

CASE REPORT

Dilated cardiomyopathy-related stroke mimicking large-artery atherosclerosis-related stroke: report of two cases

Guozhen Qiu^{1,†}, Lijie Ren^{2,3,†}, Hongliang Jiang⁴, Xin Shi⁵, Liming Cao^{2,3,*}

¹Department of Neurology, The Third Affiliated Hospital of Shenzhen University, 518000 Shenzhen, Guangdong, China

²Department of Neurology, The First Affiliated Hospital of Shenzhen University, 518000 Shenzhen, Guangdong, China

³Department of Neurology, Shenzhen Second People's Hospital, 518000 Shenzhen, Guangdong, China

⁴Department of Neurology, The Third People's Hospital of Yiyang City, 413000 Yiyang, Hunan, China

⁵Business School, Manchester Metropolitan University, M15 6BH Manchester, UK

***Correspondence**

caolm-2007@163.com

(Liming Cao)

† These authors contributed equally.

Abstract

The clinical characteristics and treatment of stroke caused by dilated cardiomyopathy (DCM) are not clear, especially in patients with large-artery atherosclerosis (LAA)-related stenosis, which commonly causes acute ischemic stroke (AIS); therefore, the diagnosis and treatment of such patients are challenging. Herein, we summarize the clinical characteristics and suggest clues to guide the diagnosis and treatment of two cases. Case 1: A 67-year-old woman with a history of DCM presented with sudden-onset slurred speech and left limb weakness (>2 hours duration), which worsened after intravenous thrombolysis. Repeated brain computed tomography showed no hemorrhage; thus, cerebral artery occlusion or embolism was suspected. Emergency magnetic resonance imaging (MRI) and angiography (MRA) revealed acute multiple bilateral cerebral infarctions and severe left middle cerebral artery stenosis, respectively. We considered a DCM-related stroke and administered anticoagulation therapy. Subsequently, the patient's symptoms improved significantly, and she was discharged on day 9, after showing no abnormal neurological signs. Case 2: A 49-year-old man with a history of DCM presented with acute headache and blurred vision for 4 days. MRI and MRA revealed multiple acute cerebral infarctions and left vertebral artery stenosis, respectively. We considered an LAA-related stroke and administered antiplatelet and cholesterol-lowering drugs. Eventually, the patient was discharged on day 13, after his right-sided hemianopia improved significantly. Both patients had LAA, which can be easily misdiagnosed as a stroke. LAA-related and DCM-related stroke need to be differentiated. DCM-related AIS lesions are often distributed in the areas supplied by the different cerebral arteries. It is necessary to carefully analyze the shape, location, and scope of the lesions, and identify the main causes of stroke. Anticoagulant therapy is preferred for DCM-related AIS.

Keywords

Dilated cardiomyopathy; Stroke; Large artery atherosclerosis; Anticoagulation therapy; Case report

1. Introduction

Dilated cardiomyopathy (DCM) is defined by the presence of left ventricular (LV) dilatation and contractile dysfunction, manifesting as congestive heart failure, circulatory collapse, arrhythmias, and thromboembolic events [1]. DCM has a prevalence and annual incidence of 40 and 7 cases per 100,000 individuals, respectively [2–4]. Cardioembolic strokes lead to the highest in-hospital mortality rate at approximately around 20%, with poor short-term prognosis for most cases [5]. DCM is a rare cause of cardiogenic acute ischemic stroke (AIS) [6]. Although DCM increases the risk of stroke, only a few reports describe such cases. The clinical characteristics and treatment of stroke caused by DCM are also not clear, especially in patients with large-artery atherosclerotic stenosis, which com-

monly causes AIS; therefore, the diagnosis and treatment of such patients are challenging. The cause may be misidentified as arteriosclerosis and mistreated with antiplatelet agents or even emergency endovascular therapy. Herein, we summarize the clinical characteristics and suggest clues to guide the diagnosis and treatment of two cases based on our literature review.

2. Case report

2.1 Case 1

A 67-year-old woman was admitted to our hospital with slurred speech and left limb weakness (>2 hours duration) in March 2019. The patient had left heart failure, DCM, and frequent ventricular premature beats for several years, with no other

TABLE 1. Comparison of our two patients with similar cases published in the literature.

