Influence of anti-inflammatory drugs on ^{18}F-FDG accumulation in chemically induced subcutaneous inflammation tissues and tumors in rats

Kenji Uchida, Naoko Harasawa, Masahiro Natsuhori, Satoru Hatakeyama[*1], Shoji Futatsugawa[*1]
Kazunori Terasaki[*2], Keiichiro Yamaguchi[*3], Tadasi Sano, Nobuhiko Ito

Kitasato University, School of Veterinary Medicine and Animal Sciences,
Higashi 23-35-1, Towada, Aomori 034-8628 Japan

*[1] Nishina Memorial Cyclotron Center, Japan Radioisotope Association,
348-58 Tomegamori, Takizawa, Iwate 020-0173 Japan

*[2]Cyclotron Research Center, Iwate Medical University
358-58 Tomegamori, Takizawa, Iwate 020-0100 Japan

*[3]Cyclotron and Radioisotope Center, Tohoku University
Aoba, Aramaki, Aoba, Sendai, Miyagi 980-8578 Japan

Abstract

In a previous report we demonstrated that the majority of ^{18}F-FDG (FDG) accumulation in acute inflammation tissue was due to significant accumulation of FDG in neutrophils, rather than in macrophages. In this study, we investigated the effects of anti-inflammatory drugs including dexamethasone and indomethacin on the accumulation of ^{18}F-FDG (FDG) in tumor and chemically induced inflammation nodules by gamma ray counting. Inflammations were experimentally induced by the subcutaneous inoculation of turpentine oil (10μl) on the back of rats (male Donryu rats, 7wks old, n=24). Also, ascites hepatoma cells (Ah109A, 8×10^6 cells) were inoculated subcutaneously on the back of the rats. FDG (5MBq) was injected to the rats from tail vein 4 hours after intraperitoneal injection of anti-inflammatory drugs. After 90 min of FDG injection, rats were euthanized under pentobarbital injection, The subcutaneous nodules and organs were removed, weighted and their radioactivity were counted to calculate standardized uptake values (SUV). Histopathological evaluation of each tissue was also performed.

The numbers of neutrophils were about 27 times more than macrophages in inflammation tissues of 4 days after inoculation. And the numbers of neutrophils were well correlated to the SUV calculated from the results obtained from autoradiography. These results again supported that the major accumulation of FDG in the acute inflammation tissue is due to accumulation in neutrophils. In a histopathological examination, an average population of the tumor cells was only 18% of the cells which composes tumor nodule, and it did not exceed 40% of the numbers of total cells. The other majority of the cells was so called framework that includes fibroblasts, lymphocytes, and capillary cells. Dexamethasone significantly decreased SUV, especially in tumor tissues rather than in inflammation tissues. This significant decrease in tumor nodules may be partly due to a significant decrease in frameworks.