



## Case Report

# Treatment of the Left Ventricular Thrombus with Integrated Traditional Chinese and Western Medicine: A Case Report



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### Abstract

Left ventricular thrombus (LVT) is a common complication of coronary heart disease; thus, traditional Chinese medicine has become a therapeutic option. Here we report two LVT cases treated with Western medicine and traditional Chinese herbal medicine. After treatment, the thrombus became obviously smaller than before, and the clinical symptoms were also relieved. Furthermore, there was little relevant literature in this field, so we expect that this report could support the efficacy of traditional Chinese medicine in treating LVT.

### Introduction

Left ventricular thrombus (LVT) is a common clinical complication, which is often caused by acute myocardial infarction (AMI), dilated cardiomyopathy, stress cardiomyopathy, etc. Since 1995, through the application of percutaneous coronary intervention (PCI), the incidence of LVT in AMI patients has dropped from 33% to 10%.<sup>1</sup> In the era of PCI, the incidence of LVT in anterior myocardial infarction patients was 4–15%.<sup>2,3</sup> However, LVT's main clinical hazard has been systemic circulation embolism with a high rate of disability and fatality. Therefore, timely diagnosis and treatment could effectively reduce the incidence of severe embolism, and significantly favor the prognosis of the patients. In these two cases, LVT was found by an ultrasonic cardiogram (UCG), and the thrombus shrank significantly after the treatment of integrated traditional Chinese and Western medicine.

**Keywords:** Left ventricular thrombus; Integrated traditional Chinese and Western medicine; Complementary medicine; Decoction.

**Abbreviations:** AMI, acute myocardial infarction; APTT, activate partial thromboplastin time; BNP, brain natriuretic peptide; CK, creatine kinase; ECG, electrocardiogram; ET, endothelin; FIB, fibrinogen; HDL-C, high-density lipoprotein; LDH, lactate dehydrogenase; LDL-C, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; LVT, left ventricular thrombus; NOAC, new oral anticoagulant; PCI, percutaneous coronary intervention; PT, prothrombin time; TC, total cholesterol; TG, triglyceride; UCG, ultrasonic cardiogram.

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### Case presentation

#### Case 1

A male, 60 years old, was admitted to the Guangdong Provincial Hospital of Chinese Medicine-Zhuhai Hospital on May 31, 2019, because of recurrent shortness of breath for over one month, that had aggravated for two days. Present medical history: Shortness of breath at rest and aggravation after activities with a little chest tightness, difficulty in lying horizontally, edema of both lower limbs, insomnia, and loss of appetite. His past medical history showed heart failure, atrial fibrillation, and an enlarged heart without any family history. Physical examinations showed weak respiratory sounds of both lungs, extended cardiac dullness, and a low and blunt heart sound. The tongue was pale and dark with a white and greasy coating, and the pulse was string-like and slippery. Laboratory examinations indicated aspartate aminotransferase of 70.0 U/L, lactate dehydrogenase (LDH) of 1,114 U/L, creatine kinase (CK) of 241.6 U/L brain natriuretic peptide (BNP) of 6,785 pg/mL, myoglobin of 99.48 ng/mL, cTnT of 816.3 pg/mL, INR of 1.3, D-dimer of 8.35 mg/L, HbA1c of 6.8%, CHOL of 4.79 mmol/L, TG of 1.78 mmol/L, LDL-C of 3.55 mmol/L, and HDL-C of 0.76 mmol/L. The results of the electrocardiogram (ECG): Left atrium hypertrophy, left ventricular hypertrophy, T wave change, and left axis deviation. UCG: EF of 35%, enlargement of the heart, general decline in the wall activity, a change of dilated cardiomyopathy was considered, left heart insufficiency (severe), right heart insufficiency (mild), multiple solid masses in the left ventricular, multiple hypoechoic and hyperechoic masses in the left ventricular apex (41 × 17 mm, 33 × 12 mm, and 36 × 12 mm) (high likelihood of thrombosis), aortic and mitral regurgitation (mild), tricuspid regurgitation (moderate), pulmonary hypertension (mild), and pericardial effusion (small amount) (Fig. 1). Chest X-ray: 1. Increased bronchovascular shadows, 2. enlarged heart shadow, widened right