	Jeon <i>et al.</i> [7]	Ho <i>et al.</i> [8]	Karande <i>et al.</i> [9]	Dogan <i>et al.</i> [10]	Case 1	Case 2
Age/Sex	40-year-old/male	34-year-old/male	11-year-old/male	8-year-old/female	67-year-old/female	49-year-old/male
Main symptoms	Mild hemiparesis on the right side	Generalized tonic-clonic seizures	Hemiplegia and facial weakness	Left hemiplegia	Slurred speech and left limb weakness	Headache and blurred vision
Echocardiography	Severe global LV EF of 19% systolic dysfunction, dilatation (LV diameter of 65 cm in systole) and a large mobile LV thrombus	A dilated LV and LA, generalized hypokinesia with poor LV EF of 15–20%, and a mass in the LV apex	Dilatation of all 4 chambers with global LV dysfunction, EF of 15–20%, with multiple intracardiac clots	Dilated cardiomyopathy was identified	LA and LV enlargement (Fig. 1e) with moderate reflux (Fig. 1f); LV wall with diffuse hypokinesia; LV diastolic and systolic dysfunction; LVEF 28%, LV diameter of 63 cm in diastole	LA and LV enlargement (Fig. 1l); LV wall with diffuse hypokinesia and regional wall motion abnormality; LV diastolic and systolic dysfunction; LVEF 27%, LV diameter of 60 cm in diastole
Chest radiography	Mild cardiomegaly	–	Cardiomegaly with pulmonary congestion	–	Enlargement of cardiac shadow (CTR: 0.66, Fig. 1g) with LV enlargement	Cardiac enlargement (CTR: 0.55)
Electrocardiogram	non-specific ST segmental change	Marked LV hypertrophy	Sinus tachycardia and S-T segment depression pattern	–	Frequent ventricular premature beats	LV hypertrophy with ST-T changes
MR imaging	Spotty ischemic brain lesions	Infarctions over the frontal and temporal lobes; bilateral parietal and occipital; cerebellum	Infarctions in the area of caudate nuclei, putamen, brain stem and cerebellum	Infarction (31 mm × 14 mm) at the right basal ganglia	Acute infarction in the bilateral basal ganglia and right hippocampus (Fig. 1a,b)	Multiple acute infarctions and softening lesions in the left cerebellar hemisphere and occipital lobe (Fig. 1h–j)
MR angiography	–	Basilar artery, the flow signal distal to the left P3 is not visualized	–	Occlusion was detected at the M2 of the right MCA	Stenosis of the M2 segment of the MCA (Fig. 1c,d)	Cerebral arteriosclerosis with mild left vertebral artery stenosis (Fig. 1k)
Treatment	LMWH, urgent surgical removal for thrombus, and warfarin substituted	Unfractionated heparin and regular warfarin substituted and aspirin	Heparin × 7d and oral warfarin substituted	Nadroparin calcium	Argatroban ×6 d and warfarin substituted; furosemide, spironolactone and acupuncture	Clopidogrel, atorvastatin calcium, valsartan, diuretics, hypoglycemic drugs for 13 days
Outcomes and follow-up results	At 1-year follow-up, the patient was well, and his cardiac function was normalized (LV EF of 59%; LV diameter of 48 cm)	The 37th hospital day with retained visual field defect and ataxia. Follow-up showed the LV EF increased to 40% and complete resolution of the thrombus	All thrombi, except one, disappeared. The patient died 2.5 months later due to resistant cardiac failure	Symptoms resolved by the following 7th week. neurologic examinations were normal	Condition improved on day 2 post-admission (NIHSS: 1). Patient was discharged on day 9 without abnormal neurological signs (NIHSS: 0)	Patient was discharged on day 13 after right-sided hemianopia improved significantly (NIHSS: 1)

CTR, cardiothoracic ratio; DCM, dilated cardiomyopathy; EF, ejection fraction; LV, left ventricular; LA, left atrium; LMWH, Low molecular weight heparin; MCA, middle cerebral artery; MR, Magnetic resonance; NIHSS, National Institutes of Health Stroke Scale; “–” means not provided.

relevant medical or family history. Physical and neurological examination revealed normal mental status, motor aphasia, muscle strength of 4/5 in the left upper and lower extremities, a positive left pyramidal sign, and an enlarged cardiac boundary. Her National Institutes of Health Stroke Scale (NIHSS) score was 3. Emergency brain computed tomography (CT) revealed an old lacunar infarction in the left basal ganglia. The patient immediately underwent intravenous thrombolysis with a tissue-type plasminogen activator (0.6 mg/kg), and her NIHSS score dropped to 0 within an hour postoperatively. However, her condition worsened 1 hour later. Physical examination revealed lethargy, motor aphasia, muscle strength of 3/5 and 1/5 in the left and right extremities, respectively, and positive bilateral pyramidal signs. Her NIHSS score was 12. Repeated brain CT showed no hemorrhage, and cerebral artery reocclusion or cerebral embolism was suspected. Intravenous butylphthalide and edaravone were administered. Her red (RBC) and white (WBC) blood cell counts, and cholesterol, glycosylated hemoglobin, serum creatinine, and troponin T levels were within normal ranges. The patient's brain natriuretic peptide (BNP) level (3565 pg/mL) was increased. Magnetic resonance imaging (MRI) and angiography (MRA) revealed acute multiple bilateral cerebral infarctions (Fig. 1a,b) and severe left middle cerebral artery (MCA) stenosis (Fig. 1c,d), respectively. We considered a DCM-related stroke and administered anticoagulation therapy. Subsequently, her symptoms significantly improved, and she was discharged on day 9, after showing no abnormal neurological signs. Other auxiliary examinations (Fig. 1e–g) and the specific treatment plan and outcomes are described in Table 1 (Ref. [7–10]). The patient was satisfied with her treatment.

