

Regular Article

Watershed infarction associated with dementia and cerebral atrophy

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Abstract

A 63-year-old man was admitted with progressive left hemiparesis and left homonymous hemianopsia of 1 month's duration. During the 2 months before admission, he had suffered from slowly progressive dementia. The diagnosis of right-sided watershed (WS) infarction was made. He exhibited slow progression of dementia and cerebral atrophy during the period of observation after discharge. There was a positive relationship between cerebral atrophy and the degree of dementia. In the present case, WS infarction caused by right internal carotid artery occlusion might be related to dementia and cerebral atrophy.

Key words

carotid artery, cerebral atrophy, cerebral infarction, dementia, watershed infarction.

INTRODUCTION

The incidence of cerebral atrophy increases with age.¹ It has also been demonstrated that patients with Alzheimer's disease and multi-infarct dementia exhibit significantly more severe cerebral atrophy than normal subjects.² Watershed (WS) infarction is a known cause of vascular dementia.^{3,4} Watershed infarcts are ischemic lesions situated along the border zones between the territories of two major arteries (e.g. the anterior and middle or the middle and posterior cerebral arteries). However, the pathogenesis of WS infarcts is still controversial.⁵ We report a case of WS infarction associated with dementia and cerebral atrophy. To our knowledge, this is a rare case featuring progression of dementia and cerebral atrophy following the onset of WS infarction. The longitudinal changes of dementia and cerebral atrophy in WS infarction are also examined.

CASE REPORT

A 63-year-old right-handed man was admitted to the Takamatsu Red Cross Hospital, Kagawa, Japan on 6 November 1996, because of visual disturbance and

gradually progressive weakness of the left arm and leg 1 month previously. During the 2 month period preceding admission, he had developed slowly progressive memory loss and emotional incontinence. He had a history of hypertension but no history of diabetes mellitus nor cardiac disease. On 22 June 1993, he had headache without dementia but brain computed tomography (CT) revealed no infarcted area (Fig. 1a). He had smoked as many as two packs of cigarettes daily for 40 years or longer. His father had died of a stroke. On admission, he appeared alert, orientated, and fluent. He could walk with assistance. His pulse was 65 b.p.m. and regular. Blood pressure was 155/80 mmHg. There was no special findings in the chest or abdomen. The rest of the physical examination was normal.

His intellectual function was mildly impaired, and recent and remote memory were mildly decreased (Clinical Dementia Rating (CDR) 1). The CDR was found to distinguish unambiguously among older subjects with a wide range of cognitive function, from healthy to severely impaired.⁶ The staging of clinical dementia in the present case was rated on a rating of healthy (CDR 0), questionable (CDR 0.5), mild (CDR 1), moderate (CDR 2), or severe dementia (CDR 3). He had mild emotional incontinence, but aphasia, apraxia, and agnosia were not present. Neurological examination revealed left homonymous hemianopsia with preservation of cranial nerve functions. His left arm and leg had about 75% normal movement but there were no extrapyramidal signs.

