

Multi-organ embolism caused by oscillating aortic valve vegetation

A case report

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Abstract

Introduction: Valvular vegetation is often due to rheumatic heart disease and infective endocarditis. However, multi-arterial embolism can happen in older patients with no history of infection, fever, and cardiac symptoms. We describe a case of multi-organ embolism caused by oscillating aortic valve vegetation.

Patient concerns: An 80-year-old woman without a history of infection, fever, and heart symptoms showed sudden loss of consciousness and symptoms of a multi-vessel embolism. Magnetic resonance imaging revealed multiple patchy ischemic foci in both cerebral hemispheres in the same time-phase, and echocardiography showed regurgitation in the aortic valve due to an abnormally hypo-hyperechoic mass measuring about 7.7×17.2 mm and oscillating aortic valve vegetation, which was induced by cardiac contraction.

Diagnosis: Multiple organ cardiac embolisms caused by oscillating aortic valve vegetation.

Interventions: Anti-platelet, fluid-supplement, and vascular-dilating therapies as well as intravenous diazepam were given to the patient.

Outcome: The patient died of epileptic attack secondary to the cerebral embolism.

Conclusions: The patient's whole-body multi-vessel ischemic events in nearly the same time-phase should have encouraged us to consider the possibility of cardiogenic embolism and thus early examination and treatment, although she was old with a relatively poor response due to early infection and physical discomfort. Clinicians should be aware that aortic valve vegetation induces generalized multi-organ embolism in the setting of infective endocarditis in order to ensure prompt recognition and treatment of this fatal complication.

Abbreviations: CK = creatine kinase, IE = infective endocarditis, MRI = magnetic resonance imaging.

Keywords: echocardiogram., embolism, valve vegetation

1. Introduction

Infective endocarditis (IE) is associated with high hospital mortality.^[1] It is characterized by the deposition of platelets, pathogens, and fibrin thrombi on valvular structures, which are prone to embolism and most commonly involved in cerebrovas-

cular circulation. The most common symptom of IE is fever and cardiac symptoms.^[1] Valvular vegetation is related to blood flow turbulence, inflammatory reactions, and injuries of the endothelial valve.^[1] Aortic valve vegetation can also occur without endocarditis.^[2] In this report, we describe the case of an older patient with multi-arterial embolism who had no history of infection, fever, and cardiac symptoms.

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2. Case report

The patient was an 80-year-old woman who was admitted because of sudden onset of loss of consciousness. She was found lying on the floor with no response to calling of her name, and her blood pressure was 112/81 mmHg. One hour later, she showed some response, but was still in a fretful and delirious state with mixed aphasia. The neurologic examination showed that her left upper extremity muscular strength was grade III/V, right upper extremity muscular strength was grade 0/V, bilateral lower extremity muscular strength was grade III/V, and left Babinski sign was positive. She had a history of hypertension for 30 years and type 2 diabetes mellitus for 20 years.

She was admitted 4 hours later. Gross hematuria was observed, and her muscle enzyme and liver enzyme levels were greatly elevated. The respective laboratory values at 2.5 hours, 10 hours, and 34 hours were as follows: creatine kinase (CK), 43 IU/L, 144

Table 1

Serum Enzyme Test.

Onset hours	CK (IU/L)	CK-MB (IU/L)	LDH (IU/L)	HBDH(mmol/L)	AST (IU/L)	ALT (IU/L)	TNT(ng/mL)	D-Dimer(ug/L)
2.5 h	43	6	227	148	36	No check	0.2	No check
10 h	144	52	2108	732	1302	858	0.52	1601
34 h	185	8	3029	1028	3587	2295	0.35	No check
49 h	195	9.8	1826	640	1670	No check	0.31	3538
3 d	185	7	917	467	1196	1667	No check	3684
4 d	96	5	545	335	580	1268	0.41	3377
5 d	28	6.6	358	249	194	No check	0.41	2304
7 d	30	2	271	219	90	479	0.19	1174
normal reference ranges	30-170	0-20	100-300	90-250	8-40	5-40	0-150	0-400

AST = aspartate aminotransferase, ALT = alanine aminotransferase, CK = creatine kinase, CK-MB = creatine kinase isoenzyme, HBDH = a-Hydroxybutyric acid dehydrogenase, LDH = lactate dehydrogenase, high density lipoprotein, TNT = Troponin T.