2.2 Case 2

A 49-year-old man was admitted to our hospital in June 2019 for headache and blurred vision for 4 days. His medical history included hypertension and diabetes, and DCM and chronic cardiac insufficiency for >8 and >3 years, respectively. He had smoked for nearly 20 years but denied alcoholism or intoxication. His family history was unremarkable. Physical examination revealed a blood pressure of 139/95 mmHg, an enlarged cardiac boundary, normal mental status, right-sided hemianopia, and a negative pyramidal sign (NIHSS score, 2). Laboratory findings showed normal WBC and RBC counts and elevated glycosylated hemoglobin (11.6%), serum creatinine (117 $\mu\text{mol/L}$), troponin T (0.038 ng/mL), and BNP (2465 pg/mL) levels. MRI revealed multiple acute cerebral infarctions in the left cerebellar hemisphere and occipital lobe (Fig. 1h–j). MRA revealed left vertebral artery stenosis (Fig. 1k). We considered a large-artery atherosclerosis (LAA)-related stroke and administered antiplatelet and cholesterol-lowering drugs. He was eventually discharged on day 13, after his right-sided hemianopia significantly improved (NIHSS score, 1). Other auxiliary examinations (Fig. 1l), treatment plan, and outcomes are detailed in Table 1. The patient was satisfied with his treatment.

3. Discussion

Cases of DCM-related stroke are rarely reported. AIS and DCM patients who have LAA can be easily misdiagnosed as having an LAA-related stroke. Despite our initial consideration, both patients had multiple cerebral infarctions that could not be attributed to a responsible artery. Hence, we suspected DCM-related stroke, despite its rarity as a stroke etiology. An important feature of DCM-related AIS is that the lesions are distributed in the areas supplied by different cerebral arteries, as was observed in our patients.

Specifically, in Case 1, the severe stenosis in the M2 segment of the left MCA was striking and could easily be misdiagnosed as a responsible artery. However, multiple acute foci of AIS were not completely in the area dominated by the MCA, with some supplied by the posterior cerebral (Fig. 2a,b) and anterior choroidal arteries (Fig. 2c,d). A chronic atherosclerotic stenosis of the MCA was more likely, because an embolism of the MCA M2 segment likely caused a large infarction in the left insula; however, diffusion-weighted imaging only showed small patchy infarctions, and no previous MRA was available for comparison. The simultaneous appearance of the new infarct in both cerebral hemispheres suggested a cardiogenic stroke, and we speculated that the deterioration following improvement with intravenous thrombolysis was probably caused by disintegration of the intracardiac embolus and formation of multiple cerebral emboli. In Case 2, the acute infarction focus was distributed in the cerebellum and occipital lobe, supplied by the posterior and anterior inferior cerebellar arteries, and posterior cerebral artery, respectively (Fig. 2e–h). Hence, the left vertebral artery stenosis was difficult to explain. In other words, we consider the severe intracranial stenosis as clinically silent in both cases. Consequently, it was considered as a cardiogenic embolism secondary to DCM, as related AIS lesions are distributed in areas supplied by the different cerebral arteries [7–9].

According to the [Chinese guidelines for diagnosing and treating dilated cardiomyopathy](#) [11], the clinical diagnostic criteria of DCM are objective evidence of ventricular enlargement and decreased myocardial systolic function as follows: LV end-diastolic dimension (LVEDd) of >5.0 cm and >5.5 cm in females and males, respectively; and LV ejection fraction of <45%. Both patients (see Table 1) met the above diagnostic criteria. Echocardiography is important not only for diagnosing DCM but also for detecting intracardiac thrombus and assessing cardiac function. Post-stroke echocardiography for 3–5 days did not reveal intracardiac emboli in both cases, as was similar to another report [10], possibly because the thrombi were autolytic, dissolved by thrombolytics/anticoagulants, or expelled from the heart after the stroke. If an intracardiac thrombus had been diagnosed, emergency surgery was required [7].