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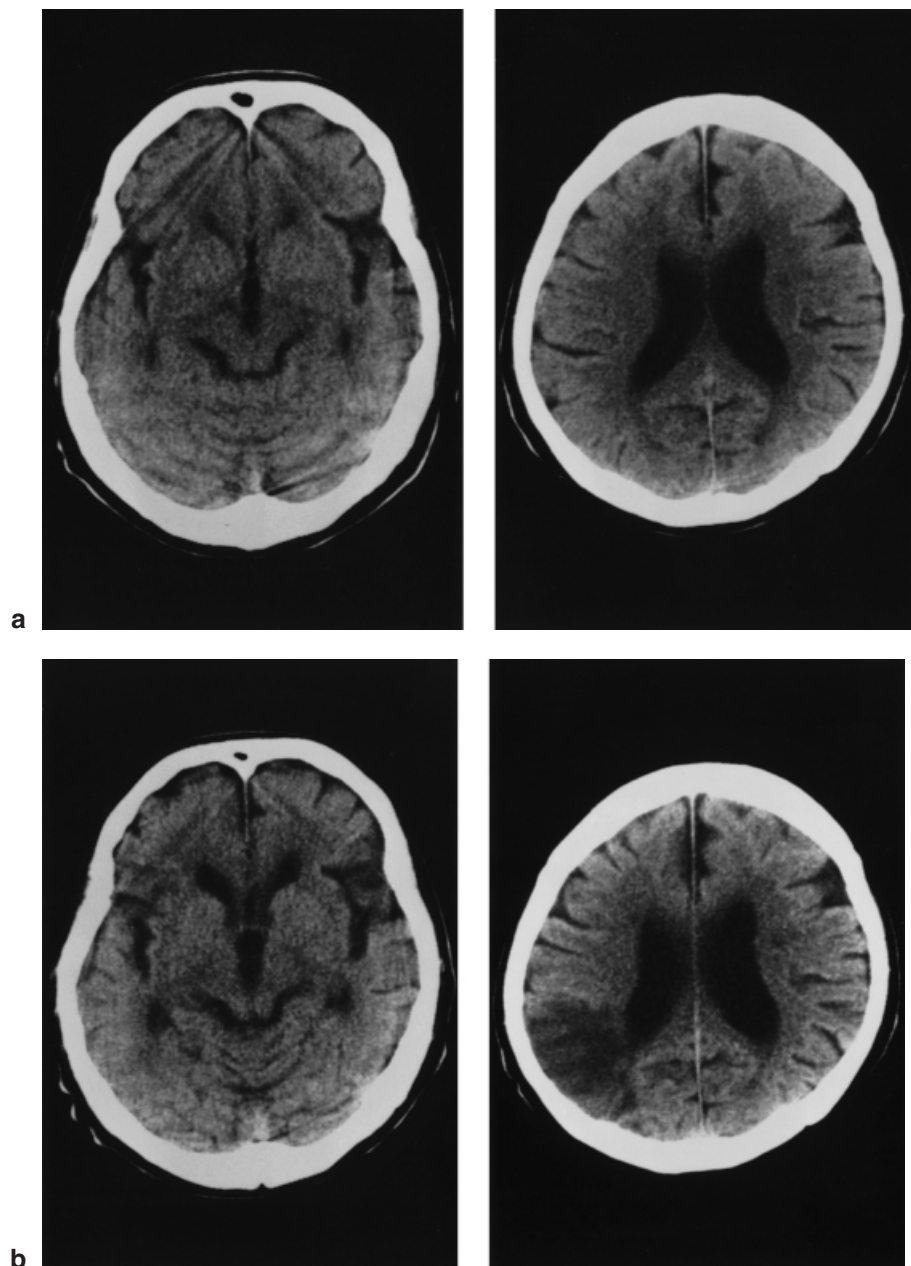


Figure 1. Serial cerebral computed tomographic (CT) examinations on dates shown. (a) On 22 June 1993, brain CT revealed no infarcted area. (b) On 6 November 1996, brain CT revealed mildly enlarged ventricles, mildly enlarged cortical sulci, and a right posterior watershed (WS) infarct. (c) On 5 December 1996, brain CT revealed a mild hypodense lesion appeared in the right anterior WS area. (d) On 23 February 1999, brain CT revealed well-defined right anterior and posterior watershed infarcts and moderate cerebral atrophy.

Muscle tone was normal in all extremities. Coordination was normal. The response to painful stimuli was judged to be nearly normal in all extremities. The deep tendon reflexes were hyperactive in the left upper limb and bilateral legs without pathological reflexes.

Laboratory findings, including a peripheral blood cell count, serum biochemical analysis and urinalysis, were all normal. An electroencephalogram (EEG) obtained 7 days after admission demonstrated a 4–7 Hz theta rhythm of about 10–40 μ V in the right pos-

terior quadrant. Photic stimulation elicited no driving response in the right posterior region on the EEG. Brain CT on admission demonstrated mildly enlarged ventricles, mildly enlarged cortical sulci, and a hypodense lesion in the right posterior WS area (Fig. 1b). Brain MRI demonstrated infarctions in the right posterior WS area and the left corona radiata (Fig. 2). ^{99m}Tc -hexamethylpropyleneamine oxime single photon emission computed tomography (HMPAO-SPECT) imaging revealed hypoperfusion in WS areas over the right hemisphere, and in the left