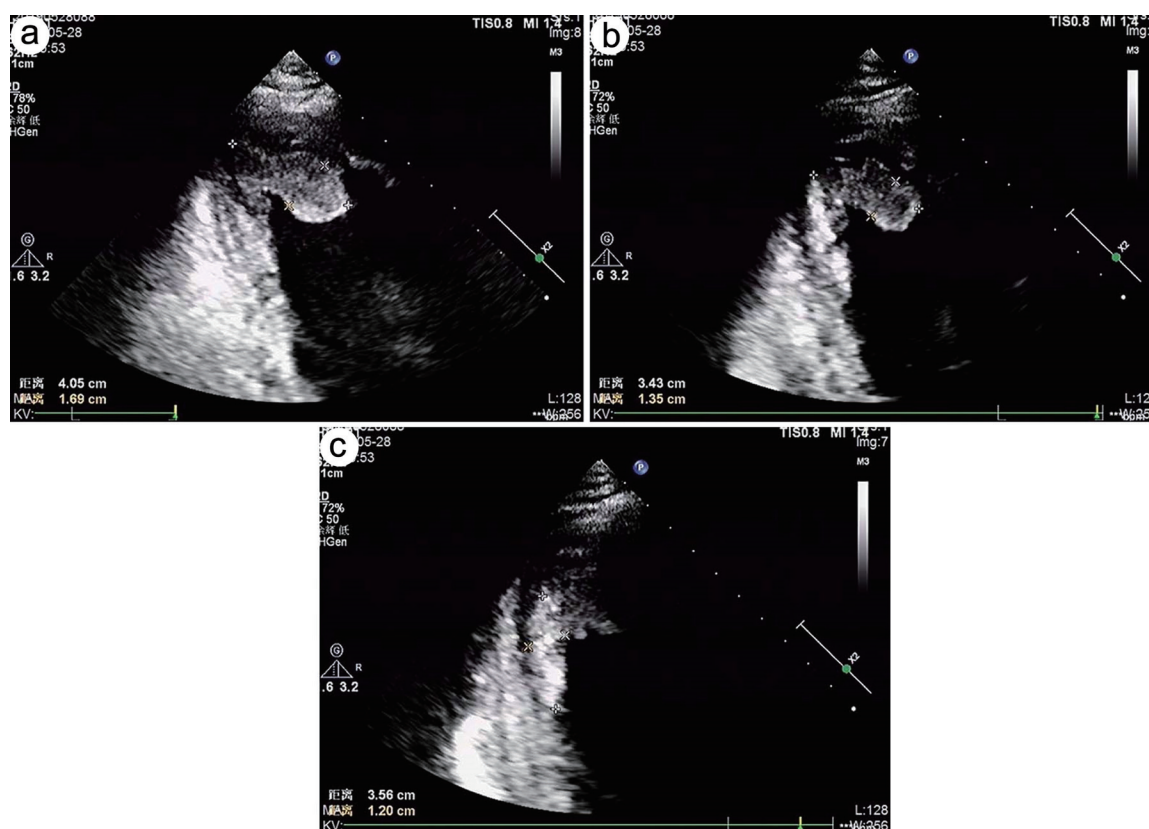


Fig. 1. Admission UCG of case 1. UCG, ultrasonic cardiogram.

lower pulmonary trunk, aortic tortuosity, and calcification, and 3. bilateral costophrenic angles becoming blunt, and pleura thickening. Color doppler ultrasound of the cervical and vertebral arteries: Slight incassation of the intima-media membrane of the bilateral carotid arteries, plaque in the right side, slight tortuosity of the bilateral vertebral arteries, and reduction of the diameter and blood flow of the right side. Color doppler ultrasonography of the veins of both lower extremities: Dysfunction with regurgitation (severe) of the bilateral femoral-saphenous vein valves and great saphenous vein valves, and varicosity of the great saphenous vein.

