

Cite this article as: Neural Regen Res. 2012;7(12):943-947.

Cortical infarction of the right parietal lobe and neurogenic heart disease

A report of three cases[★]

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Abstract

Three male patients were diagnosed with new cortical infarctions of the right parietal lobe on the basis of head magnetic resonance imaging; high-intensity signals indicating lesions in the right parietal lobe were noted on diffusion-weighted images at admission. Two of them presented with left hand weakness, and one exhibited left upper limb weakness. Treatment for improving blood supply to the brain was administered. One patient died suddenly because of ventricular fibrillation 3 days after admission. The other two patients had increased troponin levels and abnormal electrocardiograms, and were diagnosed with acute myocardial infarction half a month after admission. When lesions exist in field 7 of the parietal cortex (resulting in paralysis of the contralateral hand), the sympathetic center of the posterior lateral nucleus of the hypothalamus demonstrates compensatory excitement, which easily causes tachyarrhythmia and sudden death. Our experimental findings indicate that close electrocardiograph monitoring and cerebral infarction treatment should be standard procedures to predict and help prevent heart disease in patients with cerebral infarction in the right parietal lobe and left upper limb weakness as the main complaint.

Key Words: left upper limb weakness; right parietal lobe; cortical infarction; neurogenic heart disease; autonomic nerve; case report

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Received: 2011-11-10
Accepted: 2012-02-06
(N20111207002/WJ)

Li F, Jia YJ. Cortical infarction of the right parietal lobe and neurogenic heart disease: a report of three cases. Neural Regen Res. 2012;7(12): 943-947.

www.crter.cn
www.nrronline.org

doi:10.3969/j.issn.1673-5374.2012.12.011

INTRODUCTION

Neurocardiology is a multifaceted discipline that comprises three major areas of study: effect of the heart on the brain (e.g., cardiac-source embolic stroke), effect of the brain on the heart (e.g., neurogenic heart disease), and neurocardiac syndromes (e.g., Friedreich's disease)^[1].

Cerebrovascular diseases arising from injury to certain brain areas, including the insular cortex, hypothalamus, limbic system and brain stem, may lead to dysfunction of the autonomic nervous system, imbalance between the sympathetic and parasympathetic nervous systems, and pathological manifestations in body organs. A number of studies have investigated the relationship between heart disease and lesions of the insular cortex, hypothalamus, limbic system and brain stem^[1-3]. However, the relationship between lesions in the parietal cortex and the onset of heart disease remains unclear.

In this study, we report one case of sudden

cardiac death and two cases of non-fatal myocardial infarction caused by injury to the right parietal cortex following stroke.

CASE REPORT

Clinical information

Clinical information on the three cases is shown in Table 1 and Figure 1.

Cardiac examination

Cardiac examination results at admission and after admission for the three cases are shown in Figure 2 and Table 2.