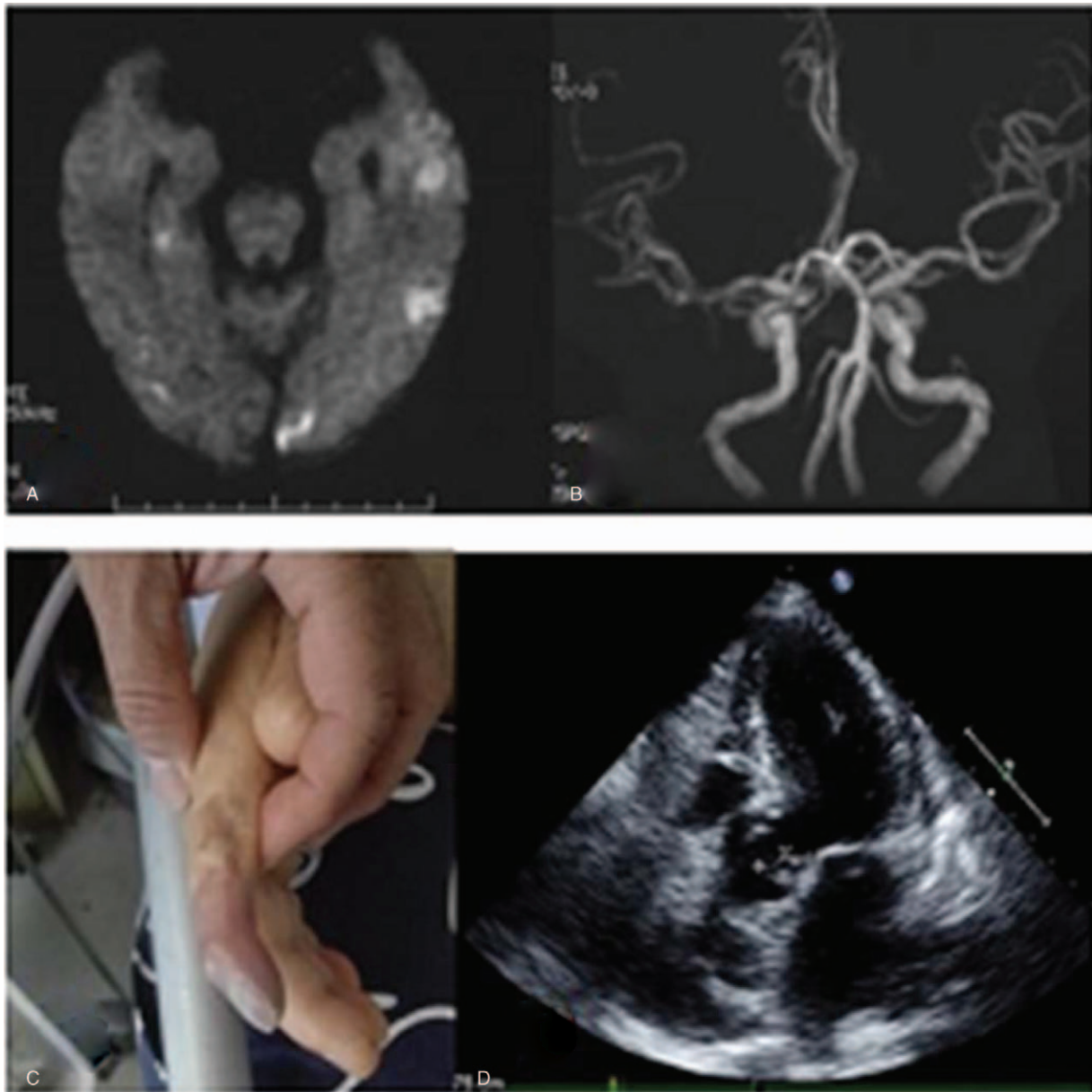


Figure 1. Imaging and physical findings. A: Diffusion-weighted imaging sequence showing multiple patchy abnormal signals in both cerebral hemispheres. B: Magnetic resonance angiography revealing that the middle cerebral artery, anterior cerebral artery, and posterior median artery in both cerebral hemispheres are normal. C: The tip of the patient's index finger of her right hand is murky grey with a low skin temperature and black nails. D: Echocardiogram shows that the aortic valve has large vegetation measuring about 7.7 × 17.2mm.

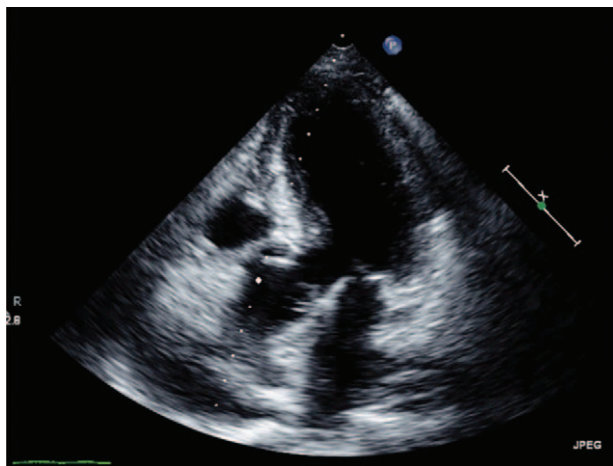


Figure 2. Echocardiogram showing valve vegetation oscillating along with the valve.

IU/L, and 185 IU/L; lactate dehydrogenase, 227 IU/L, 2108 IU/L, and 3029 IU/L; aspartate aminotransferase, 36 IU/L, 1302 IU/L, and 3587 IU/L; and alanine aminotransferase 480 IU/L, 858 IU/L, and 2295 IU/L. Her D-dimer levels at 10 hours, 34 hours, and 49 hours after onset were 1601 $\mu\text{g/L}$, 3538 $\mu\text{g/L}$, and 3684 $\mu\text{g/L}$ (Table 1). Head magnetic resonance imaging (MRI) indicated multiple patchy high signals in T2-weighted imaging and fluid-attenuated inversion recovery imaging scans of both cerebellar hemispheres, the frontal, temporal, and occipital lobe subcortical regions. T1-weighted imaging showed relatively low signals. Multiple patchy high signals (Fig. 1A) were seen in the aforementioned lesion regions in diffusion-weighted images. Cranial magnetic resonance angiography indicated generally normal intracranial vascular distribution and no obvious stenosis (Fig. 1B).

Three days after admission, her right index finger was murky and grey with a low skin temperature, and the nails turned black (Fig. 1C). Despite no history of atrial fibrillation, cardioembolism was highly suspected. Considering her age, anti-platelet, fluid-supplement, and vascular-dilating therapies were given, and her conscious returned to normal, but complete aphasia remained. Five days after admission, echocardiography indicated that (Fig. 2), the aortic valve was thickened with an intensive echo. In addition, an abnormally hypo-hyperechoic mass was explored on the left ventricular surface of the left coronary valve of the aortic valve; it moved with the valve, measured about 7.7×17.2 mm, induced closure insufficiency of the aortic valve, and was present with a moderate amount of regurgitation in the aortic valve (Fig. 1D), which indicated aortic valve vegetation exfoliation and multiple cerebral embolisms with hepatic, renal, and finger arterial embolisms.

Ten days after admission, she suddenly developed generalized tonic-clonic seizure. The epileptic attack was considered secondary to cerebral embolism. Intravenous diazepam (10 mg) was given, and the seizure stopped; however, she remained in a coma. On the next day, she developed a continuous fever and a large amount of rales and rhonchi in both lungs with hypoxemia. On the third day, she was transferred to the intensive care unit, and on the fifth day, she died.

