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Unexpected accumulation of thallium-201 in bilateral thalamic venous infarction induced by arteriovenous fistula in the posterior fossa: Report of a case

Fumihiko TAMAMOTO,* Atsushi NAKANISHI,* Tomoko TAKANASHI,* Hideyuki ISHIZAKI,* Hidekazu NAGASAWA,* Tadayuki MAEHARA,** Shingo OHNO*** and Yutaka OTSUBO***

*Department of Radiology, Tokyo Metropolitan Ohtsuka Hospital
**Department of Radiology, Juntendo University School of Medicine
***Department of Neurosurgery, Tokyo Metropolitan Ohtsuka Hospital

We encountered unexpected accumulation of thallium-201 in a patient with thalamic dementia resulting from bithalamic venous infarction induced by arteriovenous fistula in the posterior fossa. The site and degree of abnormal accumulation varied between early and delayed thallium-201 SPECT images. This unexpected and complicated accumulation of thallium-201 appeared to depend on not only breakdown of the blood-brain barrier but also on the hemodynamics of this type of venous infarction.

Key words: thallium-201, arteriovenous fistula, venous infarction, single photon emission computed tomography (SPECT)

INTRODUCTION

Thallium-201 chloride (thallium-201) single photon emission computed tomography (SPECT) is useful for distinguishing a neoplastic intracranial lesion from non-neoplastic cerebral disease.[1–3] In general, delayed washout and/or accelerated uptake of thallium-201 on delayed images are considered to indicate the neoplastic nature of lesions.[2] Although there have been some cases of abnormal thallium-201 uptake associated with cerebrovascular disease,[2–7] there has been no reported case of venous infarction secondary to an arteriovenous fistula in the posterior fossa exhibiting abnormal thallium-201 uptake in both early and delayed SPECT images. We present here a case of unexpected and paradoxical accumulation of thallium-201 on bilateral thalamic venous infarction confirmed by CT, MRI and angiography.

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For reprint contact: Fumihiko Tamamoto, M.D., Department of Radiology, Tokyo Metropolitan Ohtsuka Hospital, 2–8–1 Minami-Ohtsuka, Toshima-ku, Tokyo 170–8476, JAPAN.

E-mail: tamamoto-o@ohtsuka-hospital.toshima.tokyo.jp

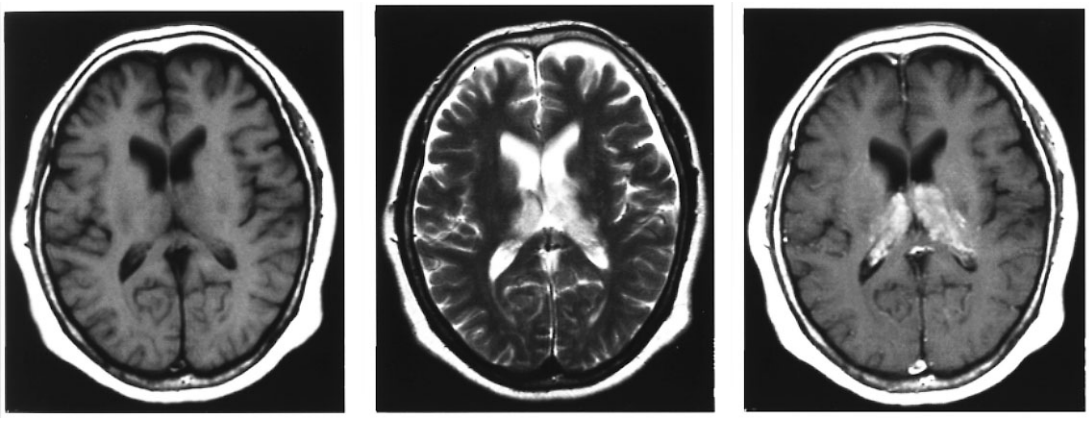
CASE REPORT

A 67-year-old woman visited our hospital with a chief complaint of incontinence and abnormal social behavior of 2-months' duration. Around December 1999, she had become depressed and in February 2000 she had complained of memory disturbance and lethargy. Laboratory data were normal but the Revised Hasegawa Dementia Scale[8] score was 18 points (cut-off is 20/21) and WAIS-R score was I.Q. = 76.

On CT images obtained at admission, blurred hypoattenuated areas were seen in the bilateral thalamic regions and basal ganglia, and after intravenous administration of contrast medium patchy and spotty enhancement effects were visible in the bilateral thalami and basal ganglia.

