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Hypercute Radiation Pneumonitis*

Allan L. Goldman, M.D.** and Robert Enquist, M.D.†

We present a patient who developed radiation pneumonitis only eight days after beginning therapy. The pneumonitis responded dramatically to prednisone on four occasions, which was a helpful point in the differential diagnosis.

Radiation therapy is commonly used to treat various thoracic neoplasms. Radiation may cause adverse effects including acute radiation pneumonitis. Radiation pneumonitis characteristically appears one to 16 weeks after completion of radiotherapy.1 A patient is presented who developed radiation pneumonitis only eight days after beginning therapy, the hyperacute onset causing great difficulty with diagnosis. Pneumonitis in his case was exquisitely corticosteroid responsive, and this proved to be a useful point in the differential diagnosis.

CASE REPORT

A 36-year-old man was admitted to Walter Reed General Hospital for evaluation of hemoptysis and an abnormal chest roentgenogram. He was a heavy smoker but denied anorexia, weight loss, chest pain, chills or fever. He underwent bronchoscopy and mediastinoscopy on June 15, 1973, which revealed nearly complete obstruction of the right mainstem bronchus and involvement of the right hilar lymph nodes by a large cell undifferentiated carcinoma. Figure 1 shows a pre-radiotherapy chest roentgenogram with the radiation ports marked. On June 19, 1973 the patient began a course of radiotherapy to consist of 5000 rads given as a daily dose of 200 rads in 25 treatments over a 37 day period. On June 26, after the 6th treatment, he developed dyspnea, a non-productive cough, fever of 101°F (38.3°C), and malaise. He had been on no drugs. A complete blood count revealed a white cell count of 8,800 with 81 neutrophiles, 12 lymphocytes, and 7 monocytes. Sputum culture showed normal flora, and blood cultures showed no growth. A chest film at this time revealed opacification on the right side most marked in the area of irradiation (Fig 2).

The patient was empirically begun on 40 mg of prednisone, and within 24 hours was asymptomatic. By June 29, 1974 the chest roentgenogram showed almost complete resolution of the infiltrate (Fig 3). Because of uncertainty of the diagnosis of radiation pneumonitis by the attending staff, the prednisone was abruptly discontinued. Within a day the patient had a clinical and roentgenologic relapse which rapidly responded to the reinstitution of prednisone. This sequence was repeated two more times, after which he has been maintained on 40 mg of prednisone until lost from followup in the middle of August. While maintained on the prednisone, he had no further acute episodes and neither clinical nor roentgenologic evidence of chronic radiation fibrosis.

*From the Pulmonary Disease Section, Walter Reed General Hospital, Washington, D.C.

**Presently Chief, Pulmonary Disease Section, University of South Florida College of Medicine.

†Fellow in Pulmonary Disease.

Reprint requests: Material Branch, Walter Reed Army Medical Center, 5401 Linden Lane, Silver Spring, Maryland 20910

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FIGURE 1. Pre-radiation therapy chest roentgenogram with radiation ports marked.

As with any therapeutic modality, there are certain inherent risks in the use of radiation. Radiation to the lung may induce either chronic interstitial fibrosis, or an acute radiation pneumonitis. The chronic fibrotic stage generally begins two to six months after completion of the radiotherapy. The patient presents with a nonproductive cough and dyspnea, and serial chest roentgenograms reveal progressive volume loss in the irradiated area.2

In contrast to the delayed insidious onset of chronic fibrosis is the more dramatic onset of acute radiation pneumonitis. The patient presents with dyspnea and a nonproductive cough. Fever may be present, and thus simulate an acute infectious process. Location of the infiltrate in the irradiated field supports the diagnosis.3 The pneumonitis characteristically appears one to 16 weeks after completion of the radiation therapy.1 This time of appearance has been useful in suggesting the diagnosis. Radiation pneumonitis may occur later if the patient has been on corticosteroids that suppress its expression but it has not been reported earlier.4,5

The occurrence of acute radiation pneumonitis is unpredictable, and its pathogenesis unclear. It has been

FIGURE 2. Chest roentgenogram after 6th treatment showing opacification on the right side most marked in the area of irradiation.