Western medicine diagnosis: 1. Acute left heart failure, 2. suspected coronary heart disease, 3. dilated cardiomyopathy, 4. left ventricular thrombus, 5. non-rheumatic tricuspid regurgitation (moderate), 6. hyperlipidemia, 7. carotid arteriosclerosis (with plaque), and 8. varicosity (lower limbs). Western medicine treatment: Aspirin (100 mg, qd, po) and clopidogrel (75 mg, qd, po) for antiplatelet aggregation, rivaroxaban (15 mg, qd, po) to prevent aggravation of the ventricular thrombus, enoxaparin (0.4 ml, qd) for anticoagulation; digoxin tablets (0.125 mg, qd, po) for the cardiotonic effect, metoprolol (12.5 mg, bid, po) to control the blood pressure and heart rate, furosemide (20 mg, bid, po) and spironolactone (20mg, bid, po) for diuresis and improving myocardial remodeling, benazepril (5 mg, qd, po) or valsartan (80 mg, qd, po) to reduce blood pressure, isosorbide mononitrate (20 mg, qd, po) to expand the coronary arteries, rosuvastatin calcium tablets (20 mg, qd, po) to reduce the lipid level and plaque, potassium chloride (1 g, qd, po) sustained release tablets as a supplement, and oxygen inhalation for life support.

Chinese medical diagnosis: Heart failure disease (syndrome of

*Yang* deficiency and flooding). Etiology and pathogenesis: The patient's chest tightness and shortness of breath were caused by the damage of *Yang Qi* for a long time, and the *Yang* could not spread water for usage but accumulated in the heart and lungs. The tongue was dull, the coating was white and greasy, and the pulse was stringy and slippery; thus, the syndrome was characterized as a *Yang* deficiency and flooding. Chinese medicine treatment: To address both symptoms and root causes, modified Zhenwu decoction (ZWD) was given to the patient. Prescription: *Radix Aconiti Lateralis Praeparata* 15 g, *Radix Paeoniae Alba* 15 g, *Rhizoma Zingiberis Recens* 10 g, *Rhizoma Atractylodis Macrocephalae* 15 g, *Poria* 20 g, *Semen Lepidii* 10 g, *Fructus Jujubae* 10 g, and *Bulbus Fritillariae Thunbergii* 10 g. These herbs were boiled in water for decoction usage, po, and qd. Prescription analysis: In the ZWD, *Radix Aconiti Lateralis Praeparata* was the monarch drug to warm the *Yang* of the kidney and spleen to promote the circulation of *Qi* and body fluid, *Rhizoma Atractylodis Macrocephalae* and *Poria* were minister drugs to tonify the spleen to promote urination and draining, *Rhizoma Zingiberis Recens* was the assistant drug to warm the *Yang* for dispelling cold and dampness, *Radix Paeoniae Alba* was the guide drug to promote urination and nourish the *Yin*. This traditional Chinese decoction treated both the symptoms and causes as the principle, and took the method of benefiting the *Qi* and *Yang* in order to evacuate the water and dredge the collaterals.

Reexamination after a week: LDH: 668.0 U/L, CK: 46.2 U/L, BNP: 1,246.0 pg/ml, cTnT: 193.8 pg/ml, INR: 1.2, D-dimer: 0.7 mg/L, UCG: EF 34%, a change of dilated cardiomyopathy was considered, general decline and desynchrony in the left ventricular

