Case Studies

Three-Case Report of Embolic Stroke of Undetermined Source

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Background: Embolic stroke of undetermined source (ESUS) is a new clinical construct. It signifies that the embolus in the thromboembolic ischemic stroke is of unknown origin. The anticoagulants are usually prescribed for antithrombotic prophylaxis, but whether it is appropriate for all patients with ESUS is still unknown. *Methods and Results:* In this article, we describe 3 cases of ESUS, all of whose antithrombotic therapy was antiplatelet medication, and the 3 patients had no recurrence on 3- to 7-month follow-up. *Conclusions:* Because there was no obvious risk factor found in these ESUS cases, the recurrence risk is difficult to evaluate, and the optimum means of secondary prevention are still unknown. Hence, many aspects warrant resolution. **Key Words:** Cryptogenic stroke—embolic stroke—embolic stroke of undetermined source (ESUS)—antiplatelet therapy—antiplatelet and anticoagulation treatment for secondary stroke prevention.

Introduction

Cryptogenic ischemic strokes are symptomatic cerebral infarcts for which no probable cause is identified after adequate diagnostic evaluation.[1] There is accumulating evidence that most strokes of undetermined origin are thromboembolic.[2,3] The optimal antithrombotic prophylaxis, as secondary prevention for these patients, is still unknown. Recently, a new clinical construct, termed embolic stroke of undetermined source (ESUS), was introduced by the Cryptogenic Stroke/ESUS International Working Group as a potential therapeutically relevant entity, with an indication for anticoagulation.[4] In this article, we reported 3 cases of ESUS, which are considered highly cryptogenic, for the embolus is unknown after standard evaluation and special evaluation. We also discussed the secondary prevention for the 3 cases.

Case 1

One night, around midnight, a 45-year-old man got up to use the washroom after 4 hours of sleep. Suddenly he felt dizzy and nauseous, and he could not move his left limbs. These symptoms lasted approximately half an hour and were followed by persistent ataxia, unstable gait, and slurred speech. He was admitted to our hospital (The First Affiliated Hospital of Wenzhou Medical University, Wenzhou, Zhejiang, China) 6 hours after onset. He did not have neck pain. He did not have a history of cigarette smoking or alcohol abuse or any other pathology. On his arrival, we observed horizontal nystagmus, bilateral ataxia (the right limbs were obvious), and Babinski's sign in the left leg. His National Institutes of Health Stroke Scale (NIHSS)[5] score was 2.

Magnetic resonance imaging (MRI) showed infarction at the right side of the pons and bilaterally at the cerebellum (Fig 1). Magnetic resonance angiography yielded normal findings for both the intracranial and extracranial arteries. No aortic arch plaques were found in the

