Case Report

Cerebral Vasospasm and Delayed Cerebral Ischemia after Warfarin-Induced Subarachnoid Hemorrhage

Irapuá Ferreira Ricarte, MD,* Fabrício G. Calente, MD,* Maramélia M. Alves, MD,* Daniela L. Gomes, MD,* Raul A. Valiente, MD,* Flávio A. Carvalho, MD,*† and Gisele S. Silva, MD, PhD*†

Background: Subarachnoid hemorrhage (SAH) associated with anticoagulation is a rare event. About 30% of in-hospital patients with aneurysmal SAH develop delayed cerebral ischemia (DCI); however, the occurrence of vasospasm and DCI in patients with nonaneurysmal SAH is still controversial. *Methods:* This study is a case report of a patient experiencing an anticoagulation-induced SAH complicated by vasospasm and DCI. *Results:* A 38-year-old woman presented to our hospital with a sudden onset of severe headache. Head computed tomography (CT) showed bleeding in the posterior fossa subarachnoid space. There was no aneurysm in the CT angiography. The International Normalized Ratio at hospital admission was 9. Anticoagulation was held, and she was treated with fresh frozen plasma (15 mg/kg) and parenteral vitamin K. The patient remained in the intensive care unit and had daily transcranial Doppler (TCD) monitorization. TCD examination detected increased blood flow velocities in the basilar and vertebral arteries, meeting criteria for definitive vasospasm. CT angiography confirmed the presence of posterior circulation vasospasm. Magnetic resonance (MR) imaging 2 weeks after the bleeding showed a small area of restricted diffusion in the left superior cerebellar artery territory. MR angiography showed resolution of the vasospasm at this time point and TCD velocities normalized. *Conclusions:* In conclusion, this case report suggests that vasospasm and consequent DCI is a possible mechanism of secondary lesion after anticoagulation-induced SAH. To our knowledge, this is the first report of vasospasm and DCI due to warfarin-associated SAH. **Key Words:** Cerebral vasospasm—transcranial Doppler—delayed cerebral ischemia—subarachnoid hemorrhage.

Cerebral ischemia that occurs after subarachnoid hemorrhage (SAH) is called delayed cerebral ischemia (DCI). About 30% of in-hospital patients with aneurysmal SAH develop DCI as a complication within the first 2 weeks after the hemorrhage; however, the occurrence of vasospasm and DCI in patients with nonaneurysmal SAH is still controversial.[1] To our knowledge, this is the first report of vasospasm and DCI due to warfarin-induced SAH.

From the *Department of Neurology, Federal University of São Paulo, Hospital São Paulo, São Paulo; and †Neurology Program and Stroke Center, Hospital Israelita Albert Einstein, São Paulo, Brazil.

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The authors declare that they have no conflict of interest and have nothing to disclose.

Full consent was obtained from the patient for publication.

Address correspondence to Irapuá Ferreira Ricarte, MD, Universidade Federal de São Paulo, Departamento de Neurologia e Neurocirurgia, Rua Pedro de Toledo, 650, CEP 04039-000, São Paulo, SP, Brazil. E-mail: irapuaferreir@hotmail.com.

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Case Report

A 38-year-old woman presented to our hospital with a sudden onset of severe headache. Her medical history was remarkable for hypertension and a remote history of Chagas disease. She had experienced a left middle cerebral artery (MCA) ischemic stroke 3 months before (Fig 1, A) and a pulmonary embolism 1 month before. Her cardiac evaluation following the stroke including transthoracic and transesophageal echocardiogram was within the normal limits. A 24-hour Holter monitoring showed only rare atrial ectopic beats. She was using warfarin irregularly since the pulmonary embolism. On neurologic examination, the patient was somnolent and had right-sided hemiparesis (related to the previous left MCA stroke). Head computed tomography (CT) showed bleeding in the posterior fossa subarachnoid space (Fig 1, B). There was no aneurysm in the CT angiography. The International Normalized Ratio (INR) at hospital admission was 9, probably because the patient did not follow medical's recommendation to perform regular INR control. Anticoagulation was held, and she was treated with fresh frozen plasma (15 mg/kg) and parenteral vitamin K.

