Interstitial Densities Following Radiotherapy

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R.D. is a 51-year-old man who presented to his local rural hospital in April 2002 with facial flushing, shortness of breath, cough, and a syncopal episode. He reported that his symptoms had persisted for three weeks, and he had been treated for a sinus infection three times prior to this admission. Because of fatigue, he was being evaluated for possible thyroid dysfunction. He also had a past medical history of long-term smoking and coal dust exposure. A chest x-ray (see Figure 1) and a computerized tomography (CT) scan were ordered. A large mediastinal mass was noted as well as marked obstruction of the superior vena cava from the mass. R.D. was transferred immediately to a regional medical center where a mediastinoscopy for biopsy was performed; a Groshong catheter also was inserted. He received three radiation treatments to the chest over the course of a weekend. A diagnosis of stage III-B non-small cell carcinoma of the lung was confirmed, and the patient’s treatment plan consisted of 28 days of radiotherapy to the mediastinum and six cycles of paclitaxel and carboplatin. R.D. received his first cycle of chemotherapy in the hospital and was discharged on April 12 with continued outpatient radiotherapy; his next cycle of chemotherapy was scheduled for mid-May. R.D. and his family planned to stay in town Monday through Friday for his daily radiation treatments and return to their home community on Saturdays and Sundays.

R.D. received his second cycle of paclitaxel and carboplatin as scheduled on May 8 and returned to his hometown for the weekend on May 11. He was near completion of the thoracic radiotherapy. On May 12, he presented to his local emergency department with complaints of fever and shaking chills. His diagnostic studies revealed the following: white blood cell count 2,900/mm³, hemoglobin 13.2 g/dl, and platelet count 311,000/mm³. His physical examination showed significant erythema and purulent drainage at the mediastinal incision. He was transferred to the regional medical center with a diagnosis of febrile neutropenia and wound sepsis. Upon his arrival, admitting orders included laboratory tests, a chest x-ray, a CT scan, and an ultrasound. Results of his diagnostic studies were as follows: white blood cell count 10,100/mm³, platelet count 461,000/mm³, partial thromboplastin time 157 seconds, and prothrombin time (PT) 14.1 seconds. R.D.’s chest x-ray revealed interstitial densities with extensive infiltration through the right lung and left perihilar area. Although his chest x-ray revealed that the mass was smaller, new central interstitial densities were more prominent on the right and his right diaphragm was elevated. The radiologist’s report further stated that the findings were consistent with pulmonary edema or more likely, lymphangitic spread of neoplasm or postradiation pneumonitis (see Figure 2). The CT scan results were consistent with radiation pneumonitis and multiple bilateral pulmonary emboli. An ultrasound of R.D.’s chest was significant for clots in the left internal jugular vein.

His recovery was uncomplicated, and he was discharged from the hospital on May 17 with IV antibiotics and dressing changes to the mediastinal wound. The last of his radiotherapy was completed wound cultures, initiation of antibiotics and filgrastim, placement on neutropenic precautions, and a surgical consult. The mediastinal incision was opened, and a deep abscess on the anterior chest wall was drained. A CT scan was performed at that time that revealed a decrease in the tumor mass. R.D.’s chest x-ray revealed interstitial densities following radiotherapy, radiation pneumonitis, and multiple bilateral pulmonary emboli. An ultrasound of R.D.’s chest was significant for clots in the left internal jugular vein.

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vein and left subclavian vein because of the Groshong placement. The patient was started on a heparin protocol, warfarin, and steroids. His oxygen demand continued to be problematic. He used a nasal cannula with saturations in the high 80 to low 90 percentile. R.D. became extremely hypoxic with minimal exertion and used a nonrebreather mask as needed. His international normalization ratio on June 24 was 2.3; therefore, heparin was discontinued. His diagnostic tests at the time were as follows: white blood cell count 11,300/mm³, platelet count 625,000/mm³, and PT 27 seconds. On June 25, he was discharged with a nonrebreather mask for his hypoxia and coumadin for his clotting problem.

Four days later, on June 29, R.D. presented to his local emergency room with acute dyspnea and a two-day history of low-grade fever. His family reported that R.D. had developed slight swelling of his neck and left arm since his discharge from the medical center. He was admitted to the local facility, intubated to manage his oxygenation, and received heparin and antibiotics. Once stabilized, he was transported emergently to the regional medical center. R.D. was admitted to the intensive care unit for ventilatory support and further evaluation. The admitting chest x-ray showed increased infiltration in the right upper lobe and new infiltrates in the left lower lung (see Figure 3). A diagnosis of progressive radiation pneumonitis was confirmed.

Pneumonitis

Radiation-induced pneumonitis is defined as an inflammation of the lungs resulting from radiation exposure (Segawa, Katoaka, Takigawa, & Eguchi, 1998). Radiation causes direct injury to the endothelial and epithelial cells, which results in alveolitis leading to accumulation of inflammatory and immune cells in the alveolar walls and spaces. This accumulation, which is believed to play a role in the subsequent development of pulmonary fibrosis or chronic inflammation (Stover & Kaner, 2001), distorts the normal structures of the alveoli and results in the release of lymphokines and monokines.