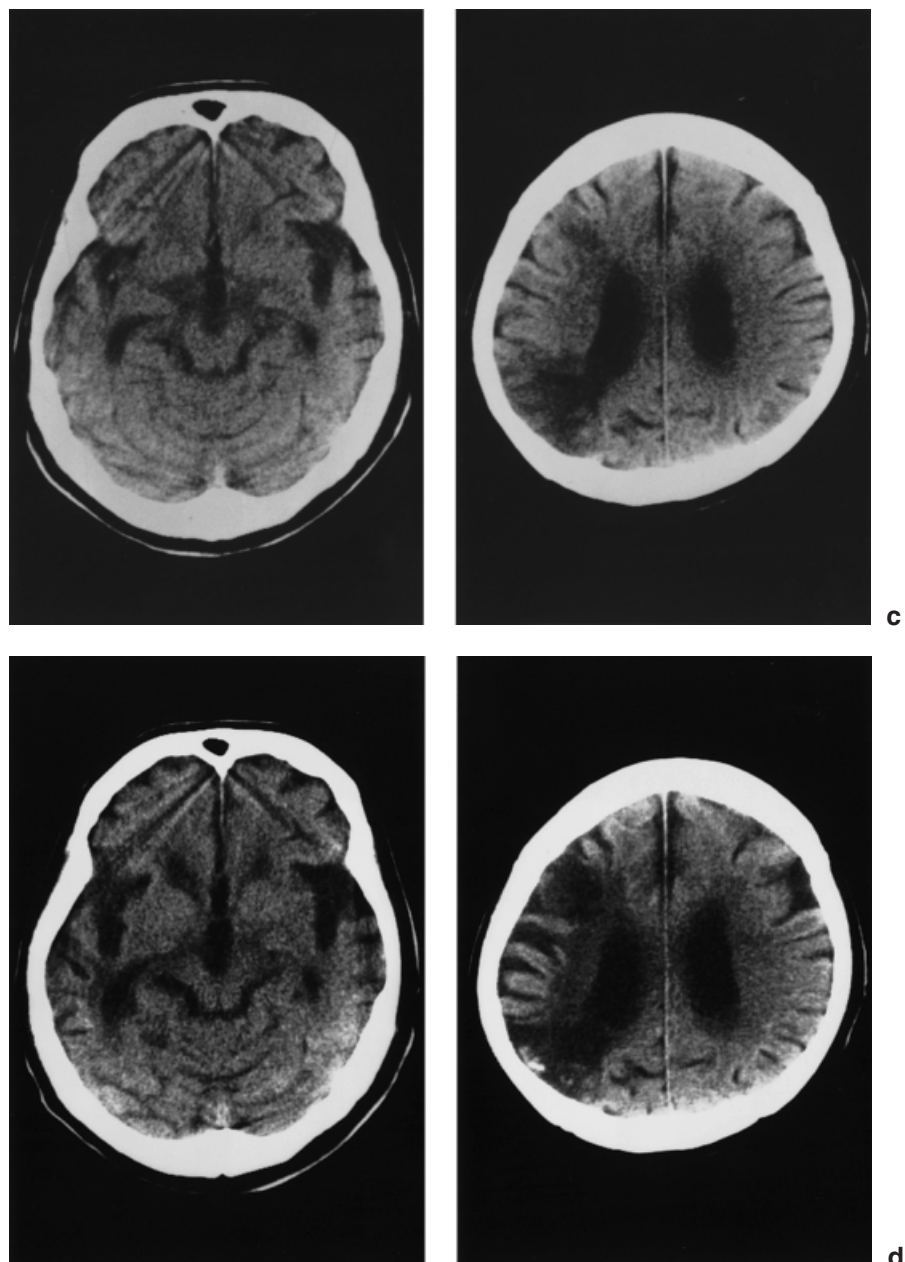


Figure 1. *Continued*

cerebellum (Fig. 3). Magnetic resonance angiography and color-coded duplex ultrasonography of his neck arteries revealed a right internal carotid artery (ICA) occlusion. Based on these findings, we diagnosed WS infarction caused by right ICA occlusion.

The patient's symptoms worsened during the month following admission despite treatment. He developed moderate memory loss, severe emotional incontinence, and was sometimes disoriented to time (CDR 2). On brain CT taken 29 days after admission, a mild hypodense lesion appeared in the right ante-

rior WS area (Fig. 1c). On discharge, he had moderate emotional incontinence (CDR 2). However, progression of dementia was noted during the period of observation following discharge. Two years after discharge, he developed severe memory loss, mild emotional incontinence, and was usually disoriented to place and often to time (CDR 3). However, aphasia, apraxia, and agnosia were not present. His left arm is still disabled, and his left leg had about 75% normal movement (Fig. 4). Brain CT revealed significant progression of cerebral atrophy (Fig. 1d).