DISCUSSION

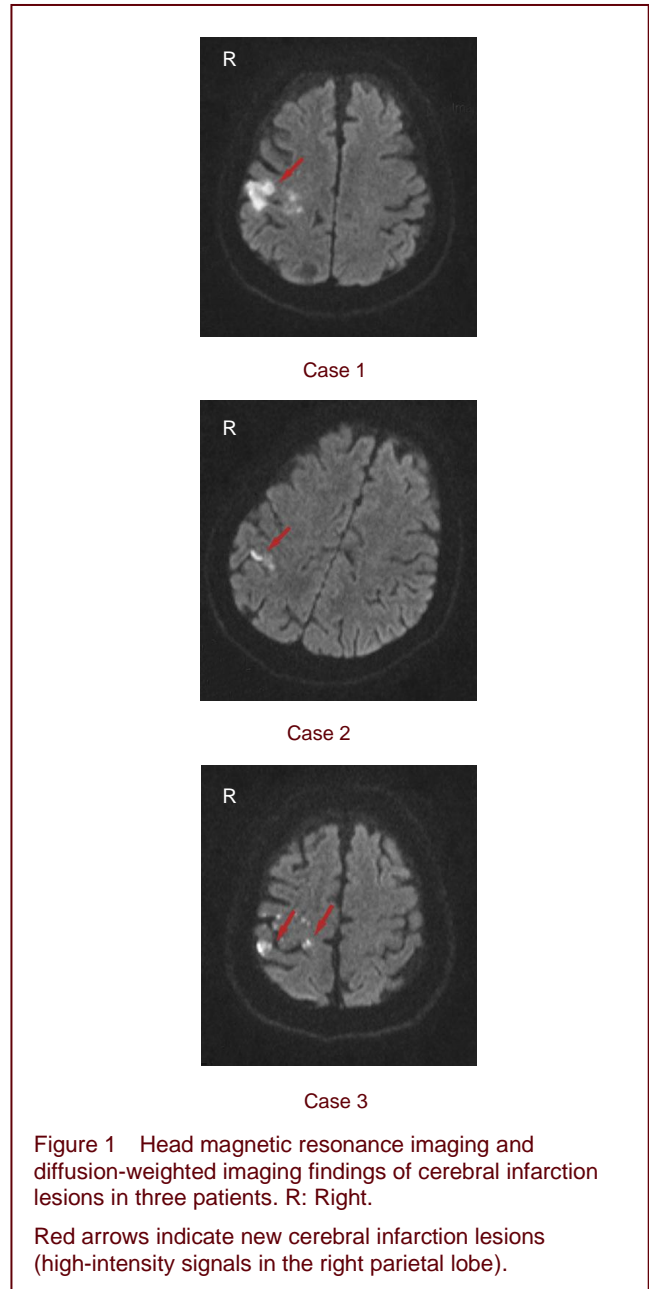
In this report, we describe three male patients with cortical infarction with no history of heart disease. Clinical symptoms were very mild during cerebral infarction. All patients had monoplegia, but no sensory disturbance. The patients experienced no cardiac symptoms during the cerebral infarction attack. Conventional cardiac examinations were performed at admission, including electrocardiography, myocardial enzyme levels and troponin level; all results were normal.

Table 1 Clinical information on three cases with new infarction in the right parietal cortex

Item	Case 1
General information	Male, 65 years old
Chief complaint	Left hand weakness for 3 days
Medical history	No
Physical signs at admission	The grip strength of the left hand is grade 2
Head MRI-DWI at admission (Figure 1)	High-intensity signals in the right parietal lobe ^a
Treatment strategy	Improving blood supply to the brain
Changes in condition	On the afternoon of the third day after admission, the patient complained of breath shortness, and his blood pressure had dropped to 90/60 mm Hg (1 mm Hg=0.133 kPa). The patient was re-examined by electrocardiograph, and he died of ventricular fibrillation during the electrocardiograph test.
Outcome	Sudden death
Item	Case 2
General information	Male, 60 years old
Chief complaint	Left hand weakness for 1 day
Medical history	Hypertension for 10 years
Physical signs at admission	The grip strength of the left hand is grade 3
Head MRI-DWI at admission (Figure 1)	High-intensity signals in the right parietal lobe
Treatment strategy	Improving blood supply to the brain and fibrinolytic therapy
Changes in condition	At 16 days after admission, the patient experienced a sudden tightness in the chest, and then transferred to the cardiology department for further treatment.
Outcome	Improved
Item	Case 3
General information	Male, 62 years old
Chief complaint	Left upper limb weakness for 4 hours
Medical history	Hypertension for 10 years
Physical signs at admission	The muscle strength of the left upper limb is grade 4
Head MRI-DWI at admission (Figure 1)	High-intensity signals in the right parietal lobe
Treatment strategy	Improving blood supply to the brain
Changes in condition	At 14 days after admission, the patient presented with shortness of breath and wheezing, and the patient was diagnosed with acute non-ST-segment elevation myocardial infarction. Wheezing improved and the troponin level returned to normal after treatment for coronary distension.
Outcome	Improved
MRI-DWI: Magnetic resonance imaging and diffusion-weighted imaging. ^a Head MRI-DWI suggested a new infarction of the right parietal lobe.	

