung outwith the radiotherapy field. Acute radiation pneumonitis is characterized by interstitial inflammation occurring up to 4 months after radiotherapy and then resolving over a matter of weeks or months. Radiation fibrosis, which can occur without preceding pneumonitis, develops about 6 months after radiotherapy and may progress over 6–24 months; it does not resolve, but usually stabilizes by 2 years. Pathogenesis Factors which influence the development of radiation pneumonitis and fibrosis include the volume of lung irradiated, the total radiation dose administered, and the dose rate and fractionation. Concomitant use of chemotherapeutic drugs such as taxanes, erlotinib, bleomycin, doxorubicin, methotrexate, and cyclophosphamide can aggravate radiation lung injury. Furthermore, when chemotherapy is given after radiotherapy, ‘recall pneumonitis’ may develop in the areas of lung previously irradiated. Tamoxifen has been shown to enhance lung injury in patients receiving radiotherapy for breast cancer, which may be due to increased release of transforming growth factor β (TGF β). Corticosteroid withdrawal may also precipitate radiation pneumonitis, and there is increased risk with pre-existing lung fibrosis or current lung infection. Absorption of radiation by lung tissues accelerates electrons, generating ion pairs and reactive oxygen species, which damage DNA and produce chemical and biological effects in cells. Rapidly dividing cells, such as endothelial cells and type II pneumocytes, are most affected. The earliest changes involve injury to small vessels with thrombosis, increased permeability, and exudation of protein-rich fluid into the alveoli. Epithelial injury results in sloughing of cells, hyaline membrane formation, and proliferation of type II pneumocytes. Inflammatory cells accumulate in the alveolar walls, followed at a later stage by fibroblasts. Increased plasma concentrations of TGF β and intercellular adhesion molecule (ICAM)-1 correlate with an increased incidence of radiation pneumonitis. ICAM-1 stimulates the accumulation of inflammatory cells, whereas TGF β stimulates fibroblast proliferation and induces synthesis of collagen, and genetic polymorphisms that result in high production of TGF β are associated with more severe radiation fibrosis. Cellular expression of CD95 and CD95-ligand are increased after radiotherapy, and these receptors are involved in the induction of apoptosis, inflammatory cytokine responses, and the attraction of inflammatory cells. These immunologically mediated responses are not confined to the radiotherapy field. Clinical features Asymptomatic changes are detectable on a chest radiograph in about 50% of patients after radiotherapy. Characteristically there is an area of opacification that does not show a segmental or lobar distribution: it crosses the normal anatomical structures and is demarcated by a sharp margin corresponding to the limits of the radiotherapy field (Fig. 18.14.12.1). Air bronchograms are often present and there is usually a loss of volume.

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