

Repeated Transient Wallenberg's Syndrome: Probable Association with Ipsilateral Vertebral Artery Hypoplasia and Aortic Valve Disease

Feng-Di Liu, MD,* Rong Zhao, MD,* Shuo Wang, MD,* Wen-Ting Li, MD,*
Xiao-Xiao Tao, MD,* Ting Sun, MD,† Xiao-Lei Shen, MD,* and Jian-Ren Liu, MD, PhD*

Background: Here we report a rare case of repeated transient Wallenberg's syndrome and discuss its mechanism. **Methods:** Case report and literature review. **Results:** A 57-year-old man was admitted for 1.5-month repeated transient Wallenberg's syndrome, including right-sided Horner's syndrome, lower limb weakness, and paresthesia on the right side of the body and face. His symptom appeared mostly during physical activity. Symptoms occurred nearly everyday and lasted from 5 minutes to 30 minutes. His cranial magnetic resonance imaging (MRI) including diffusion-weighted MRI imaging was normal, and his cervical contrast-enhanced magnetic resonance angiography reflected right vertebral artery hypoplasia. Twenty-four-hour electrocardiogram and electroencephalography showed no abnormalities. Echocardiography showed aortic valve calcification with moderate aortic stenosis, moderate aortic insufficiency, and dilated aorta. Dual-antiplatelets or warfarin (international normalized ratio reached 2.07) were not effective to reduce the attacks. **Conclusions:** Hemodynamic instability due to valve disease combined with right vertebral artery hypoplasia could lead to transient Wallenberg's syndrome. Antithrombotics are often ineffective for this kind of patients and the best therapy for them could be to cure their valve disease. Repeated transient Wallenberg's syndrome is rare and that caused by ipsilateral vertebral artery hypoplasia and severe valve disease has not been reported up till now to our knowledge, so it will widen the knowledge on etiologies of transient ischemic attacks and provide information and reference to cardiologists and neurologists in diagnosis and treatment for patients with similar clinical manifestations. **Key Words:** Aortic valve stenosis and insufficiency—Horner's syndrome—transient ischemic attack—vertebral artery hypoplasia—Wallenberg's syndrome.
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From the *Department of Neurology, Jiuyuan Municipal Stroke Center of Shanghai, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai; and †Department of Cardiology, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China PR.

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F.-D.L. and R.Z. contributed equally.

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Address correspondence to Jian-Ren Liu, MD, PhD, Department of Neurology, Jiuyuan Municipal Stroke Center of Shanghai, Shanghai Ninth People's Hospital, Shanghai Jiao Tong University School of Medicine, 639 Zhizaoju Road, Huangpu District, Shanghai 200011, China PR. E-mail: liujr021_201410@126.com.

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Introduction

Here we reported a rare case with repeated transient Wallenberg's syndrome. Ipsilateral vertebral artery hypoplasia and aortic valve disease were considered to be the cause.

Case Report

A 57-year-old man was admitted for 1.5-month repeated transient Wallenberg's syndrome, including right-sided Horner's syndrome (Fig 1, A,B), lower limb weakness, and paresthesia on the right side of the body and face. His symptom appeared mostly during physical activity. Symptoms occurred nearly everyday and lasted from 5 minutes to 30 minutes. His cranial magnetic resonance imaging including diffusion-weighted magnetic resonance imaging (DWI) was normal, and his cervical contrast-enhanced magnetic resonance angiography reflected right vertebral artery hypoplasia (Fig 1, C). Twenty-four-hour electrocardiogram and electroencephalography showed no abnormalities. Echocardiography showed aortic valve calcification with moderate aortic stenosis, moderate aortic insufficiency, and dilated aorta (Fig 1, E-H). Clopidogrel (Plavix, 75 mg once every day

(q.d.) take orally (p.o.), aspirin (100 mg q.d. p.o.), and atorvastatin (Lipitor, 20 mg once every night (q.n.) p.o.) were given, but his symptoms did not improve. During his hospitalization, he underwent an acute ischemic stroke on his left parietal lobe that was confirmed by his second cranial DWI (Fig 1, D) and persistent right facial palsy was left. Warfarin (4.5 mg q.d. p.o.) was given then and international normalized ratio reached 2.07; however, repeated transient Wallenberg's syndrome continued with similar frequency. We considered that his valve disease combined with ipsilateral vertebral artery hypoplasia led to hemodynamic insufficiency and paroxysmal ischemia of posterior inferior cerebellar artery area, so we suggested him to receive aortic valve replacement surgery but he refused.