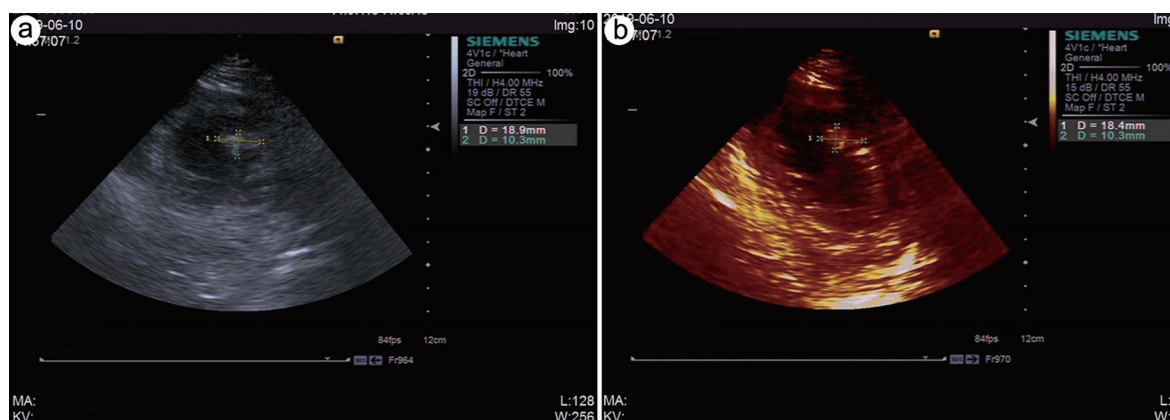


Fig. 2. Rechecked UCG of case 1. UCG, ultrasonic cardiogram.

wall activity, reduction of the left ventricular systolic and diastolic function, thrombus in the left ventricular apical ( $18 \times 10$  mm), aortic, mitral and tricuspid regurgitation (mild), and pericardial effusion (small amount) (Fig. 2). The patient's condition improved after treatment, and he was discharged on June 12, 2019. The patient's spirit had improved with no obvious shortness of breath at rest, no chest pain and radiating pain, and no edema in both lower extremities. The tongue was dull, the coating was white and greasy, and the pulse was stringy and slippery. Physical examination: The breath sounds of both lungs were better, the heart rhythm was uniform, and no pathological murmur was heard in the auscultation area of each valve. The diagnosis and treatment from June 12, 2019 to July 9, 2019 were similar to that in hospital, except the enoxaparin, and decreased dosage of furosemide (10 mg, qd, po) and spironolactone (10 mg, qd, po). Rechecked UCG on July 9 showed an EF of 37% with no abnormal mass in the left ventricle.

### Case 2

A male, 37 years old, was admitted to Guangdong Provincial Hospital of Chinese Medicine-Zhuhai Hospital on February 10, 2018, because of chest distress and shortness of breath for two weeks, and edema of both lower limbs for one week. Present medical history: Shortness of breath and chest tightness that was aggravated after activities, unable to be supine, severe edema from the lower extremities to the thighs, cough, and sputum with blood. The past medical history showed heart failure and hypertension without any family history. Physical examinations showed weak respiratory sounds and

moist rales of both lungs, extended cardiac dullness, diffuse apex beats, and weak heart sounds. The tongue was pale and dark with a white coating, and the pulse was rapid. Laboratory examinations: WBC  $13.37 \times 10^9/L$ , NEUT% of 81.1%, GLU of 7.6 mmol/L, D-dimer of 2.18 mg/L, PT of 13 s, INR of 1.16, FIB of 6.13 g/L, PCT of 1.5 ng/ml, BNP of 13,988 pg/ml, cTnT of 73.12 pg/ml, and LDH of 835 U/L. ECG: Nodal tachycardia, T wave changes, and left atrial enlargement. UCG: EF of 24%, whole heart enlargement, combined bi-side cardiac dysfunction, left ventricular mural thrombus ( $43 \times 22$  mm), ultrasound changes of hypertension and coronary heart disease, mitral regurgitation (mild), tricuspid regurgitation (moderate), pulmonary hypertension (mild), and pericardial effusion (small-medium amount) (Fig. 3).

Western diagnosis: 1. Heart failure, 2. Grade 3 hypertension (very high-risk group), 3. hypertensive heart disease, 4. left ventricular thrombus, 5. alcoholic cardiomyopathy, 6. suspicious coronary heart disease, 7. tricuspid regurgitation (moderate), and 8. pericardial effusion (non-inflammatory) (small-medium amount). Western medicine treatment: Aspirin (100 mg, qd, po) and clopidogrel (75 mg, qd, po) for antiplatelet aggregation, rivaroxaban (15 mg, qd, po) for anticoagulation, cedilan (0.2 mg, prn, iv) and digoxin (0.125 mg, qd, po) for a cardiotonic effect, sodium nitroprusside (50 mg + 5% GS 250 ml, prn, ivd) to expand the coronary arteries and reduce the blood pressure, metoprolol (12.5 mg, bid, po) to control the heart rate, furosemide (40 mg, qd, po) and spironolactone (20 mg, bid, po) for diuresis and preventing myocardial fibrosis, benazepril (5 mg, qd, po) to reduce blood pressure

