A CASE OF INTERNAL CONTAMINATION WITH PLUTONIUM OXIDE

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This paper describes a case of plutonium and americium internal contamination due to an accidental glove-box explosion occurred in 1974 at the Casaccia Plutonium Plant. The involved person showed a small contaminated wound (3x 0.5 cm) on his right cheek, a diffused contamination on the hair and a considerable activity in the nose which would indicate a possible incorporation by inhalation. On the basis of the information obtained at the Plutonium Plant the material contained in the exploded glove-box resulted to be a powder of PuO_2 calcinated at high temperature. In order to know just the isotopic and weight composition of the contaminating material the following radiometric and chemical measures were carried out on the nose-blow sample: gamma spectrometry (^{241}Am); gamma+X spectrometry with a thin NaI(Tl) crystal and Be window (^{241}Am and Pu); liquid scintillation (^{241}Pu and alpha emitters); alpha spectrometry (^{238}Pu + ^{241}Am and ^{239}Pu + ^{240}Pu); chemical separation of americium from plutonium. The following data were obtained: 97.56% in weight for alpha emitters (0.17% ^{238}Pu, 97.05% $^{239,240}Pu$, 0.34% ^{241}Am) and 2.44% ^{241}Pu; the activity distribution was 95.9% beta activity (^{241}Pu) and 4.1% alpha activity ($^{238,239,240}Pu$ and ^{241}Am); the distribution of alpha activity resulted to be 65% $^{239,240}Pu$, 25% ^{238}Pu and 10% ^{241}Am. The knowledge of the isotopic composition was necessary to correctly estimate the initial plutonium and americium activity in the wound, the lung burden calculated by W.B.C. and the dose committment to the different organs.

DIAGNOSTIC AND THERAPEUTIC ACTIONS

The following actions were taken to reduce the initial contamination and to get the maximum information on the residual contamination and on the dose committment. a) The wound was washed with DTPA and the activity was removed by a surgical toilet; b) the hair and the nose were decontaminated; c) some direct lung countings were performed; d) many urine and fecal samples were analyzed for Pu and ^{241}Am; e) some blood samples were collected for the determination of plutonium and for the detection of possible chromosomial aberrations; f) at the second day a DTPA treatment was started consisting on three dayly 0.5 g DTPA intravenous injections followed by 3 others on alternate days and on a 0.5 g DTPA aerosol inhalation during 2 consecutive days.

RESULTS

The following results were obtained. a) The activity in the wound was determined (1) by using a special NaI(Tl) probe suitable to detect the weak X emission of plutonium (17 KeV) and the X+gamma emission of ^{241}Am (17 KeV and 60 KeV); the localization of the superficial alpha activity was obtained by using a probe with a 7 mm^2 solid state alpha detector. The initial activity resulted to be $\sim$ 30 nCi, and it was reduced to background levels by washing with DTPA and by carrying out a surgical toilet. b) The initial activity in the hair was $\sim$83 nCi and it was reduced to negligible values by using a shampoo containing DTPA. The activity of the nose blow, collected just after the incident, was 7.5 nCi. A direct lung counting (2,3) of the subject, based on the detection of both the 17 KeV X-rays emitted by the plutonium isotopes and the 60 KeV gamma-rays of the ^{241}Am, was performed at various times after the incident. A 12.5 cm diameter x 0.1 mm thick NaI(Tl) phosphwich crystal positioned on the right lung or over the sternum was employed. The calibration factor applied to lung counting of the plutonium isotopes was obtained on the basis of both phantom and "in vivo" calibration (3,4) taking into account both the chest size of the subject and the isotopic composition of the contaminating mixture. The calibration factor for the ^{241}Am in vivo counting was based on phantom calibration only. The $^{238,239,240}Pu$ lung contents $\pm$ 2σ as a function of time elapsed from the incident were the following: 56 $\pm$ 20 nCi (5 h.); 25 $\pm$ 15 nCi (22 h.); 13 $\pm$ 10 nCi (5 d.); $<$10 nCi (19 d.). The corresponding ^{241}Am lung contents $\pm$ 2σ resulted to be: 2.0 $\pm$ 0.5 nCi; 1.5 $\pm$ 0.5 nCi; 0.7 $\pm$ 0.3 nCi; 0.4 $\pm$ 0.3 nCi (40 d.); 0.15 nCi (70 d.). d) Taking into account the 55 urine analyses (37 of Pu and 18 of ^{241}Am) and the 34 feces analyses (26 of Pu and 8 of ^{241}Am) (5), the excretion curves shown in Fig. 1 and 2 have been obtained. e) No plutonium activity greater than the sensitivity limit (0.04 pCi) was detected in 10 ml of blood and no cromosomial aberration was found in 200 cells. f) No effect due to the DTPA treatment was shown in the urinary excretion curves.