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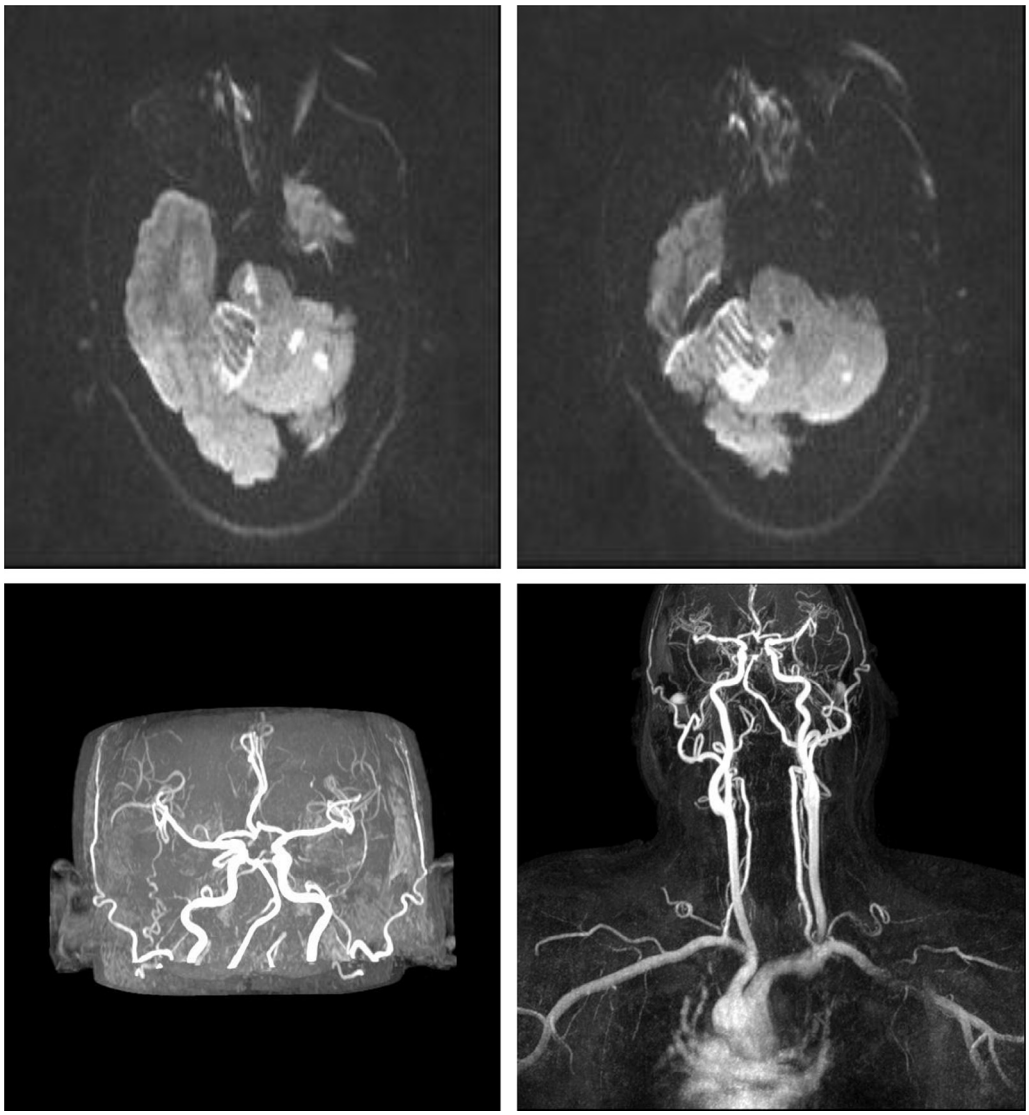

Figure 1. *The MRI and MRA of the first case. The DWI showed the right of pons and bilateral cerebellum infarct, with mild hemorrhagic infarct in the right cerebellum. The MRA showed normal findings in both intracranial and extracranial arteries. Abbreviations: DWI, diffusion-weighted image; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.*

brain computed tomography (CT) angiography. A 12-lead electrocardiogram (ECG) and Holter monitor results were normal. Cardiac telemetry during the first 3 days of hospitalization revealed no dysrhythmias. The complete blood count, prothrombin time, and activated partial thromboplastin time were normal. There were no indications for hypercoagulability (Table 1). The transthoracic echocardiogram (TTE) result was also normal. During the inpatient period, we observed that the patient was mildly hypertensive. According to the patient's history and his MRI results, we considered this to be a case of ESUS. We advised anticoagulation therapy and antiplatelet therapy for the patient; he chose antiplatelet therapy, in view of convenience and cost-effectiveness. The patient received

Table 1. *Some related laboratory data in serum of the 3 patients*

Case	Hemoglobin (g/L)	Platelet count (*109/L)	PT (s)	APTT (s)	Fibrin (g/L)	D-dimer (mg/L)	Tumor marker	Creatinine (μmol/L)
Case 1	162	241	15.0	33.9	2.06	1.02	Normal	73
Case 2	162	146	14.1	36.6	3.09	.78	Normal	81
Case 3	149	411	13.9	38.5	2.97	.90	Normal	71

Abbreviations: APTT, activated partial thromboplastin time; PT, prothrombin time.