Lung Uptake of Technetium-99m Microaggregated Albumin in the Rats After Immunization with Macroaggregated Albumin

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During the study of liver uptake of Tc-99m human serum albumin (Tc-HSA) in the sensitized dogs, one of authors recognized and reported the occasional occurrence of lung uptake of the Tc-HSA. As a possible cause of lung uptake, pulmonary microembolization of intravascular aggregated albumin was proposed. The purpose of the present study is to verify this hypothesis and to investigate the exact mechanism involved in this phenomenon by using Tc-99m microaggregated albumin (Tc-MIAA).

Wister strain male rats were immunized by two subcutaneous injections of 5 mg of HSA or human macroaggregated albumin (MAA) in incomplete adjuvant at two weeks interval. Two weeks after the second injection of antigen, lung and liver scannings were performed using Tc-MIAA, Tc-HSA and Tc-sulfur colloid. MIAA was prepared by the sonication of MAA. The particle size was assessed microscopically and by organ distribution studies in the control rats.

As the results, in all rats immunized with MAA, MIAA were markedly accumulated in the lungs with similar or greater activity than in the liver.

However, no lung was visualized in the same group when rats were injected Tc-sulfur colloid or Tc-HSA. On the other hand, in the group which was immunized with HSA, there was no visualization of the lung by Tc-HSA, Tc-MIAA or Tc-sulfur colloid.

Increased deposition of Tc-MIAA in the lung of the rats after immunization with MAA strongly suggested that the mechanism for lung visualization in the present study was due to microembolization of clumping particle (antigen-antibody complex). In clinical condition, immune mechanism may be an unlikely cause for intravascular aggregation of liver particles. However, some other unknown mechanism, particularly abnormality in the plasma protein may cause intravascular aggregation of liver particles.

The absence of liver accumulation of HSA in the rats sensitized with HSA were unexpected from the previous experience with dogs. This appears to suggest some difference in immune response between two species. However, exact mechanism for this difference remain for the further study.

A Basic Study for Clinical Application of 111In-chloride Bone Marrow Agent

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It is assumed that trace amounts of ionic Indium-111 is specifically bound to serum transferrin and then in part distributed to the erythroid line in active bone marrow when injected intravenously in the form of 111In-Cl₂ at acid pH.

However, the exact distribution and metabolism has not as yet solved, so that there are practically several difficulties in the evaluation of scan images.

8 cases with various hematological disorders (aplastic anemia, myeloid leukemia, malignant lymphoma, multiple myeloma, iron-deficiency anemia) were evaluated with scan images and organ distribution of Indium compared with ⁵⁹Fe-citrate metabolism. Indium-binding affinity to serum protein was also studied with single radial immuno-diffusion method combined with autoradiography.

It was made clear that Indium was bound to transferrin specifically in all cases, and accumulates