

## Transient Uptake of Thallium-201 into a Cerebral Infarction: A Case Report

Daijiro Morimoto, Daizo Yoshida, Masahiro Noha,  
Mitsuyoshi Sasaki, Hiroshi Takahashi and Akira Teramoto

Department of Neurosurgery, Graduate School of Medicine, Nippon Medical School

### Abstract

We describe a 51-year-old woman with a cerebral infarction that showed transient accumulation of thallium-201. On admission, this lesion was well-enhanced by gadolinium injection and gradually expanded, mimicking a malignant brain tumor. A cerebral angiogram, however, did not indicate the presence of a malignant brain tumor. Ethyl cysteinate dimer single photon computerized tomography showed perfusion defects throughout hospitalization. The final diagnosis of cerebral infarction was established by pathological examination. Six months after onset, the enhancement by gadolinium and the expansion of the lesion disappeared. A cerebral infarction showing transient uptake of thallium-201, and lesion expansion is indicative of a lesion in the liquefaction stage that might mimic a malignant tumor. Although thallium-201 scintigrams are useful for the differential diagnosis of radiation necrosis and recurrent brain tumor, the findings in this patient should alert clinicians to the differential diagnosis of intracerebral expansive lesions.

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**Key words:** cerebral infarction, thallium-201, inflammatory process, neuroimaging

### Introduction

Thallium-201 scintigrams are critical in the differential diagnosis of recurrent brain tumors and radiation necrosis. Accumulation of thallium-201 has also been reported in several inflammatory diseases. We experienced a patient with a cerebral infarction associated with hot uptake of thallium-201.

### Case Report

A 51-year-old woman presented with sudden onset of right-sided facial hemiparesis. On day 8 after onset, after the hemiparesis had resolved somewhat,

the patient was hospitalized in our department. Physical examination disclosed right hemiparesis (manual muscle test [MMT], upper extremity, 1/5; lower extremity, 1/5). On day 20, computed tomography (CT) of the brain showed a low-density area in the left frontal lobe, and magnetic resonance (MR) imaging with gadolinium enhancement showed an area of heterogeneous enhancement, surrounded by an area of moderate edema (**Fig. 1-A**). Ethyl cysteinate dimer single photon computerized tomography showed a perfusion defect, and thallium-201 scintigraphy demonstrated high accumulation at the lesion site (**Fig. 1-B**).

Because serial CT scans and MR images showed that the lesion was becoming larger, a needle biopsy

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Correspondence to Daijiro Morimoto, MD, Department of Neurosurgery, Nippon Medical School, 1-1-5 Sendagi, Bunkyo-ku, Tokyo 113-8603, Japan  
E-mail: dai\_sampo@yahoo.co.jp  
Journal Website (<http://www.nms.ac.jp/jnms/>)

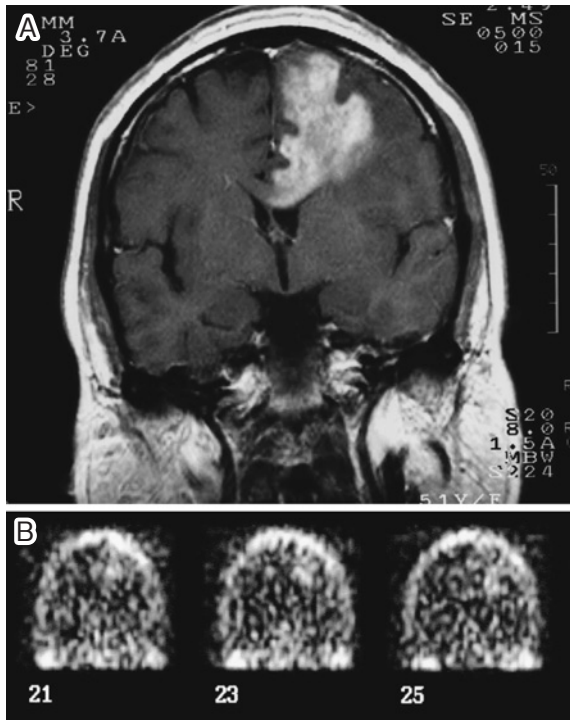


Fig. 1

- A: Contrast-enhanced T1-weighted MR image obtained at admission showing an areas of heterogeneous enhancement surrounded by edema in the left frontal lobe.
- B: Thallium-201 scintigram obtained at admission showing high accumulation at the lesion site.