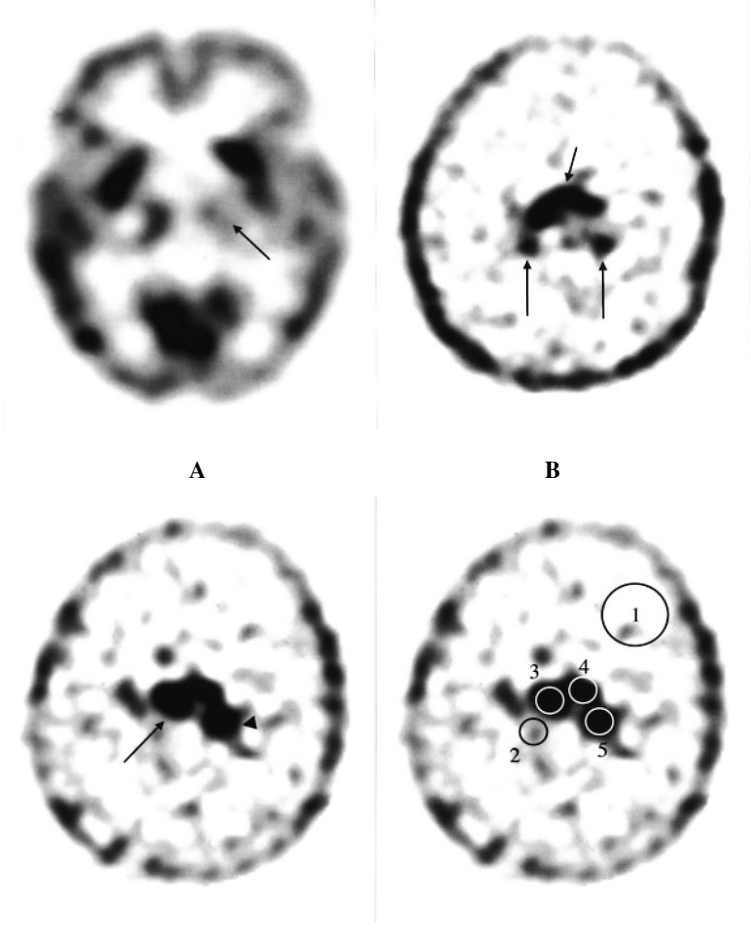
T1-weighted MR images revealed an area of hypointensity in the bilateral thalami, and the lesions were visualized as areas of discrete hyperintensity on T2-weighted MR images. After intravenous administration of gadolinium-diethylene triamine pentaacetic acid (Gd-DTPA), heterogenous enhancement was demonstrated in the lesions (Fig. 1-A, B, C).

Cerebral blood flow (CBF) scintigraphy using technetium-99m ethylcysteinate dimer (^{99m}Tc-ECD)



A B C

Fig. 1 A: Axial unenhanced T1-weighted MR image showing slightly hypointense areas in the bilateral thalamic region. B: Axial T2-weighted image demonstrating areas of distinct hyperintensity in the bilateral thalami. C: Axial gadolinium-diethylene triamine pentaacetic acid (Gd-DTPA) enhanced T1-weighted image showing heterogenous enhancement in the bilateral thalami.



A B

C D

Fig. 2 A: SPECT images obtained using ^{99m}Tc-ECD showing hypoperfusion in the bilateral thalami with left-sided predominance (*arrow*). B: Early (15 minutes after administration of tracer) SPECT images obtained using thallium-201 revealing abnormal uptake in the anterior part of the bilateral thalami (*arrow*) and spotty abnormal accumulation in the posterior part of the bilateral thalami (*long arrow*). C: Delayed (3 hours after administration of tracer) SPECT images revealed that uptake in the anterior part of the thalami was almost the same or partially increased visually (*arrow*) and that uptake in the posterior part of the right thalamus had been washed out. Uptake in the posterior part of the left thalamus was markedly increased (*arrowhead*). D: ROI setting for semiquantitative analysis.