One patient, who had presented with left hand weakness, died suddenly because of ventricular fibrillation 3 days after admission, while another patient with left hand weakness had an acute myocardial infarction half a month after admission. The third patient, who also

initially presented with left upper limb weakness, had non-ST-segment elevation myocardial infarction half a month after admission. All cerebral infarctions were located in the right parietal cortex. Patients with more pronounced weakness in the distal end of the limb had more severe heart disease.



When investigating such cases, the following questions should be considered: Which problem is more serious, cerebral infarction or heart disease? Is the heart disease caused by a brain lesion or by a primary cardiac lesion? Are these cases representative of neurogenic heart disease? Is the right parietal cortex the site of neurogenic heart disease? Do cerebral lesions closer to functional areas controlling left hand movement induce more serious heart attacks? Are the incidences and severity of neurogenic heart disease (including the time interval

between heart disease and cerebral infarction) related to the location of cerebral infarction rather than cerebral infarction size? Finally, what is the mechanism of neurogenic heart disease, and could it involve a role of the autonomic nervous system? All of these questions require further research to be answered.

In terms of the clinical course of the three patients, cerebral infarction was the first diagnosis and cardiac symptoms were not initially apparent. However, the ensuing heart episodes appear to be associated with cerebral infarction in time of onset and in stroke location. This suggests that left hand or upper limb weakness may be a predictor of neurogenic heart disease.

Table 2 Cardiac examination of three cases with new infarctions in the right parietal cortex	
Item	Case 1
At admission	
Myocardial creatine kinase isoenzyme (U/L)	6
Troponin level (ng/mL)	0.001
Electrocardiograph	Normal
After admission	
Myocardial creatine kinase isoenzyme (U/L)	Not tested
Troponin level (ng/mL)	Not tested
Electrocardiograph	Ventricular fibrillation
Item	Case 2
At admission	
Myocardial creatine kinase isoenzyme (U/L)	2
Troponin level (ng/mL)	0.002
Electrocardiograph	Normal
After admission	
Myocardial creatine kinase isoenzyme (U/L)	140
Troponin level (ng/mL)	0.825
Electrocardiograph	ST V ₁ -V ₃ elevation of 0.1-0.3 mV, inverted T V ₁ -V ₃ (acute myocardial infarction)
Item	Case 3
At admission	
Myocardial creatine kinase isoenzyme (U/L)	6
Troponin level (ng/mL)	0.059
Electrocardiograph	Normal
After admission	
Myocardial creatine kinase isoenzyme (U/L)	80
Troponin level (ng/mL)	0.413
Electrocardiograph	ST V ₂ -V ₆ depression of 0.05-0.1 mV, inverted and flat T V ₂ -V ₆ (acute non-ST-segment elevation myocardial infarction)

In studies addressing stroke location and its association with fatal cardiac outcome, Rincon *et al*^[4] concluded that clinical diagnosis of infarction in the left parietal lobe was related to the occurrence of fatal heart disease or myocardial infarction and that imaging diagnosis of

infarction in the left or right parietal lobe was associated with fatal heart disease or myocardial infarction.