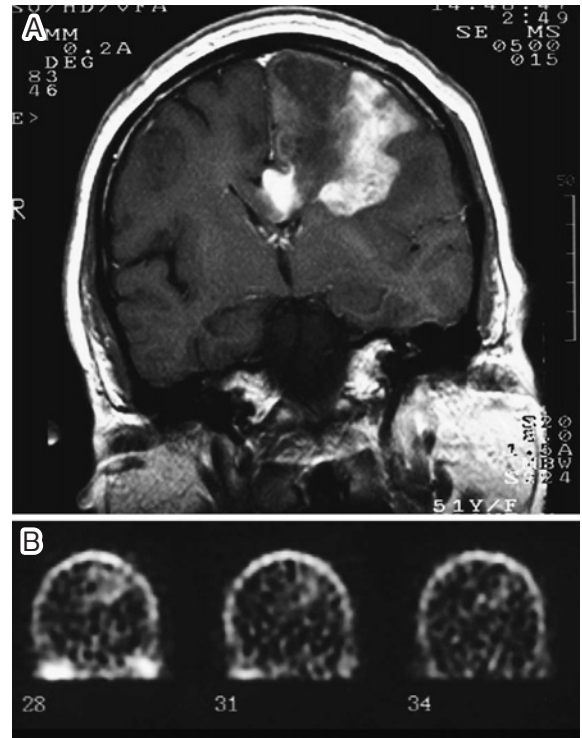


Fig. 3

- A: An MR image obtained after needle biopsy showing expansion of the lesion and an increased mass effect.
- B: Thallium-201 scintigram obtained after needle biopsy showing enhanced accumulation of thallium-201.

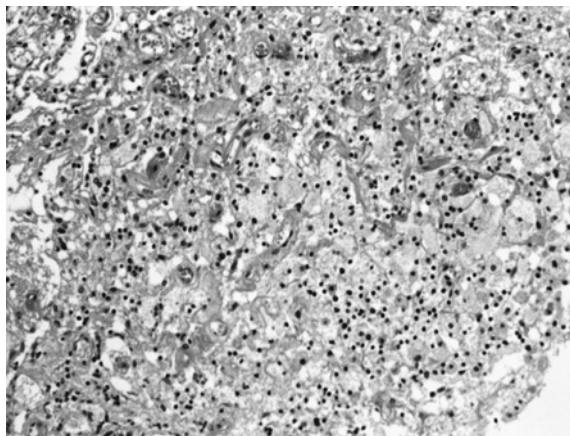


Fig. 2 Microscopic view showing infiltration of a large amount of inflammatory cells around the extravascular space (H&E, 50×).

was performed on day 51 to determine whether the lesion was a malignant tumor. The biopsy specimen showed enrichment of foamy cells, inflammatory cells infiltrating around the extravascular space, and partially necrotic tissue, but no tumor cells were found (Fig. 2). Cerebral infarction was diagnosed

with pathologic examination.

Postoperative CT and MR showed a gradual expansion of the lesion, with an increased mass effect (Fig. 3-A). Furthermore, thallium-201 scintigraphy on day 59 showed greater accumulation than at admission, although ethyl cysteinate dimer single photon computerized tomography showed a perfusion deficit at the lesion site (Fig. 3-B). Cerebral angiography showed a shift of the bilateral anterior cerebral arteries to the right, with no evidence of a malignant tumor. A second open biopsy was performed on day 90. The pathological diagnosis, cerebral infarction, was identical to the previous diagnosis. Both MR and thallium-201 scintigraphy on day 95 showed a reduction in the size of the lesion, along with gradual improvement in symptoms (Fig. 4-A, B).