Radiation-induced pneumonitis typically occurs after radiation treatment for cancer within the chest or breast, can develop in 5%–15% of patients, and usually presents two to six months after completion of therapy (Chisam & Douglas, 2002). The severity of the pneumonitis is dependent on the dose and volume of lung exposed to the radiation field and the use of chemotherapy agents (Taghian et al., 2001). Administration of paclitaxel is associated specifically with a high incidence of radiation-induced pneumonitis; therefore, the combination of paclitaxel and radiation therapy should be used with caution (Powell, 2001; Smith, 2001). The relationship between paclitaxel and radiation therapy currently is under study (Hanna, Baglan, Stromberg, Vicini, & Decker, 2002).

Other chemotherapeutic agents have been associated with pulmonary parenchymal damage and subsequent pneumonitis (see Figure 4). How chemotherapy-induced pneumonitis occurs is not clearly understood. Like radiation-induced pneumonitis, chemotherapy-induced pneumonitis is characterized by abnormalities in endothelial and epithelial cells. The symptoms experienced by these patients are similar to radiation-induced pneumonitis (Camp-Sorrell, 2000; Makimoto, Tsuchiya, Hayakawa, Siatoh, & Mori, 1999; Stover & Kaner, 2001). Although research findings have not demonstrated a causal relationship between high-risk chemotherapy agents and radiation therapy delivered to the chest, patients who have been treated previously with these medications may be at higher risk for developing lung complications such as pneumonitis. Other risk factors that may predispose patients to pneumonitis include a history of smoking, poor nutritional status, age (e.g., the elderly are at highest risk), collagen vascular disease, and disease-related factors (e.g., congestive obstructive pulmonary disease, cytomegalovirus) (Camp-Sorrell; Makimoto et al.).

The clinical features of cancer treatment-related pneumonitis are vague, and patients initially may be misdiagnosed. The most common presenting symptoms include vague chest pain, dyspnea, shortness of breath upon activity, nonproductive cough, fatigue, and audible wheezes. These may be self-limiting, but they can progress to severe respiratory distress (Camp-Sorrell, 2000; Stover & Kaner, 2001). Fever and hemoptysis are unusual; however, they may present in some cases. The hallmark symptom of treatment-related pneumonitis is dyspnea.

Blood analysis can indicate inflammation by an elevated white blood cell count and increased sedimentation rate. A radiographic characteristic of radiation-induced pneumonitis is a diffuse infiltrate corresponding to a previous treatment area. If this persists, radiation-induced pneumonitis can cause scarring of the lungs, called radiation fibrosis. The fibrosis is the repair process that follows the inflammatory response. Presenting as progressive fibrosis of the alveolar walls...
thickened by elastic fibers, this typically occurs a year after completion of radiation treatment; however, this complication can take months or years to evolve. Radiation-induced pneumonitis may be reversible with medications that reduce inflammation, but radiation fibrosis is usually irreversible (Camp-Sorrell, 2000).

Corticosteroids remain the treatment of choice for radiation-induced pneumonitis. The recommended treatment is to begin prednisone 1 mg/kg as soon as the diagnosis is reasonably certain. Steroids must be tapered slowly. If tapered too soon or too quickly, exacerbation of symptoms has been reported, requiring higher dose steroids and longer treatments. This may occur in 2%–9% of patients treated for lung cancer with chemotherapy and radiation treatments (Camp-Sorrell, 2000; Chisam & Douglas, 2002).

Summary

After admission on June 30, R.D. remained intubated, and he continued on IV steroids, heparin, and warfarin. Nutritional needs were met with a nasointestinal feeding tube and nutritional preparations. He was alert and oriented and communicated appropriately with family and staff via written notes. The patient and his wife wanted to try a ventilator for a period of time before considering a “No CPR” order. His chest wound continued to be open but was healing slowly. Over the next few days, R.D. became more hypoxic with increased respiratory effort and required sedation and assist-control ventilator settings. On July 1, he required more sedation to keep him comfortable, but remained alert and oriented and continued to communicate with his family. On July 3, he sat in a chair for 1.5 hours. On July 4, he developed a large right pneumothorax and a chest tube was placed. He continued to indicate that he was short of breath. The patient remained very anxious and was started on a propofol drip. Later that day, his wife had a discussion with the healthcare team; the decision was made not to resuscitate the patient. On July 5, R.D.’s agitation increased and he was started on additional propofol for sedation, vecuronium bromide to facilitate breathing, and lorazepam IV push for relaxation. R.D.’s oxygenation-ventilation status declined through the night. After a discussion between the family and the physician on July 6, life support was withdrawn, and R.D. died later that day.

Some of the factors that may have led to R.D.’s radiation-induced pneumonitis include his prior history of smoking as well as his former occupation as a coal miner. He received 15 radiation treatments to his chest area. He also received chemotherapy, including the drug paclitaxel; this combination may have contributed to his radiation-induced pneumonitis. The pneumonitis led to his immunosuppressed condition. R.D.’s superior vena cava syndrome led to the formation of clots for which he received heparin and coumadin. He received steroids to reduce the inflammation from the mediastinoscopy site and in his lungs. All of these factors contributed to R.D.’s outcome.

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References