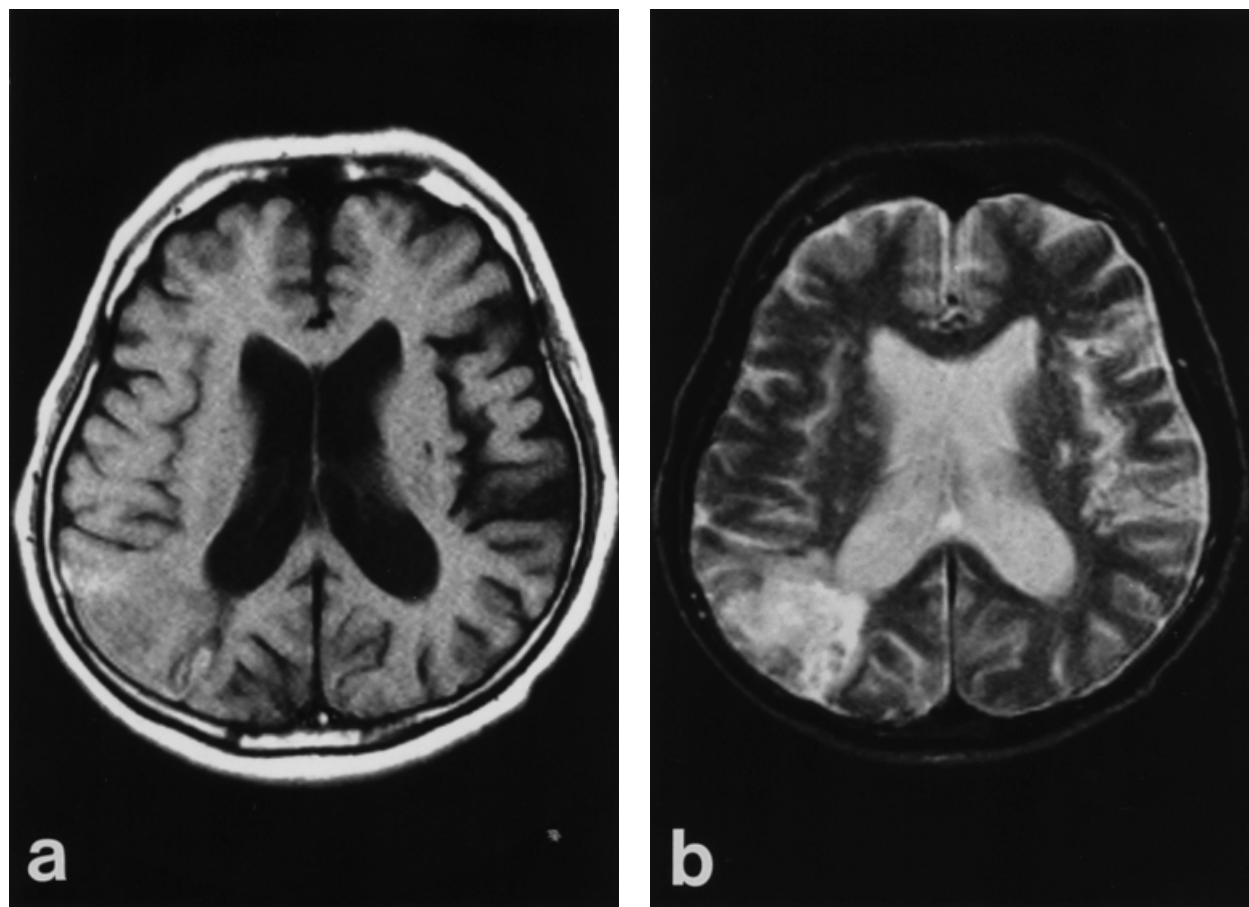


Figure 2. Brain magnetic resonance image 10 days after admission reveals infarctions in the right posterior watershed area and the left corona radiata. (a, T_1 -weighted image; b, T_2 -weighted image).

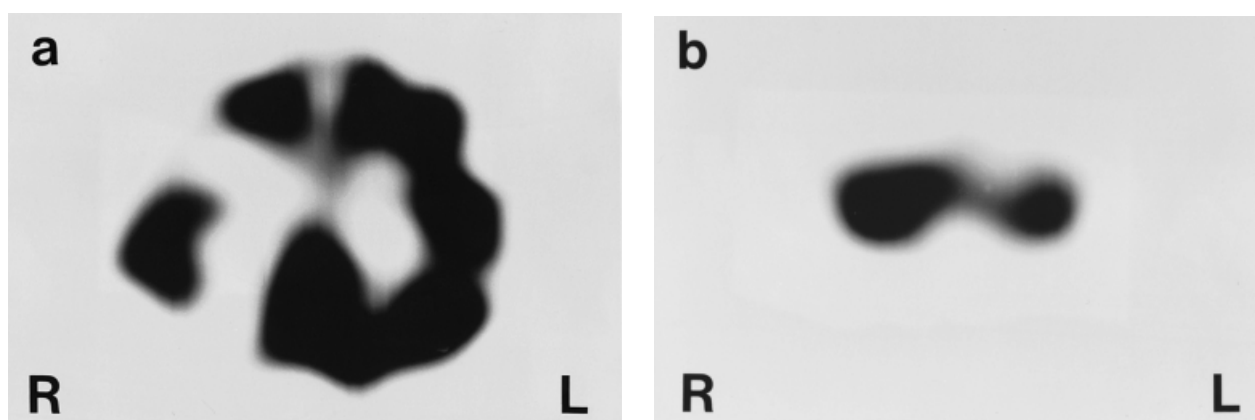


Figure 3. Axial ^{99m}Tc -hexamethylpropyleneamine oxime single photon emission computed tomography taken 16 days after admission shows hypoperfusion in the watershed areas over (a) the right hemisphere and (b) the left cerebellum.