Discussion

Aortic valve stenosis and insufficiency can lead to cardiogenic cerebral embolism^{1,2} and was supposed to be the reason for the patient's acute left parietal stroke. Very early diastolic insufficiency was found because E (54; E peak, which represents peak flow velocity of left ventricular in early diastolic period) < A

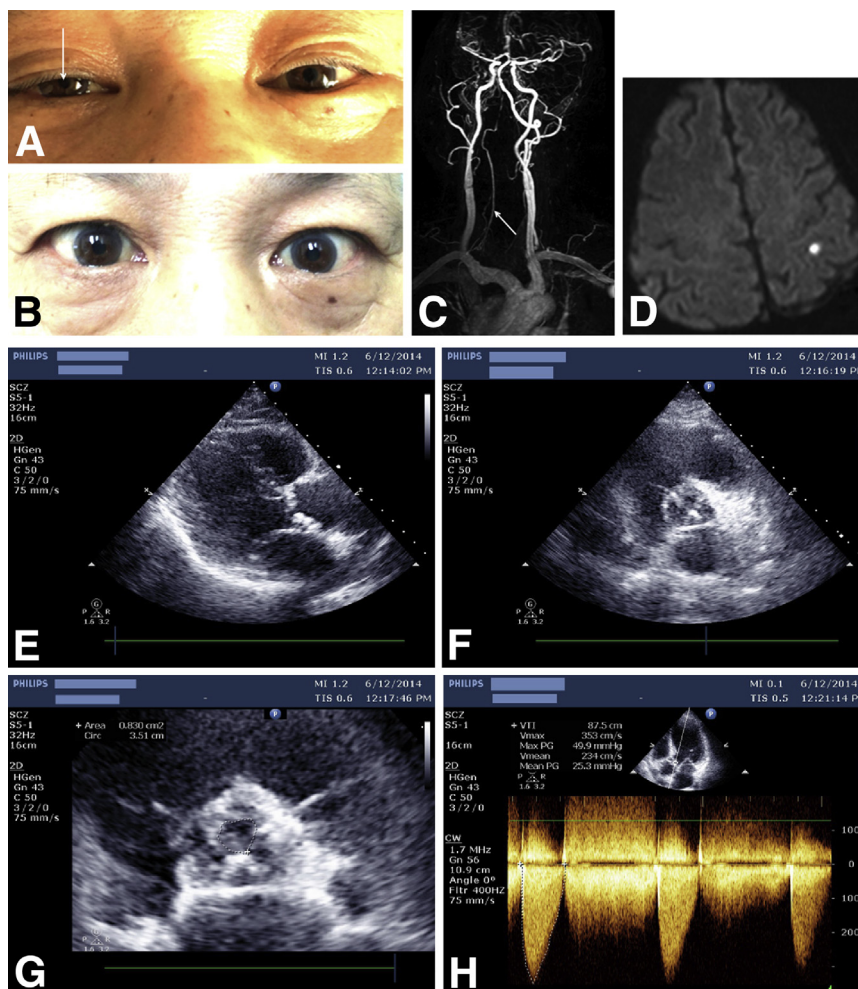


Figure 1. Patient's manifestation, neuroimaging, and echocardiography. (A) Right-sided Horner's syndrome (miosis and partial ptosis) during a transient ischemic attack. The arrow in panel A shows miosis of right pupil; (B) the Horner's sign disappears after the transient ischemic attack; (C) cervical contrast-enhanced magnetic resonance (MR) angiography reflects right vertebral artery hypoplasia. The arrow in panel C shows right vertebral artery hypoplasia; (D) diffusion-weighted MR imaging shows an acute ischemic stroke lesion on the left parietal lobe; (E, F) aortic valve calcification, moderate aortic stenosis and insufficiency, and dilated aorta; (G) aortic valve orifice area is $.83 \text{ cm}^2$; and (H) peak across valve pressure is 49.9 mm Hg .

(78; A peak, which represents peak flow velocity of left ventricular in late diastolic period), and Ea (8.4; Ea represents mitral annulus velocity in early diastolic period) < Aa (11; Aa represents mitral annulus velocity in late diastolic period) according to his echocardiography report, so even if his ejection fraction (EF) (65%) was normal, his effective cardiac output was low. Furthermore, his hypoplastic right vertebral artery aggravated transient ischemia of posterior inferior cerebellar artery area, and could give symptoms of Wallenberg's syndrome.³ Symptoms that occurred only in activities supported our speculation. In addition, DWI had excluded permanent stroke of the posterior circulation, and his electroencephalography did not show epileptiform activity. Transient Horner's sign and ataxia seldom happened in epilepsy patients. Therefore, we considered

that the transient Wallenberg's syndrome in this patient was caused by the combined effects of aortic valve disease and ipsilateral vertebral hypoplasia.

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