Genetic causes, endocrine disorders, collagen vascular diseases, drugs, congenital metabolism diseases, muscular dystrophies, structural heart diseases, acute and chronic myocarditis, and toxins can be considered as etiologic factors of DCM. However, 50% of the cases are idiopathic, which are similar to our cases [10]. Typical DCM includes signs of LV dilatation and contractile dysfunction. LV thrombus [12],

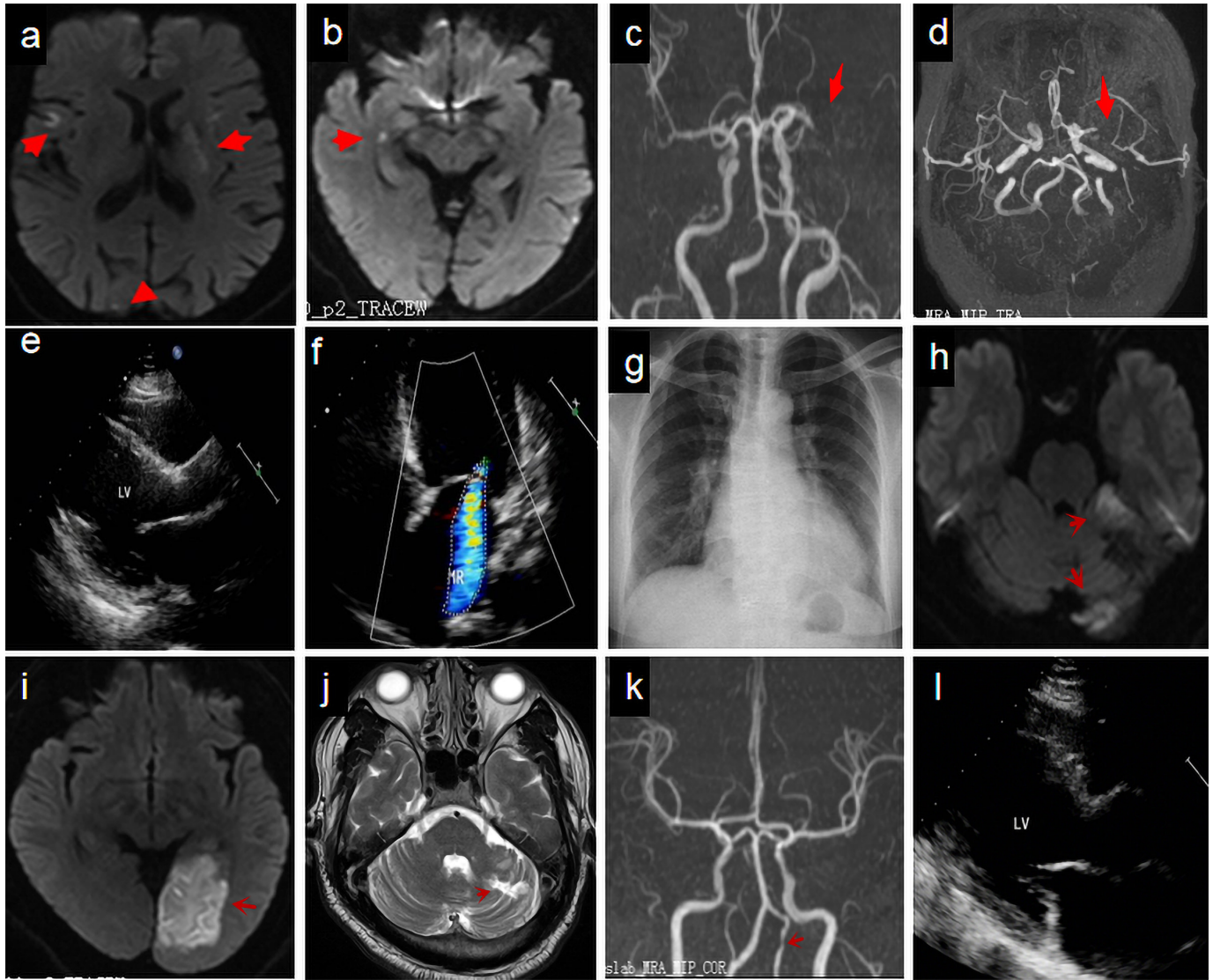


FIGURE 1. Imaging and echocardiography results.

Findings from Case 1. Diffusion-weighted magnetic resonance imaging showed acute infarction lesions in the left basal ganglia, right temporal lobe, right occipital lobe (a, arrows), and right hippocampus (b, arrow). Magnetic resonance angiography (MRA) revealed severe stenosis or occlusion of the M2 segment of the middle cerebral artery (arrow), with compensatory meningeal collateral circulation (c,d). Echocardiography showed left atrial and left ventricular (LV) enlargement (e, LV diastolic diameter, 63 cm) with moderate reflux (f). Chest radiography showed cardiac enlargement (cardiothoracic ratio, 0.66), mainly in the left ventricle (g).