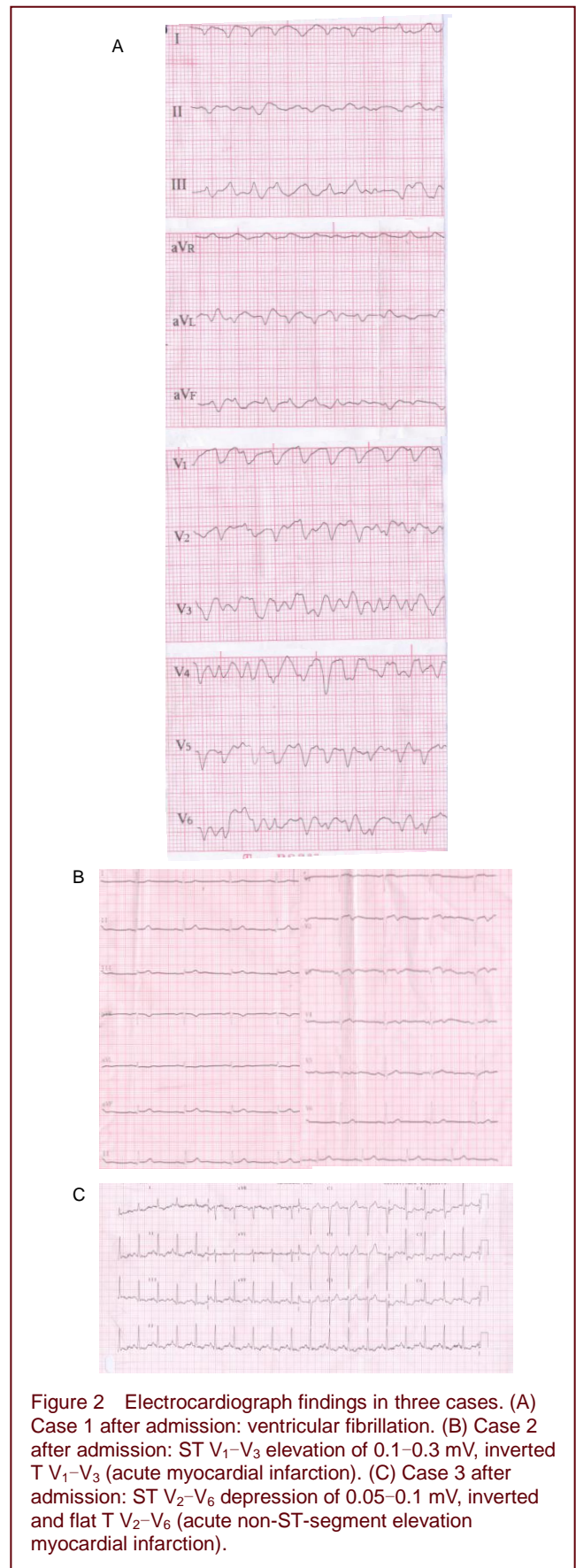


Figure 2 Electrocardiograph findings in three cases. (A) Case 1 after admission: ventricular fibrillation. (B) Case 2 after admission: ST V₁-V₃ elevation of 0.1-0.3 mV, inverted T V₁-V₃ (acute myocardial infarction). (C) Case 3 after admission: ST V₂-V₆ depression of 0.05-0.1 mV, inverted and flat T V₂-V₆ (acute non-ST-segment elevation myocardial infarction).

Notably, frontal, temporal and insular lobe infarctions were not associated with fatal heart disease. Hilz *et al*^[5] found that right parietal lobe infarction is a major risk factor for fatal heart disease, even when taking into account the size of the infarction.

In studies investigating the relationships between serum troponin level, stroke location and prognosis, Song *et al*^[6] concluded that insular infarction was associated with elevated serum troponin levels. In a previous study using functional magnetic resonance imaging in normal patients^[7], the right parietal cortical region was activated after repeated movement of the left hand, confirming that functional areas controlling the left hand are located in the parietal cortex of the right hemisphere. "Deadly left hand," as the authors summarized, is basically consistent with Rincon's "deadly parietal lobe"^[4]. We speculate that the severity of neurogenic heart disease may be related to the site of cerebral infarction, which is often localized to areas involved in the control of left hand movement.

On the basis of Brodmann's partition scheme for the cerebral cortex^[8], Ipekchyan^[9] conducted studies investigating the quantitative distribution of efferent fibers in the cortex and hypothalamus after creating local lesions in fields 5 and 7 of the parietal cortex. Their study showed that efferent fibers from fields 5 and 7 of the parietal cortex terminated in thalamic reticular nuclei and parafascicular nuclei, with more fibers from field 5 compared with field 7. In contrast, there were more efferent fibers from field 7 in the posterior lateral nucleus of the thalamus than from field 5, while very few efferent fibers originated from the thalamic central lateral nucleus, ventral anterior nucleus and occipital parietal cortex. Different functional areas of the cerebral cortex have their own distinct areas for autonomic nervous functions, which are located near the corresponding motor areas or overlap with these areas^[10].