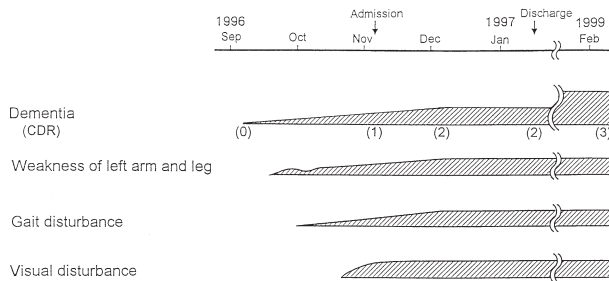


Figure 4. Clinical course of the patient.

DISCUSSION

In the present case, brain CT on admission demonstrated a right posterior WS infarct. In addition, HMPAO-SPECT revealed hypoperfusion in the right anterior WS area. Right cerebral hemisphere lesions may cause crossed cerebellar diaschisis on HMPAO-SPECT.⁷ However, the patient had no clinical signs of cerebellar dysfunction. Binswanger's disease was excluded by the absence of cerebral white matter lesions on brain MRI. The clinical picture consisting of slow progression of dementia and cerebral atrophy on brain CT after discharge suggested WS infarction complicated by Alzheimer's disease. However, the possibility of Alzheimer's disease was remote by the absence of aphasia, apraxia, and agnosia.⁸ Based on these findings, the diagnosis of WS infarction on the right side was made.

Watershed infarcts occur in the border zones between two main artery territories. Unilateral WS infarcts may occur when there is pre-existing ipsilateral vascular disease with focal cerebral hypoperfusion in the farthest periphery during episodes of systemic hypotension.⁹ In the present case, carotid stenosis initially produced inadequate cerebral blood flow (CBF) in the distal branches supplying the borderzone, where perfusion pressure was lowest, and with progression to occlusion, CBF also became insufficient more proximally, producing larger cortical and subcortical infarcts.

There are two types of WS infarcts: those in the WS area between the superficial territories of the anterior and middle cerebral arteries (anterior WS infarct), and those in the WS area between the superficial territories of the middle and posterior cerebral arteries (posterior WS infarct). Anterior WS infarct causes mainly crural hemiparesis sparing the face. With non-dominant hemisphere lesions, mood disturbances (apathy or euphoria) are typical. In posterior WS infarct, hemianopia is the most common abnormality.¹⁰ The present patient's neurological symptoms and signs were compatible with WS infarcts in the non-

dominant hemisphere. The WS infarct is a clinically important cerebral lesion in elderly individuals with vascular dementia. Patients with multi-infarct dementia have many lacunar infarcts, but those with WS infarction have infarcts in both the cerebral cortex and white matter. Of WS infarctions caused by ICA occlusion, those which are posterior cause amnesia.³ A total of 82% of patients with WS infarcts had dementia. Demented patients with WS infarcts in the right hemisphere are twice as frequent as those with infarcts in the left hemisphere. Watershed infarcts in the bilateral frontal lobes produce severe dementia.⁴ In the present case, the anterior and posterior WS infarcts in the right hemisphere and diffuse ischemic lesions caused by right ICA occlusion might have been etiologically related to the patient's dementia.

Many CT studies have reported that cerebral atrophy progresses with advancing age.¹¹ Follow-up studies showed that progression of cerebral atrophy is observed in 44.4% of patients with transient ischemic attacks and reversible ischemic neurological deficit.¹² Moreover, in patients with chronic cerebrovascular disease (CVD), progression of cerebral atrophy preceded the changes in CBF without new neurological symptoms.¹³ There have thus been many studies concerning CBF and cerebral atrophy in CVD, but no longitudinal study of the relationship between dementia and cerebral atrophy has been reported. In our patient, WS infarction caused by right ICA occlusion might be related to dementia and cerebral atrophy. If a patient presents with progressive dementia and cerebral atrophy, ICA occlusive disease must be considered along with the more common causes of Alzheimer's disease.

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