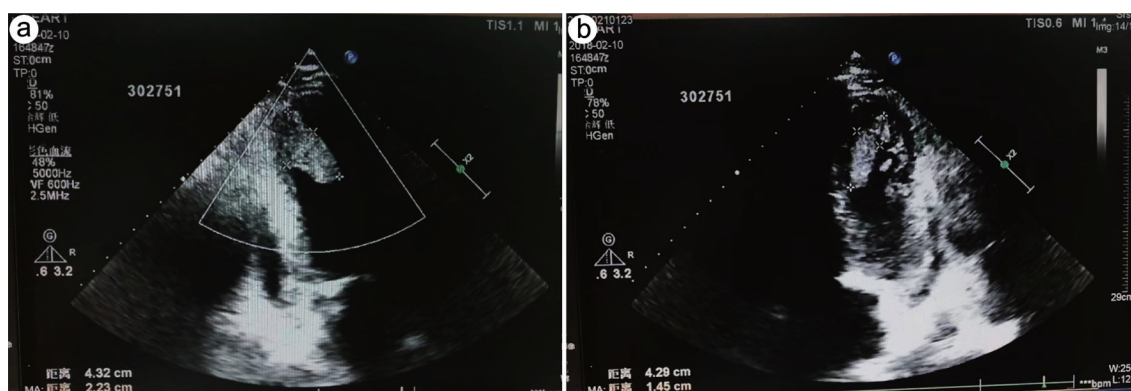


Fig. 3. Admission UCG of case 2. UCG, ultrasonic cardiogram.



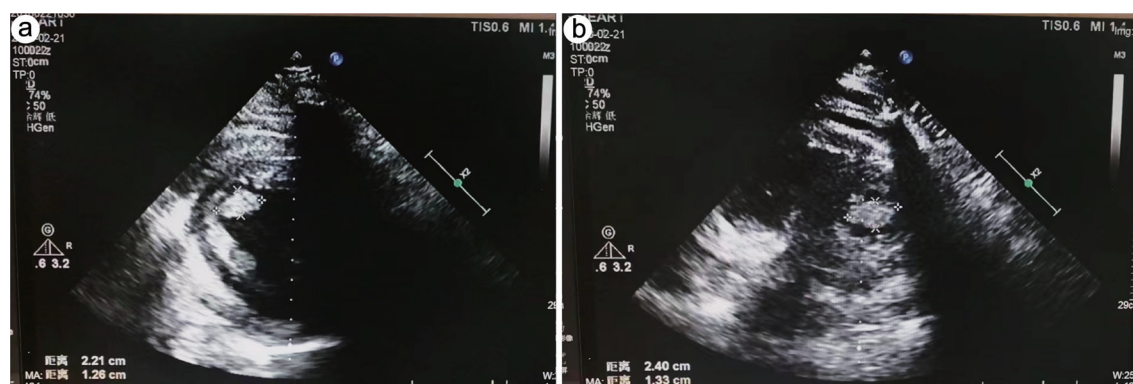


Fig. 4. Rechecked UCG of case 2. UCG, ultrasonic cardiogram.

and prevent myocardial remodeling, and rosuvastatin calcium (20 mg, qd, po) tablets to reduce the lipid level and plaque.

Chinese medical diagnosis: Heart failure (syndrome of the *Yang* deficiency and water stasis, and blood stasis blocking collaterals). Etiology and pathogenesis: The patient's chest tightness and shortness of breath were caused by the damage of the *Yang Qi* for a long time, and the *Yang* could not spread water for usage but accumulated it in the heart and lungs with blood stasis. The tongue was pale and dark with a white coating, and the pulse was rapid; thus, the syndrome was characterized as a *Yang* deficiency and water stasis with blood stasis blocking the collaterals. Chinese medicine treatment: To address both symptoms and root causes, modified Wuling decoction was given to the patient. Prescription: *Pericarpium Trichosanthis* 10 g, *Semen Lepidii* 10 g, *Ramulus Cinnamomi* 15 g, *Poria* 20 g, *Polyporus* 20 g, *Rhizoma Atractylodis Macrocephalae* 30 g, and *Radix Pseudostellariae* 15 g. These herbs were boiled in water for decoction usage, po, and qd. Prescription analysis: In the Wuling powder, *Rhizoma Alismatis* was the monarch drug for clearing dampness and promoting diuresis, *Poria*, *Polyporus*, and *Rhizoma Atractylodis Macrocephalae* were the minister drugs to strengthen the effect of the monarch drug, *Ramulus Cinnamomi* was the guide drug to warm the *Yang* to promote the circulation of *Qi* and body fluid, and dispelling external pathogens. This traditional Chinese decoction treated both the symptoms and causes as the principle, consequently benefiting the *Qi* and *Yang* in order to activate the blood and invigorate the water.