The parietal cortex is divided into three areas, with the superior parietal area comprising fields 5 and 7. The superior parietal area, especially the superior parietal lobule, is involved in controlling sophisticated movement of the contralateral limb^[11]. Bilateral posterior lesions in the superior parietal lobule can cause hand movement disorders under visual guidance^[12]. When lesions exist in field 7 of the parietal cortex (resulting in paralysis of the contralateral hand), the sympathetic center of the posterior lateral nucleus of the hypothalamus demonstrates compensatory excitement, which can easily cause tachyarrhythmia and sudden death. Fields 5 and 7 comprise the sensory representative area of the cortex. In the present study, all three patients had decreased muscle strength and sensory disturbances that were not clearly identified. The lack of multimodal sensory feedback, due to parietal lobe lesions, could explain unilateral akinesia without loss of feeling^[13]. All three patients^[14] had right hemisphere lesions. In a previous study, Naver and colleagues confirmed that asymmetric distribution of cardiac autonomic nerves in

the cerebral hemispheres was an important factor determining death-related heart rate variability. Risk of sudden death after cerebral infarction was found to be related to infarction site and side, with right hemisphere infarctions posing a higher risk. The right hemisphere is known to mainly regulate sympathetic nerve activity^[15]. Decreases in cardiac parasympathetic activity caused by right hemisphere infarction were more obvious than those caused by left cerebral hemisphere infarction^[16]. Lesions in the cerebral hemispheres after acute infarction induced different types of arrhythmias with varying severity. Lesions in the right hemisphere were more likely to cause tachyarrhythmias^[17-18].

A number of studies have attributed the cause of neurocardiological disease to catecholamine infusion, stress with or without administration of steroids, nervous system stimulation and reperfusion, the essential feature of which is sympathetic overactivity with secondary catecholamine toxicity^[19]. In addition, cardiovascular autonomic failure may also result from impaired parasympathetic function^[20].

SUMMARY

The clinical characteristics of the three patients with neurogenic heart disease suggest that symptoms of myocardial ischemia in early stages of the disease are not stereotypical. None of the three patients showed abnormalities in electrocardiograph examination at admission, and the only complaint was left hand or upper limb weakness. Consequently, the doctors focused on brain diseases rather than heart abnormalities. Thus, in all cases, the doctors failed to make the correct diagnoses and interventions.

Many reports have shown that patients with acute cerebral infarction often do not report cardiac symptoms due to aphasia and speech disturbances caused by the stroke. In addition, heart attack symptoms are frequently not typical and may be easily concealed by infection and shock. Asymptomatic myocardial ischemia also occurs frequently and is easily overlooked, resulting in poor prognosis. The first patient in this report was not monitored for cardiac changes that may have indicated heart disease after admission, and he died suddenly during electrocardiograph retesting. This suggests that electrocardiography and determination of cardiac enzyme and troponin levels should be a part of routine examination of these patients to ensure early diagnosis of the disease. Therefore, we suggest that close monitoring of electrocardiographs and dynamic attention to troponin and cardiac enzyme levels both at and after admission should be standard procedure for patients with cerebral infarction and upper limb weakness (especially those patients with lesions in the right parietal lobe) as the main complaint. This may facilitate the early diagnosis of neurogenic heart disease and allow for the administration of preventative treatment, thereby reducing mortality.

In this study, coronary angiography was not performed on the patients. Therefore, we cannot completely rule out the presence of primary cardiac disease which may have followed cerebral infarction. In addition, the number of cases is small. Thus, further observation and research are needed to confirm our findings.

Author contributions: All authors made important scientific contributions to the study and are thoroughly familiar with the primary data.

Conflicts of interest: None declared.

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(Edited by Min LQ, Wu Y/Yang Y/Song LP)