Findings from Case 2. Diffusion-weighted sequence demonstrated multiple acute cerebral infarctions in the left cerebellar hemisphere (h, arrows) and left occipital lobe (i, arrow). T2-weighted sequence showed multiple softening left cerebellar lesions (j, arrow). MRA revealed cerebral atherosclerosis with mild left vertebral artery stenosis (k, arrow). Color Doppler echocardiography showed left atrial and ventricular enlargement (l, LV diastolic diameter, 60 cm).

LV systolic impairment, and heart failure [13] independently increase stroke risk. A mural thrombus in the enlarged atrium or ventricle (specifically, the left ventricle) might have been dislodged and entered the systemic circulation, resulting in a cerebral embolism (Fig. 3). Clinical features suggestive of cardioembolic stroke, including sudden-onset maximum deficits and decreased levels of consciousness, Wernicke’s aphasia or global aphasia without hemiparesis, Valsalva maneuver at stroke onset, and simultaneous cerebral and systemic emboli [14]. Lacunar clinical presentations, such as single or multiple infarcts, make cardioembolic origin unlikely [14].

Theoretically, anticoagulant therapy is preferred for cardioembolic stroke, as it can not only prevent thromboembolic events but also effectively dissolve an existing cardiac thrombus and improve cardiac function [7–9]. The timing of anticoagulant use in AIS patients remains a difficult issue, needing assessment of a complex benefit-risk balance. The 2016 ESC Guidelines stated that the initiation of anticoagulant therapy is determined by the NIHSS score, which indicates severity. Specifically, anticoagulant therapy can be started after 3 days and after 6–12 days for mild and moderate-to-severe stroke, respectively [15]. Anticoagulant administration