DOSIMETRIC EVALUATION

Taking into account the data supplied by the lung counting and by the excretion curves, the following conclusions can be drown: a) the ratio $^{238,239,240}Pu/^{241}Am$ for fecal excretion is about 10, just as the ratio of the alpha activity present in the contaminating material: the similar metabolism observed for Pu and Am can be due to the fact

that both the elements were present as a very insoluble oxide; b) both the Pu and Am fecal excretion curves are very steep during the first few days (Peak activity/Plateau activity $\sim 10^5$) and this datum is in good agreement with the sharp decrease of the lung content in the same period: it appears threfore that the material granulometry was high (1 ÷ 10 /um), mainly deposited in the upper part of the respiratory tract and thus fastly removed by the ordinary clearance mechanisms. c) The high ratio E_f/E_u ($\sim 10^4$ in the first few days) and the inefectiveness of DTPA confirm the biological non-trasportability of the contaminant. d) The plutonium activity excreted in the first few days with feces is $\sim$130 nCi which may correspond to an initial lung burden comprised between 13 and 65 nCi; this value is in good agreement with that found by the W.B.C. at the first day (56 $\pm$ 20 nCi). e) Taking into account the fecal curve after the first ten days, a lung half-time of about 100 days can be deduced in accordance with the values reported in the literature (6) for insoluble compounds. f) The fecal excretion after 100 days (0.5 pCi) would indicate a plutonium residual lung burden of 0.25 ÷ 1 nCi (5). g) Taking into account the urinary excretion after 100 days (0.2 pCi) a plutonium systemic burden of 3 nCi can be obtained (7). h) The committed lung dose, calculated on the basis of reference (6) and a biological half-life of 500 days for Pu and Am, resulted in the range of 60 ÷ 240 mrad with a corresponding maximum dose rate of 30 ÷ 120 mrad/y. i) The dose due to systemic contamination has been evaluated on the basis of reference (8) and considering the following percentage depositions and biological half-lives: Pu 42% in bone ($T_b = 5.5 \cdot 10^4$ d.), 56% in liver ($T_b = 5.5 \cdot 10^4$ d.); Am 25% in bone ($T_b = 7.3 \cdot 10^3$ d.), 35% in liver ($T_b = 3.5 \cdot 10^3$ d.) and 3% in kidneys ($T_b = 2.7 \cdot 10^4$ d.). For the contribution of lung contamination to systemic dose, the T_b in lung was considered 90 days. l) The calculated absorbed dose rate for bone was rather constant being in the range of 30 ÷ 40 mrad/y slowly increasing with time; for liver a rather constant dose rate of 200 mrad/y; for kidneys a rather constant dose rate of 3 mrad/y slowly decreasing with time. m) The committed dose equivalents, calculated on the basis of ICRP recent metabolic models (9,10) with Q = 20 for alpha particles, are: lung 1.2 ÷ 4.8 rem (12 ÷ 48 mSv); bone 40 rem (400 mSv); liver 100 rem (1 Sv); kydneys 3 rem (30 mSv). The effective total body committed dose equivalent is 7.5 ÷ 8 rem (75 ÷ 80 mSv). From a medical point of view, the operator was readmitted

to unlimited radiation work, but caution was taken not to involve him in high-risk contamination areas or operations.

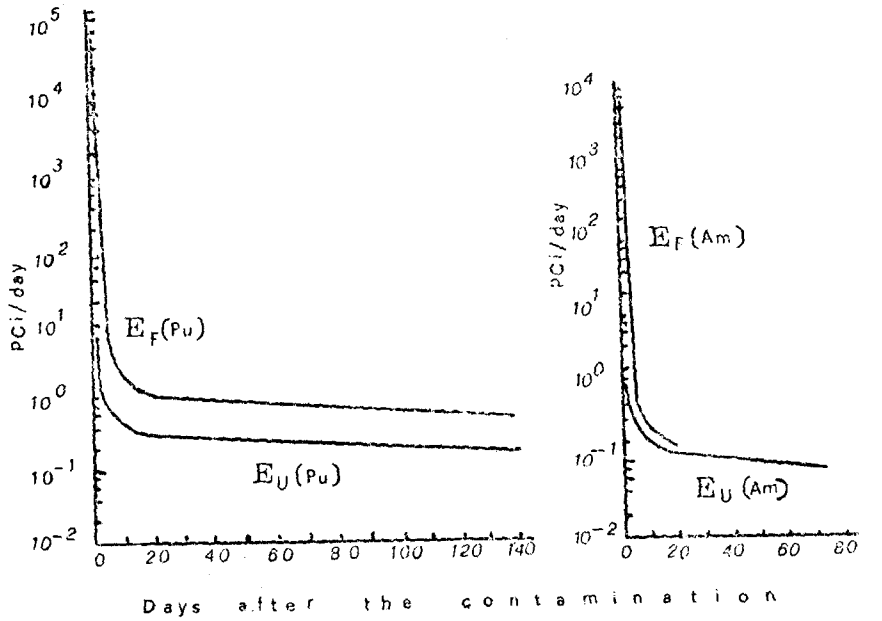

Fig.1 and 2. Urinary (E_u) and fecal (E_f) excretion of plutonium and americium.

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