CASE REPORT

Is ionizing radiation a risk factor to diffuse panbronchiolitis?

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Learning point for clinicians

An interesting hypothesis that ionizing radiation from atomic bombs and nuclear power plants is a risk factor to diffuse panbronchiolitis, and the evidence of the possible risks are based on the analysis of the epidemiological studies and the studies of different types and absorbed doses of ionizing radiation.

Case report

In 2012, a 64-year-old male presented with a 10-year history of productive cough and progressive dyspnea. He had never smoked and had been treated for refractory asthma and chronic sinusitis for several years. He had no known exposure to any occupational respiratory hazards except that he had worked in a nuclear power plant in Taiwan since 1977. Physical examination revealed crackles and inspiratory and expiratory wheezes in both lungs on auscultation. Chest radiography showed mildly increased interstitial markings. He was admitted due to his symptoms and progressively worse airway obstruction. A computed tomography (CT) scan of the chest showed bronchiec-tasis, centrilobular nodules and tree-in-bud opacities bilaterally suggesting diffused panbronchiolitis (DPB) (Figure 1A). A transbronchial lung biopsy confirmed the pathology for DPB. This patient received macrolide therapy with clarithromycin 1000 mg daily. Over the next 18 months after the initiation of macrolide therapy, he experienced a dramatic improvement. A chest CT scan was performed 18 months later to confirm the clinical improvement (Figure 1B).

Discussion

The first case of DPB was reported in 1969, about 20 years after the end of World War II, and many cases of DPB were reported in the following years in Japan. Many cases of DPB were reported after the 1980s in countries with nuclear power plants outside Japan. Atomic weapons and nuclear reactors use a chain reaction to respectively induce an uncontrolled and controlled rate of nuclear fission in fissile material, releasing both energy and different types and doses of ionizing radiation including alpha particles, beta particles and gamma rays.

Many people died and became severely injured due to initial ionizing radiation, mainly consisting of extremely high dose of gamma rays caused by the atomic bombings of Hiroshima and Nagasaki in Japan, the Mayak incident and the Chernobyl disaster. In these events, sequelae occurred from the residual radiation.1–3 Although the Life Span Study has been able to characterize the dose response of medium to high doses of
ionizing radiation for a variety of outcomes, the long-term residual low-dose effects of ionizing radiation remain uncertain.4

Internal exposure by alpha particles is strongly ionizing with a higher biological effect in ionizing radiation. However, due to their short range of absorption, their inability to penetrate outer layers of the skin, and their being easily shielded against, alpha particles are generally not dangerous to life unless the source is ingested or inhaled. The incidence of chronic bronchitis in a cohort of Mayak workers showed a statistically significant linear dose–response relationship with cumulative doses of external gamma-ray and internal alpha particle-related radiation. There are increasing documented evidence to prove internal alpha particles are risk factors to chronic airway disease.5

In other uses of ionizing radiation apart from atomic weapons and nuclear reactors, nuclear fission rarely happens and types and doses of ionizing radiation are strictly controlled. This is to reduce the possibility of DPB in those uses of ionizing radiation. Generally, unexpected different types and doses of ionizing radiation should not happen, but nuclear power plants may have chance to produce unexpected ionizing radiation for DPB.

It is highly suspected that internal alpha particles from atomic weapons or nuclear reactors may play an important role in DPB depends on the epidemiological studies and the studies of different types and absorbed doses of ionizing radiation. Those alpha particles inhaled into the lungs, thereby inducing chronic inflammation of the bronchial tree leading to DPB. We also found that many of the DPB cases had chronic sinusitis, an opening of the airway, in which induced chronic inflammation leading to chronic sinusitis is caused by alpha particles deposition.

Conflict of interest: None declared.

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Figure 1. CT scan of the chest showed a case of DPB before and after receiving macrolide therapy with clarithromycin. (A) CT scan of the chest showed bronchiectasis, centrilobular nodules and tree-in-bud opacities bilaterally before receiving macrolide therapy. (B) CT scan of the chest confirmed the clinical improvement after receiving macrolide therapy with clarithromycin 1000 mg daily for 18 months.