Basic and Clinical Studies on Imaging for Tumor and Inflammatory Lesion by Ga-67 and In-111

1986 Fiscal Year Final Research Report Summary

Basic and Clinical Studies on Imaging for Tumor and Inflammatory Lesion by Ga-67 and In-111

Research Project

Project/Area Number

60570476

Research Category

Grant-in-Aid for General Scientific Research (C)

Allocation Type

Single-year Grants

Research Field

Radiation science

Research Institution

School of Allied Medical Professions, Kanazawa University

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Keywords

Malignant tumor / Gallium-67 / Acid mucopolysaccharide / Keratan polysulfate / 炎症巣

Research Abstract

This study was undertaken to investigate the mechanism of accumulation of Ga-67 in the malignant tumor and the inflammatory lesion. It was determined from measuring neutral saccharide in the structure that the principal Ga-67-binding acid mucopolysaccharide in the tumor was keratan sulfate and/or keratan polysulfate. On the other hand, it was clarified from the results of mucopolysaccharase treatment that the main Ga-67-binding acid mucopolysaccharide in tumor was not keratan sulfate, heparan sulfate, heparin, nor chondroitin sulfate A,B,or C. Based on the present results, it was deduced that the main Ga-67-binding acid mucopolysaccharide in tumor is keratan polysulfate and that this acid mucopolysaccharide plays the most important role in tumor accumulation of Ga-67.

The uptake rate of Ga-67 in the inflammatory lesion was increased with time after injection of turpentine oil and reached a plateau five days later, and at this time the rate for the lesion was larger than those for any other tissues. From the observation by autoradiography, it was revealed that Ga-67 was avidly accumulated corresponding to the subcutaneous tissue infiltrated with neutrophils and macrophages, and this nuclide was concentrated intercellularly around these cells. And it became clear from biochemical study that Ga-67 was bound to the acid mucopolysaccharide (keratan polysulfate, etc.) in the lesion. On the other hand, capillary permeability in this lesion was much greater than that of normal tissues.
based on the present results, mechanisms of Ga-67 uptake into inflammatory lesion are concluded as follows:accumulation of Ga-67 in the inflammatory lesion is primarily due to leakage of Ga-67 into the subcutaneous tissue infiltrated with neutrophils and macrophages through capillaries with increased permeability. In the inflammatory lesion, Ga-67 is preferentially bound to the acid mucopolysaccharides(keratan polysulfate, etc.) which compose intercellular substances.

Research Products (11 results)

All | Other

All | Publications (11 results)

[Publications] Atsushi Ando: Eur.J.Nucl.Med.10. 262-268 (1985)

[Publications] Atsushi Ando: Int.J.Nucl.Med.Biol.12. 357-362 (1985)

[Publications] Atsushi Ando: Eur.J.Nucl.Med.11. 235-239 (1985)

[Publications] Atsushi Ando: J.Radioanal Chem.Artic.99. 97-107 (1986)

[Publications] Atsushi Ando: Nucl.-Med.25. 45-49 (1986)

[Publications] 安東醇: 蛋白質・核酸・酵素. 13. 1585-1590 (1986)

[Publications] Atsushi Ando: "Tumor and liver uptake models of <^(67)Ga> -citrate" Eur. J. Nucl. Med.10. 262-268 (1985)

[Publications] Atsushi Ando: "Species of <^(67)Ga> -binding acid mucopolysaccharide in liver" Int. J. Nucl. Med. Biol.12. 357-362 (1985)

[Publications] Atsushi Ando: " <^(67)Ga> -binding substances in stomach, small intestine, pancreas, and muscle" Eur. J. Nucl. Med.11. 235-239 (1985)

[Publications] Atsushi Ando: "On the <^(67)Ga> -binding acid mucopolysaccharide in malignant tumor" J. Radioanal. Chem. Artic.99. 97-107 (1986)

[Publications] Atsushi Ando: "Retention and subcellular distribution of <^(67)Ga> in normal organs" Nucl.-Med.25. 45-49 (1986)

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