demonstrated bithalamic hypoperfusion with left-sided predominance (Fig. 2-A). Thallium-201 SPECT was performed to differentiate neoplastic lesions from non-neoplastic disease. On the early images (15 minutes after administration of 74 MBq of thallium-201), abnormal uptake was seen mainly in the anterior part of the bilateral thalamic region, and spotty abnormal accumulations were also found in the posterior portion of the bilateral thalami. On delayed images (3 hours after administration of the tracer), the degree of abnormal accumulation in the anterior part of the bilateral thalamic region was visually almost the same or partially increased, and abnormal uptake in the posterior part of the left thalamus was also visually increased. Only spotty abnormal uptake in the posterior part of the right thalamus had been washed out (Fig. 2-B, C). For further investigation of thallium-201 uptake, three indices (early thallium-201 uptake index, delayed thallium-201 uptake index and thallium-201 retained index; D/E index) were calculated based on setting of regions of interest (ROI) in the area of abnormal thallium-201 accumulation (Fig. 2-D). Each uptake index was the ratio of thallium-201 uptake in the lesion (counts/pixel in ROI of the lesion) to that in the area of normal brain parenchyma (counts/pixel in ROI of normal parenchyma) and the D/E index was the ratio of the delayed thallium-201 index to the early thallium-201 index.[2] The D/E index values in the individual ROIs of the abnormal thallium-201 uptake ranged from 0.56 to 1.06 (Table 1).

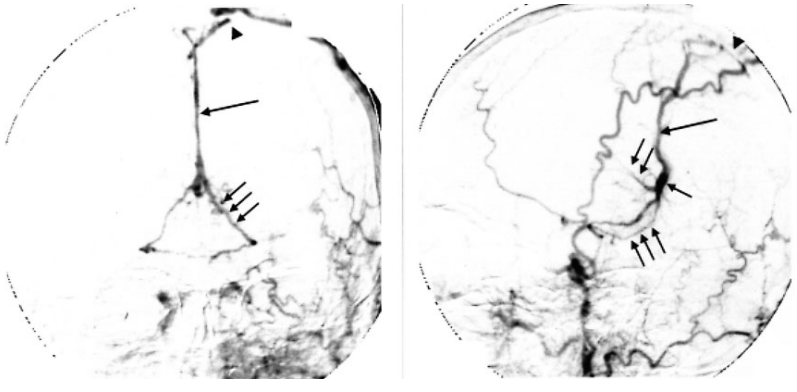
Table 1 Thallium-201 uptake indices and retained index (D/E index) value

No. of ROI	Counts/Pixel		Uptake index		Retained index (D/E index)
	Early	Delayed	Early	Delayed	
1	10.52	18.43	—	—	—
2	36.67	36.08	3.49	1.96	0.58
3	48.81	74.90	4.64	4.06	0.88
4	45.74	66.49	4.35	3.61	0.83
5	35.80	66.28	3.40	3.60	1.06

Angiography confirmed an arteriovenous fistula on the lateral wall of the superior parasagittal sinus. The feeders were the left superficial temporal artery, right middle meningeal artery and meningeal branch of the left vertebral artery. On the left external cervical arteriogram, the fistula was seen on the lateral wall of the superior parasagittal sinus and the dilated abnormal meningeal vein descended along the falx cerebri. Finally, retrograde opacity was seen in the great vein of Galen, internal cerebral vein and basal vein of Rosenthal (Fig. 3-A, B). Neither tumor vessels nor tumor stain was visible. The diagnosis of bithalamic venous infarction induced by increased deep venous pressure around the basal ganglia secondary to arteriovenous fistula was made. Endovascular embolization therapy with polyvinyl alcohol particles was performed for the left superficial temporal artery and right middle meningeal artery. These arteries were successfully embolized, but embolization of the meningeal branch of the left vertebral artery was not performed because of the difficulty of the catheterization. On angiography performed one month after the procedure, the arteriovenous fistula had totally disappeared, and the patient's symptoms had improved. The Revised Hasegawa Dementia Scale score was 23 points.

DISCUSSION

Dementia caused by a thalamic lesion is called "thalamic dementia," and in the case of secondary thalamic dementia due to an arteriovenous fistula in the posterior fossa, as in our patient, it is considered a "reversible dementia," because of improvement of symptoms after endovascular embolization therapy or surgical procedures.[9] In the case of arteriovenous fistula in the posterior fossa, venous



A B

Fig. 3 Left external carotid arteriogram revealing an arteriovenous fistula (*arrowheads*) through the superficial temporal artery (A) and an abnormal meningeal vein (*long arrow*) descending along the falx cerebri (A, B), and retrograde opacification of the great vein of Galen (*single short arrow*), internal cerebral vein (*double short arrows*) and bilateral basal veins of Rosenthal (*triple short arrows*).

ischemia and/or infarction can be induced by chronic passive congestion secondary to retrograde increase in venous pressure toward the venous drainage routes of the thalamus.[9] On the other hand, since the diagnosis of bithalamic abnormality on CT and/or MR images includes tumor infiltration, basilar artery distribution infarct and deep venous occlusion,[10] thallium-201 SPECT is widely performed to distinguish intracranial neoplastic lesions from non-neoplastic abnormalities.[2]