FIGURE 3. Chest roentgenogram showing almost complete resolution of the infiltrate after three days of prednisone therapy.
ILL-Effects of Cardiac Resuscitation: Report of Two Unusual Cases*

Steven G. Atcheson, M.D.,** Gary V. Petersen, M.D.,† and Herbert L. Fred, M.D., F.C.C.P.*

Two mishaps associated with closed-chest cardiac resuscitation are presented. One—pneumoperitoneum—became evident during life, created considerable diagnostic difficulty, and evoked treatment that possibly hastened the patient’s death. The other—cardiac puncture—appeared at autopsy and its mechanism may be unique.

We recently witnessed two bizarre complications of cardiac resuscitation. The therapeutic and philosophic implications of these accidents are important and form the basis of this communication.

CASE REPORTS

Case 1

At entry into the hospital, this 82-year-old woman had physical and chest roentgenographic signs of congestive heart failure. Therapy with digoxin and diuretics resulted in substantial clinical improvement. One week after admission she suddenly manifested ventricular fibrillation. External cardiac massage, electric countershock, and ventilation via face mask restored sinus rhythm and normal blood pressure in ten minutes. Midway through the procedure, progressive abdominal distension occurred. Chest roentgenogram revealed free air beneath each hemidiaphragm, but none in the mediastinum, pericardium, pleura, or subcutaneous tissues. During the next several hours, abdominal distension persisted and her rectal temperature rose to 38.9° C. Gastric perforation seemed likely, and we reluctantly proceeded with celiotomy. Operation demonstrated massive, unexplained pneumoperitoneum. Her condition then steadily worsened, and she died four days later.

Autopsy established no cause of her pneumoperitoneum. She did have bronchopneumonia in both lower lobes, severe coronary atherosclerosis, and recent subendocardial infarction. Notably absent was evidence for lacerated intestine, peritonitis, fractured ribs, or intrathoracic injury.

Comment: This case emphasizes that pneumoperitoneum consequent to closed-chest cardiac resuscitation need not reflect perforated gut. At least five patients have displayed postresuscitative pneumoperitoneum. In two, our patient and another,† the site and nature of air leak defied detection. Two others‡,§ had ruptured stomach, and the fifth¶ had ruptured esophagus just proximal to the stomach.

A second point commands attention. Prompt surgical intervention in all of the aforementioned patients benefited just two.¶,† Hence, when pneumoperitoneum complicates resuscitation, a trial of judicious medical management may be wise.

Case 2

Several hours after sustaining an acute inferior myocardial infarction, a 78-year-old man suffered cardiac standstill. Resuscitative efforts failed. Postmortem examination disclosed extensive coronary atherosclerosis and a number of exceptional abnormalities. Several sharply pointed vertebral osteophytes measuring 2 x 2 x 1 cm and resembling railroad spikes lay directly behind and impinged upon the heart. Liquid and clotted blood filled the pericardium. A half-centimeter hole extended through the pericardium and posterior wall of the left ventricle. Muscle surrounding the hole looked normal; on cut sections, however, the fibers were fragmented and incompletely striated, findings suggesting early infarction. Although the mechanism of perforation remains uncertain, we believe that during chest-wall compression an osteophyte “backstabbed” the patient, piercing his heart.

Comment: This case brings into focus a fact and two questions. The fact is: Complications of successful closed-chest cardiac resuscitation kill some patients.¶,† The questions are: 1) What kills the patient such as ours?¶,† cardiac arrest or cardiac massage? and 2) Can one prevent catastrophes of resuscitation? Answers are conjectural at best.

*From the Departments of Internal Medicine, St. Joseph Hospital, Houston, Texas and McKay-Dee Hospital, Ogden, Utah.
**Resident in Medicine, The University of Texas Medical School at Houston.
†Staff Cardiologist, McKay-Dee Hospital, Ogden, Utah. Clinical Instructor, Department of Internal Medicine, University of Utah College of Medicine, Salt Lake City.
‡Director of Medical Education, St. Joseph Hospital, and Professor and Vice Chairman, Department of Internal Medicine, The University of Texas Medical School at Houston.

Reprint requests: Dr. Fred, St. Joseph Hospital, Houston, 77002