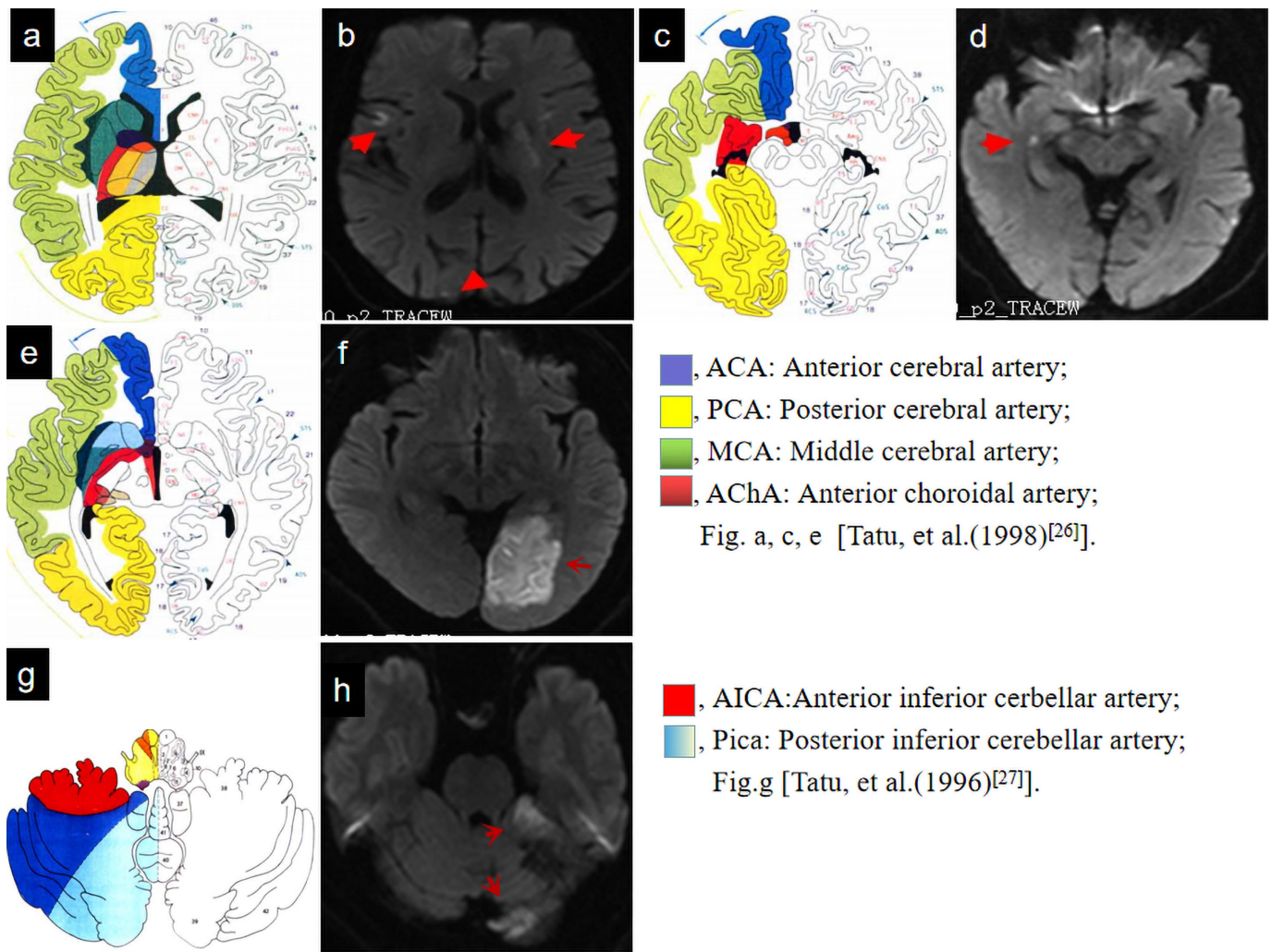


FIGURE 2. The different territories involved by acute ischemic stroke lesions. Fig. 2a,c,e [26], Fig. 2g [27].

In Case 1, multiple acute foci were supplied by the bilateral middle cerebral (a,b), posterior cerebral (a,b), and anterior choroidal (c,d) arteries.

In Case 2, the acute infarction lesions were supplied by the posterior cerebral (e,f), and the posterior and anterior inferior cerebellar (g,h) arteries.

within 72 hours after the stroke is not recommended [16]. If the patient's risk of an intracerebral hemorrhage or general bleeding is transiently increased, waiting for ≥ 2 weeks after the stroke is recommended [16]. If this risk persists, anti-coagulant administration may be further delayed. Based on our initial experience, infarct size is also directly proportional to the risk of hemorrhagic transformation, warranting more cautious anticoagulant use. After appropriate treatment, the outcome of DCM-related stroke is good if cardiac function is stable.

Patient 1 presented with a progressive stroke; therefore, the treatment is challenging. We used butylphthalide and edaravone to stabilize the patient. Butylphthalide has improved the symptoms and long-term prognosis of stroke [17], primarily by improving microcirculation and protecting the mitochondria, in addition to its antioxidant, anti-apoptotic, anti-inflammatory, and anti-thrombotic properties [17, 18]. Edaravone, a free radical scavenger, is a potentially useful addition to thrombolytic therapy in AIS patients [19] as it improves the recanalization rate, reduces the incidence of

intracranial hemorrhage, and improves prognosis [20]. Edaravone can inhibit tissue damage, causing cerebral edema, and delay neuronal death caused by AIS [21]. It also offers good neuroprotection against diabetic stroke by interrupting the endoplasmic reticulum stress-mediated apoptotic pathways [22]. Other drugs and surgical treatments for progressive stroke include ezetimibe [23], early superficial temporal artery-MCA double anastomoses, which result in rapid neurological improvement in patients with progressive stroke due to main trunk artery occlusion [24], and percutaneous transluminal angioplasty for associated vertebral artery stenosis [25].

A limitation of this study is that it described only two cases. A large-sample, multi-institution research program is needed in this respect.

4. Conclusions

Our findings can further reinforce the understanding of the clinical features of DCM-related stroke for more accurate diagnosis and treatment. LAA-related stroke needs to be distin-

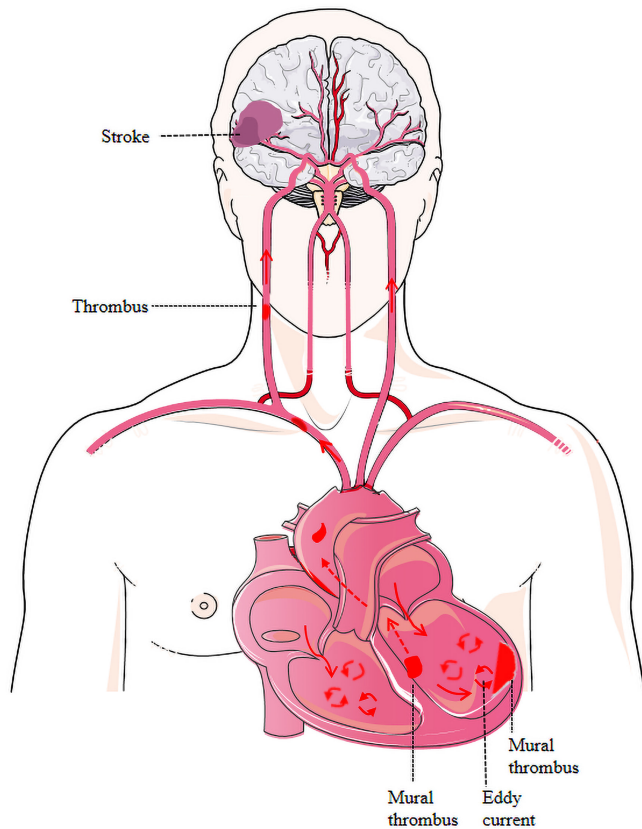


FIGURE 3. Graphical abstract.