3. Discussion

Valvular vegetation is secondary to congenital heart disease in most cases. The patients with valvular vegetation suffer from rheumatic heart disease and IE, which are the primary underlying heart lesions, and when local ulcer surfaces are formed, vegetation (mural thrombus) appear, and the vegetation are mostly lymphocytes with a small amount of polymorphonuclear leukocytes.^[3] The present patient had no history of infection, fever, and cardiac symptoms before the onset. We considered that because of her age, she may have had a relatively poor response to the early infection, and her daily activities were limited, with relatively small requirements on physical strength and heart ejection fraction.

The embolism induced by vegetation exfoliation is a clinically common complication, which may cause arterial embolism in important organs. Cardiac cerebral arterial embolism is the most common complication that usually causes multiple bilateral lesions, followed by splenic, renal, and mesenteric embolisms. The common manifestations are stereotype of attacks and multiple embolisms, and the clinical features quickly peak after onset.^[4] Large moveable valvular lesions pose a greater risk of stroke and arterial embolization.^[5] The patients are apt to suffer from syncope and epileptic seizure, but in most of cases, there is no aura of transient cerebral ischemic attack, with a high mortality.^[6] This patient suddenly lost consciousness as the initial symptom, and subsequently, she developed hemiplegia, gross hematuria, an obvious increase in CK and liver enzyme levels, indicating clinically gradual deterioration. After onset, there was a progressive increase in liver enzyme and CK levels, indicating that both muscles and the liver system were damaged. The patient's D-dimer level was also gradually and dramatically elevated, which reflects that the in-vivo blood is in the hypercoagulable and hyperfibrinolytic state. Patients with cerebral infarction will experience an increase in the D-dimer level, but a level higher than 1000 $\mu\text{g/L}$ has not been reported.^[7,8] Our patient's D-Dimer level reached to 3684 $\mu\text{g/L}$ at 46 hours, indicating multiple organ embolisms.

In the present patient, head MRI indicated multiple fresh infarctions in both cerebral hemispheres, and heart ultrasonography showed that the aortic valve had large oscillating vegetation, which led to aortic valve closure insufficiency and a moderate amount of regurgitation. Echocardiography is the main diagnostic modality for evaluating, diagnosing, and managing systemic and pulmonary embolisms as well as stroke.^[9] Generalized multi-organ embolism induced by transient sudden break-up and exfoliation of the aortic valve vegetation was considered, and it caused the patient's loss of consciousness, hemiplegia, and progressively increasing CK and liver enzyme levels. However, 3 days after onset, the liver enzyme, CK, and D-Dimer levels significantly decreased, indicating that the cardiac disease included embolic stroke, syncope, and intracerebral bleeding. Because of the patient's old age and development of embolic stroke after admission, we did not administer an anticoagulant to prevent bleeding. Anticoagulation therapy has been used for native aortic valve thrombosis in primary antiphospholipid syndrome, and echocardiography showed complete resolution of the aortic valve vegetation after a few months; the patient improved clinically and had no lingering symptoms.^[10] It has been suggested that vegetations in antiphospholipid syndrome can be treated successfully with anticoagulation treatment, regardless of their size.^[10] The

epidemiological practices indicated that in recent years, the incidence of IE has increased, especially in elderly individuals and hospitalized patients.^[11,12] In our case, we think that after the patient was suspected to have multi-organ cardiac embolism, early and integrally repeated echocardiography should have been performed as well as risk factor screening; then after weighing the advantage and disadvantage, anticoagulation therapy should have been considered earlier. Clinicians should be aware that aortic valve vegetation induces generalized multi-organ embolism in the setting of IE in order to ensure prompt recognition and treatment of this fatal complication.

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Supervision: Ainan Xu, Guang Huang.

Validation: Rong Xiong.

Visualization: Rong Xiong.

Writing – original draft: Ainan Xu.

Writing – review & editing: Ainan Xu.

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