The patient remained in the intensive care unit and had daily transcranial Doppler (TCD) monitorization. On the fifth day after hospital admission, TCD examination detected increased blood flow velocities in the basilar and vertebral arteries, meeting criteria for definitive vasospasm. CT angiography confirmed the presence of posterior circulation vasospasm (Fig 1, E). Nimodipine was initiated. The mean arterial pressure was kept within the normal limits (systolic blood pressure around 150 mm Hg, without vasopressors), and the patient was normovolemic and had normal magnesium values. She had no signs or symptoms from the posterior circulation vasospasm. TCD velocities

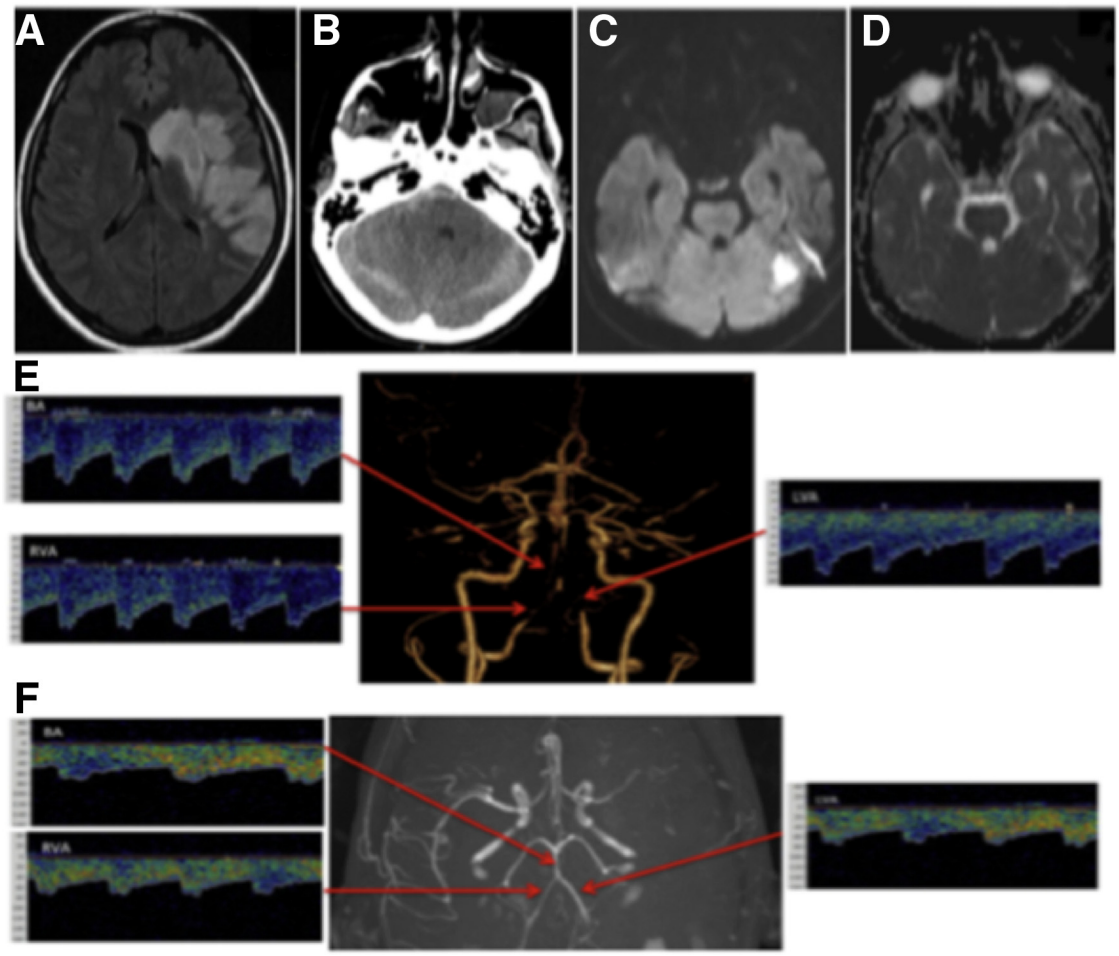

Figure 1. *(A) Brain MRI showing an ischemic stroke in the territory of the left middle cerebral artery. (B) Brain CT scan showing bleeding in the posterior fossa. (C) Diffusion-weighted brain MRI showing a small area of restricted diffusion in the left SCA territory. (D) Apparent diffusion coefficient map disclosing a hypointense lesion in the left SCA territory corroborating the presence of an acute ischemic stroke. (E) TCD sonography and CT angiography showing severe narrowing of bilateral vertebral arteries and basilar artery. (F) TCD velocities became normal after 2 weeks, and MR angiography did not show narrowing of these arteries. Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging; SCA, superior cerebellar artery; TCD, transcranial Doppler.*