### Discussion

Thallium-201 scintigraphy has been used in the differential diagnosis of radiation necrosis and recurrent brain tumors in the field of

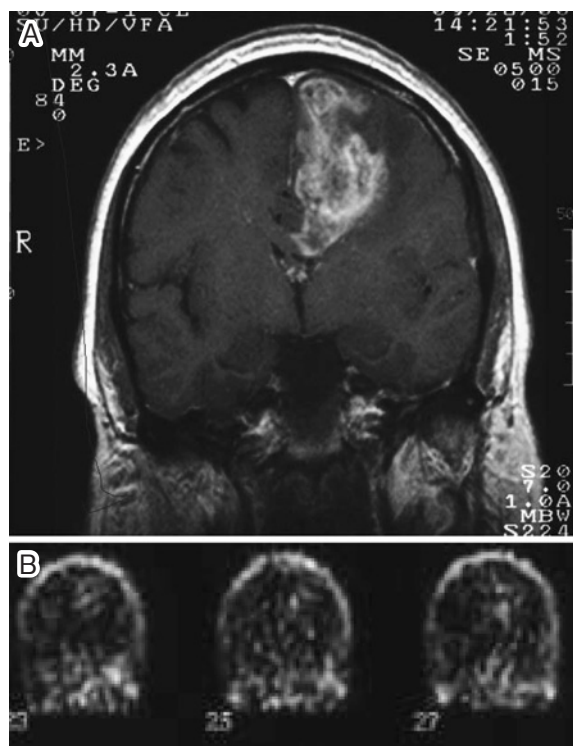


Fig. 4

- A: An MR image obtained after open biopsy showing reduction of the lesion.
- B: A thallium-201 scintigram obtained after open biopsy showing reduction in thallium-201 accumulation.

neuroradiology. However, the biological mechanism underlying thallium-201 uptake by brain tumors remains unclear. A comparative study of thallium-201, technetium-99m, and gallium-67 uptake into malignant brain tumors has shown that technetium-99m and gallium-67 simultaneously accumulate in tumor tissue and in areas of radiation necrosis and perifocal edema, whereas accumulation of thallium-201 into tumor cells was more selective<sup>1</sup>. Although thallium-201 can normally cross the blood-brain barrier (BBB), disruption of the BBB can enhance thallium-201 accumulation<sup>1-3</sup>. This type of disruption, however, may occur in pathologic conditions other than cerebral infarction, such as ischemic or inflammatory changes<sup>4</sup>, suggesting that the specific uptake of thallium-201 by the brain is not due only to the disruption of the BBB. During disruption of the BBB, accumulation of thallium-201 is dependent on adenosine triphosphatase (ATP-ase) of the sodium-potassium channel in tumor cells, because thallium-201 behaves very much like potassium<sup>15</sup>.

In addition, thallium-201 uptake has been reported in patients with cerebral candidiasis and pulmonary

actinomycosis<sup>6,7</sup>, and in conditions related to the inflammatory process, including inflammatory subcutaneous tissues infiltrated with neutrophils and macrophages<sup>5</sup>. Many phagocytic cells appear during the liquefaction stage of cerebral infarction<sup>8</sup>. Indeed, we observed infiltration of a large number of phagocytic cells in the operative specimens of our patient, which may explain, at least part, the mechanism underlying thallium-201 uptake during the liquefaction stage of cerebral infarction<sup>8</sup>. We observed infiltration of inflammatory cells around the extravascular space in the liquefaction stage of infarction, suggesting that, in our patient, thallium-201 accumulation may be explained by the presence of these inflammatory cells.

Recent descriptions have emphasized that thallium-201 accumulates specifically in brain tumor tissues but accumulates at low levels in areas of necrosis, postirradiation necrosis, or ischemia. In this report, we have presented a case of cerebral infarction associated with accumulation of thallium-201. Unexpected uptake of thallium-201 into a cerebral infarction in the liquefaction stage may be a neuroradiologic pitfall.

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