Patients with dilated cardiomyopathy can develop hemodynamic disorders, such as eddy currents in the dilated ventricles, and hemodynamic disorders can easily result in intracardiac mural thrombi, especially in the dilated LV. In this case, the mural thrombus might have become dislodged and entered the systemic circulation, resulting in a cerebral embolism.

guished from DCM-related stroke. DCM-related AIS lesions are often distributed in the areas supplied by various arteries. It is necessary to carefully analyze the shape, location, scope of the lesions, and identify the main causes of stroke. Anti-coagulant therapy is preferred for DCM-related AIS. A more comprehensive understanding of the pathogenesis of DCM-related AIS is needed.

AUTHOR CONTRIBUTIONS

LC designed the study and revised the manuscript. GQ wrote the manuscript. LR performed the research, provided advice on the discussion. HJ provided part-fund and participated in proofreading; XS provided some constructive opinions. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study design was approved by the ethics review board of the Third Affiliated Hospital of Shenzhen University (No: 2019SZLH-LW-006). Informed consent was obtained from all included participants.

ACKNOWLEDGMENT

We would like to thank Editage (www.editage.com) for English language editing.

FUNDING

This work was supported by the Science and Technology Innovation Foundation of Shenzhen (JCYJ20180302153449519 and KJYY20180703165202011), and Sanming Project of Medicine in Shenzhen (SZSM201801014). Those funds have been used to finance the article processing charge. The funders had no influence on the conduct or writing of this article.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

CONSENT FOR PUBLICATION

We obtained written consent for publication from the patients.

AVAILABILITY OF DATA

All data related to this case report are contained within the manuscript.

REFERENCES

- [1] Weintraub RG, Semsarian C, Macdonald P. Dilated cardiomyopathy. *Lancet*. 2017; 390: 400–414.
- [2] Maron BJ, Towbin JA, Thiene G, Antzelevitch C, Corrado D, Arnett D, *et al*. Contemporary definitions and classification of the cardiomyopathies: an American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation*. 2006; 113: 1807–1816.
- [3] Manolio TA, Baughman KL, Rodeheffer R, Pearson TA, Bristow JD, Michels VV, *et al*. Prevalence and etiology of idiopathic dilated cardiomyopathy (summary of a National Heart, Lung, and Blood Institute workshop). *The American Journal of Cardiology*. 1992; 69: 1458–1466.
- [4] Taylor MRG, Carniel E, Mestroni L. Cardiomyopathy, familial dilated. *Orphanet Journal of Rare Diseases*. 2006; 1: 27.
- [5] Arboix A, Garcia-Eroles L, Massons J, Oliveres M. Predictive clinical factors of in-hospital mortality in 231 consecutive patients with cardioembolic cerebral infarction. *Cerebrovascular Diseases*. 1998; 8: 8–13.
- [6] Zhdanova SG, Petrikov SS, Ramazanov GR, Khamidova LT, Aliev IS, Sarkisyan ZO. Dilated cardiomyopathy as a cause of ischemic stroke. *Zhurnal Nevrologii i Psikiatrii Imeni S.S. Korsakova*. 2016; 116: 44–47.
- [7] Jeon GJ, Song BG, Park YH, Kang GH, Chun WJ, Oh JH. Acute Stroke and Limb Ischemia Secondary to Catastrophic Massive Intracardiac Thrombus in a 40-Year-Old Patient with Dilated Cardiomyopathy. *Cardiology Research*. 2012; 3: 37–40.
- [8] Ho CH, Lin YY, Wu YC, Chu SJ, Tsai SH. Postictal confusion as the initial presentation of dilated cardiomyopathy: a case report and review of literatures. *Journal of Internal Medicine of Taiwan*. 2010; 21: 133–139.
- [9] Karande SC, Kulthe SG, Lahiri KR, Jain MK. Embolic stroke in a child with idiopathic dilated cardiomyopathy. *Journal of Postgraduate Medicine*. 1996; 42: 84–86.