The mechanisms of thallium-201 accumulation in brain tumor include 1) increased Na/K-ATPase pump activity of tumor cells, 2) breakdown of the blood-brain barrier and 3) changes in regional cerebral blood flow. The sensitivity of thallium-201 SPECT for supratentorial tumors is 71.7%, and its specificity is 80.9%.[1] Sun et al.[2] reported that early and delayed thallium-201 SPECT imaging was very useful for distinguishing low-grade from high-grade brain tumors, and that on delayed images radioactivity in high-grade brain tumors tended to increase compared with that in early images.

Few reports have described abnormal thallium-201 uptake in cerebrovascular diseases.[2–7] Bernat et al.[7] and Staffen et al.[3] reported that abnormal thallium-201 uptake was highly dependent on the blood supply in areas of hyperperfused infarct, since luxury perfusion areas depicted by technetium-99m hexamethylpropylene amine oxime (HMPAO) SPECT images and abnormal uptake areas demonstrated by thallium-201 SPECT images exhibited almost the same pattern. In fact, cases of abnormal uptake of thallium-201 reported by Dierckx et al.[1] and Arisaka et al.[5] were of hemorrhagic infarction. These findings also strongly indicate that abnormal accumulation of thallium-201 in an ischemic cerebral lesion mainly depends on the blood supply or net blood volume around the lesion.

In our patient, the abnormal accumulation of thallium-201 varied between early and delayed SPECT images. The early thallium-201 SPECT images depicted abnormal uptake mainly in the anterior portion of the bilateral thalami, and spotty abnormal accumulations were seen in the posterior part of the bilateral thalami. On delayed images, uptake in the anterior part was almost the same but partially increased visually. The spotty abnormal uptake in the posterior part of the left thalamus was also visually increased. Only the spotty uptake seen in the posterior part of the right thalamus had been washed out. In semiquantitative analysis using early and delayed thallium-201 uptake index and D/E index, the D/E index values ranged from 0.56 to 1.06. In two reported cases for which early and delayed thallium-201 SPECT images of cerebrovascular diseases were obtained,[2,5] abnormal accumulation in lesions tended to decrease or wash out on delayed images. In our patient, the D/E index in the posterior part of the right thalamus was especially low (0.56), suggesting the cerebrovascular nature of the lesion, but those in the other abnormal uptake areas ranged from 0.83 to 1.06. Since these D/E index values were higher than those of non-neoplastic diseases, we could not completely rule out neoplastic lesion. Therefore, this abnormal uptake pattern of thallium-201 in our patient was unusual among non-neoplastic or cerebrovascular diseases.

MR images in our case after administration of Gd-DTPA revealed a marked enhancement effect in bilateral thalamic areas, and it was certain that the finding resulted from breakdown of the blood-brain barrier in these areas caused by ischemia and/or infarction secondary to chronic passive congestion. But on the basis of regional cerebral blood flow, the degree of the chronic passive congestion must have varied between right and left thalamic areas because quite different perfusion defects (right side < left side) were encountered on ^{99m}Tc-ECD SPECT images. On the other hand, variation of abnormal accumulation on thallium-201 SPECT images was also seen, and especially on the delayed thallium-201 SPECT images, the abnormal accumulation tended to locate in hypoperfusion areas seen on ^{99m}Tc-ECD SPECT images. These findings in our case seemed to indicate that the abnormal accumulation on the delayed thallium-201 SPECT images was present in the areas with still severer chronic passive congestion induced by arteriovenous fistula.

This unexpected pattern of abnormal accumulation observed bithalamically in our patient appeared to be responsible not only for breakdown of the blood-brain barrier but also for the special hemodynamics (chronic passive congestion) in the arteriovenous fistula. Since this is the first reported case of abnormal thallium-201 uptake in bithalamic venous infarct induced by an arteriovenous fistula, the reason for this unexpected accumulation pattern should be further investigated. However, we believe that under these circumstances, the degree of chronic passive congestion resulting from changes in the pressure balance between arterial and venous sides, and changes in net regional blood supply are strongly responsible for the unexpected and paradoxical uptake of thallium-201.

A bithalamic lesion encountered on CT and/or MR images and an unexpected or paradoxically abnormal uptake pattern of thallium-201 in corresponding areas may be strongly suggestive of cerebrovascular disease with special hemodynamics, such as an arteriovenous fistula.

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