Recheck of the UCG on February 13: EF of 41%, changes in hypertension, left ventricular dysfunction, left ventricular mural thrombus (41 × 18 mm), mitral and tricuspid regurgitation (mild), and pericardial effusion (small amount). Ambulatory blood pressure: 1. The 24-h ambulatory blood pressure curve showed a non-spoon shape distribution and 2. blood pressure increased in the morning. Dynamic ECG: Sinus rhythm, ST-T changes, and a 24-h heart rate variability analysis showed a decrease. The patient was discharged after the symptoms improved. The patient's spirit was good, the shortness of breath and chest tightness were relieved, there was no cough and phlegm, no chest pain, slept well, and had normal bowel movements. Physical examination: The breath sounds in both lungs were slightly thicker, and no wet rales were heard. The cardiac dullness circle expanded to the left, the rhythm was uniform, and no pathological murmur was heard in the auscultation area of each valve. Diagnosis and treatment from June 13, 2018 to July 21, 2018 were similar to that in hospital, except the cedilan and sodium nitroprusside, and decreased dosage of furosemide (10 mg, qd, po) and spironolactone (10 mg, qd, po).

Reexamination of the UCG on February 21: EF of 49%, cardiac changes of hypertension and alcoholic cardiomyopathy, left ventricular dysfunction (mild), left ventricular mural thrombus (22 × 12 mm), and mitral regurgitation (mild) (Fig. 4).

## Discussion

Left ventricular thrombus (LVT) is a common complication in patients with severe heart failure (lower left ventricular ejection fraction), left ventricular regional dyskinesia (apical contractility extremely weakened or disappeared), and ventricular aneurysm.<sup>4</sup> Virchow's classic theory of thrombotic mechanism (slow blood flow, vascular endothelial injury, and hypercoagulable state) is applicable to the formation of LVT. The mechanism of the formation of LVT is mainly related to the disappearance or ataxia of the left ventricular regional wall contractility, which eventually leads to a slower blood flow or even stagnation.<sup>5</sup> In addition, patients with myocardial infarction have endocardial damage due to prolonged ischemia, and often have a hypercoagulable state of blood and various inflammatory reactions, all of which can cause ventricular thrombus. LVT is an independent risk factor for systemic embolism.<sup>6</sup> Once the thrombus falls off, it may cause systemic embolism. The condition is critical, and the prognosis is often poor. At present, there is no specific treatment for ventricular thrombus. Thrombolytic therapy, heparin therapy, aspirin for anti-aggregation, warfarin for anticoagulation, and new oral anticoagulant (NOAC) are generally used. The European Society of Cardiology (ESC) guidelines (2015) recommended AMI with LVT to receive warfarin anticoagulant therapy for 3–6 months.<sup>7</sup> However, warfarin has limitations, including a narrow therapeutic time window, high influence of food and other drugs, and frequent monitoring of the coagulation parameters.<sup>8</sup> The curative effect of anticoagulant drugs for LVT is not clear, and there is a risk of bleeding. Although Calkins *et al.* had proved that NOAC had a lower risk of bleeding than warfarin,<sup>9</sup> it still needs further research to provide evidence. The two cases provided in this study were treated with a Western medical treatment plan, which followed the current guideline treatment plan for heart failure and was combined with traditional Chinese medicine following syndrome characterization, and the rechecking of the UCG result was satisfactory and the symptoms were improved.