- [10] Doğan M, Peker E, Cagan E, Akbayram S, Acikgoz M, Caksen H, *et al.* Stroke and dilated cardiomyopathy associated with celiac disease. *World Journal of Gastroenterology*. 2010; 16: 2302–2304.
- [11] Yang J, Liao Y, Yuan J, Wang Z, Cheng X, Zhao D. Chinese guidelines for diagnosis and treatment of dilated cardiomyopathy. *Journal of Clinical Cardiology*. 2018; 34: 421–434. (In Chinese)
- [12] Crawford TC, Smith WT, Velazquez EJ, Taylor SM, Jollis JG, Kisslo J. Prognostic usefulness of left ventricular thrombus by echocardiography in dilated cardiomyopathy in predicting stroke, transient ischemic attack, and death. *The American Journal of Cardiology*. 2004; 93: 500–503.
- [13] Agarwal M, Apostolakis S, Lane DA, Lip GYH. The impact of heart failure and left ventricular dysfunction in predicting stroke, thromboembolism, and mortality in atrial fibrillation patients: a systematic review. *Clinical Therapeutics*. 2014; 36: 1135–1144.
- [14] Arboix A, Alió J. Acute cardioembolic stroke: an update. *Expert Review of Cardiovascular Therapy*. 2011; 9: 367–379.
- [15] Kirchhof P, Benussi S, Kotecha D, Ahlsson A, Atar D, Casadei B, *et al.* 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. *Europace*. 2016; 18: 1609–1678.
- [16] Boursier-Bossy V, Zuber M, Emmerich J. Ischemic stroke and non-valvular atrial fibrillation: when to introduce anticoagulant therapy? *JMV-Journal De Médecine Vasculaire*. 2020; 45: 72–80.
- [17] Wang S, Ma F, Huang L, Zhang Y, Peng Y, Xing C, *et al.* DL-3-n-Butylphthalide (NBP): a Promising Therapeutic Agent for Ischemic Stroke. *CNS & Neurological Disorders Drug Targets*. 2018; 17: 338–347.
- [18] Chen X, Qiu K, Liu H, He Q, Bai J, Lu W. Application and prospects of butylphthalide for the treatment of neurologic diseases. *Chinese Medical Journal*. 2019; 132: 1467–1477.
- [19] Kikuchi K, Miura N, Kawahara K, Murai Y, Morioka M, Lapchak PA, *et al.* Edaravone (Radicut), a free radical scavenger, is a potentially useful addition to thrombolytic therapy in patients with acute ischemic stroke. *Biomedical Reports*. 2013; 1: 7–12.
- [20] Takenaka K, Kato M, Yamauti K, Hayashi K. Simultaneous administration of recombinant tissue plasminogen activator and edaravone in acute cerebral ischemic stroke patients. *Journal of Stroke and Cerebrovascular Diseases*. 2014; 23: 2748–2752.
- [21] Li F, Zhao L, Shi Y, Liang J. Edaravone-Loaded Macrophage-Derived Exosomes Enhance Neuroprotection in the Rat Permanent Middle Cerebral Artery Occlusion Model of Stroke. *Molecular Pharmaceutics*. 2020; 17: 3192–3201.
- [22] Srinivasan K, Sharma SS. Edaravone offers neuroprotection in a diabetic stroke model via inhibition of endoplasmic reticulum stress. *Basic & Clinical Pharmacology & Toxicology*. 2012; 110: 133–140.
- [23] Yang L, Zhao P, Zhao J, Wang J, Shi L, Wang X. Effects of ezetimibe and anticoagulant combined therapy on progressing stroke: a randomized, placebo-controlled study. *Journal of Neurology*. 2016; 263: 2438–2445.
- [24] Inoue A, Kohno K, Iwata S, Ohue S, Ozaki S, Ninomiya S, *et al.* Efficacy of Early Superficial Temporal Artery-Middle Cerebral Artery Double Anastomoses for Atherosclerotic Occlusion in Patients with Progressing Stroke. *Journal of Stroke and Cerebrovascular Diseases*. 2017; 26: 741–748.
- [25] Hayashi K, Matsuo Y, Toyoda K, Hayashi Y, Shirakawa K, Kaminogo M. [a Case of Vertebral Artery Stenosis Presenting with Progressing Stroke and Treated by Percutaneous Transluminal Angioplasty] no Shinkei Geka. *Neurological Surgery*. 2016; 44: 383–389.
- [26] Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of the human brain: cerebral hemispheres. *Neurology*. 1998; 50: 1699–1708.
- [27] Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of human brain. *Neurology*. 1996; 47: 1125–1135.

How to cite this article: Guozhen Qiu, Lijie Ren, Hongliang JiangXin Shi, Liming Cao. Dilated cardiomyopathy-related stroke mimicking large-artery atherosclerosis-related stroke: report of two cases. *Signa Vitae*. 2021;17(6):150-156. doi:10.22514/sv.2021.211.