Treatment of PuO2 Lung Contamination using a Dry Powder Formulation of DTPA

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Lung contamination can result from accidental release of actinides such as Pu and Am. The therapeutic approach to reduce the effective radiation dose is to remove the α-emitting radionuclides from the body by promoting their decorporation. DTPA is the recommended treatment of internal contamina-tion by plutonium. The present work investigates the decorporation efficacy of a dry powder formulation of CaNa3-DTPA on a pulmonary contamination with PuO2. Sprague-Dawley rats were exposed to PuO2 aerosols, and 2 hours later received an intratracheal insufflation of CaNa3-DTPA (18.2 $\pm$ 1.4 μmol /kg) formulated into porous particles. Urines were collected daily for 7 days. Initial lung deposit (ILD) was determined by X ray spectrometry counting 7 days post-inhalation. Fourteen days post-inhalation, rats were euthanized, liver, femurs and lungs were collected and broncho alveolar lavages (BAL) were carried out. The total alpha activity of samples was measured by liquid scintillation counting in BAL, BAL cells and BAL fluids. The ILDs of untreated rats and DTPA-treated rats were respectively 15.6 $\pm$ 2.3 and 13.6 $\pm$ 2.3 kBq. The cumulative activity urinary excretion over 7 days was 7-fold higher after DTPA administration as compared to untreated rats, and represented approximately 7% of the ILD for DTPA-treated animals. In the main retention tissues, liver and skeleton, the deposit of activity in DTPA-treated rats was less than 5% of the one of untreated animals (1.13% of ILD in liver of untreated rats vs 0.05% in DTPA-treated rats; 2.75% in skeleton vs 0.1%). Distribution of alpha activity within lungs was determined. Activity recovered in BAL fluids from DTPA-treated rats was 7.3-times lower than in BAL fluids from untreated animals. Although the activity associ-ated with BAL cells tended to decrease, the difference between the 2 groups remained non significant, suggesting that pulmonary surfactant and/or serum-derived proteins represented the major accessible lung compartment for DTPA decorporation. However, no significant decrease in whole lung activity was ob-tained. Our study shows the efficacy of a dry DTPA powder on actinide decorporation. By inhibiting ac-tinide deposit in skeleton and liver, a limitation of the dose delivered to these tissues is expected, thus limiting the risks for radiation-induced diseases. In addition, DTPA treatment modified distribution of activity within lungs. It is generally admitted that soluble compounds leading to more homogeneous irra-diation of the lungs cause higher damage than insoluble forms. The decorporation of the most soluble fraction of radionuclide present in BAL fluids, could thus limit lung damage.

Article published by EDP Sciences and available at http://www.radioprotection.org or http://dx.doi.org/10.1051/radiopro:2008561