From the perspective of Chinese medicine, the primary diseases of ventricular thrombus will damage the heart, so there is insufficient *Yang* in the heart to promote the circulation of the blood, which finally leads to blood stasis. Simultaneously, the static blood

further causes the dysfunction of the body fluid distribution. Moreover, the aforesaid factors are associated with symptoms, such as chest tightness, asthma, and edema. The two patients in this report belonged to the pattern of edema due to a *Yang* deficiency and stasis blocking channels. Hence, the therapy was based on the principle of warming the *Yang* for diuresis, promoting blood circulation, and removing blood stasis, whose representatives were the ZWD and Wuling decoction. The prescriptions are both from the Synopsis of Golden Chamber written by Zhang Zhongjing, one of the greatest Chinese physicians in history.

Studies had also shown that Yinchen Wuling powder had the functions of regulating the blood lipid levels and anticoagulation in hyperlipidemic rats, which could prolong the prothrombin time (PT) and activate partial thromboplastin time (APTT), reduce fibrinogen (FIB), decrease the level of total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C), increase the level of high-density lipoprotein (HDL-C), reduce whole blood viscosity (high, medium, and low cut), plasma viscosity, hematocrit, and the platelet adhesion rate.<sup>10</sup> Its anti-atherosclerosis effect might be related to the downregulation of the expression of gene bcl-2 mRNA.<sup>11</sup> As a consequence, Wuling powder could significantly improve the cardiac function in patients with chronic heart failure by reducing the BNP, endothelin (ET), and improving the left ventricular ejection fraction (LVEF).<sup>12,13</sup> The ZWD could effectively improve the cardiac function in patients with chronic heart failure, including improving the LVEF, left ventricular end diastolic and systolic diameter, left ventricular shortening, heart output, and stroke volume index.<sup>14,15</sup> In addition, modified ZWD could improve the heart function of heart failure rats, which could be related to the reduction of the angiotensin, aldosterone, and antidiuretic hormone levels, and promote the excretion of Na<sup>+</sup> and K<sup>+</sup> to increase urine output.<sup>16</sup> ZWD's mechanism of warming *Yang* to promote diuresis was associated with the hypothalamic-pituitary-adrenal axis, hypothalamic-pituitary-thyroid axis, reduction of the content of nitric oxide and ET, regulation of apoptosis-related genes, inhibition of apoptosis, balance of the quantity and quality of aquaporin one and two, and regulation of the osmotic pressure.<sup>17</sup>

There are many causes and critical complications of LVT. Although anticoagulant therapy is useful for ventricular thrombus, its therapy effect is not clear and there are side effects. On the other hand, a large number of studies and clinical observations have proved the effectiveness and safety of Chinese medicine for cardiovascular disease. Therefore, in taking advantage of traditional Chinese medicine, we can treat this kind of patients based on the principle of warming *Yang* for diuresis, promoting blood circulation, and removing blood stasis. In this report, after treatment, the patients' clinical symptoms were significantly improved, and the original thrombus was reduced or even eliminated. As such, the treatment of integrated traditional Chinese and Western medicine achieved good clinical results, improved the prognosis, and the quality of the patients' lives. However, the major limitation of this report was the lack of compared patients that were only treated by Western medicine without the combination of Chinese medicine. Thus, we would design such clinical trials, *e.g.*, an RCT to test this hypothesis in the future.

### Clinical perspectives

As LVT is a common complication of coronary heart disease, there is little relevant literature about the treatment of LVT by Chinese medicine. This report may provide a sample in the clinical treat-

ment of LVT by the combination of traditional Chinese medicine and Western medicine. Further studies comparing the efficacy of Chinese medicine and Western medicine could also be designed to elucidate the underlying reasons.

### Conclusions

The two left ventricular thrombus cases we reported here would support the efficacy of traditional Chinese medicine in treating LVT combined with common Western medicine treatment.

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### Conflict of interest

No competing financial interests exist.

### Author contributions

The study was designed by QL. The manuscript writing was performed by YL, CL, PL, and QL. The manuscript was revised by QC and RY, and finalized by QL. All authors read and revised the manuscript critically.

### Ethical statement

The patients consented to anonymous publication of their data, which was gratefully acknowledged by the authors. The ethical approval was granted by the Ethics Committee of GDHCM.

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