Case Report

Stroke and Cardiac Papillary Fibroelastoma: Mechanical Thrombectomy after Thrombolytic Therapy

Ana F. Santos, MD,* João Pinho, MD,* Vítor Ramos, MD,† Joana Pardal, MD,‡ Jaime Rocha, MD,§ and Carla Ferreira, MD*

We describe a case of a 34-year-old man with a sudden development of right hemiparesis and aphasia because of infarction of the left middle cerebral artery that was submitted to intravenous (IV) recombinant tissue plasminogen activator and mechanical thrombectomy. Transesophageal echocardiogram showed a small mass on the *anterior leaflet* of the mitral valve. Cardiac surgery was performed, and histological examination of the removed material was consistent with cardiac papillary fibroelastoma (CPF). Experience in using IV thrombolysis for the treatment of embolic stroke because of CPF is limited. To the best of our knowledge, only 3 patients are reported in literature in whom acute ischemic stroke and associated CPF were treated with thrombolytic therapy. A discussion of the efficacy of IV thrombolysis and the possible superiority of mechanical thrombectomy is included. **Key Words:** Cardiac papillary fibroelastoma—stroke—thrombolytic therapy—mechanical thrombectomy.

Introduction

Cardiac papillary fibroelastoma (CPF) is a rare primary cardiac tumor, with a significant propensity to embolize.[1,2] We describe a patient in whom emboli originating from CPF caused cerebral infarction; he was submitted to intravenous (IV) recombinant tissue plasminogen activator and mechanical thrombectomy.

Case Report

A 34-year-old man, smoker, was admitted at the emergency room with right hemiparesis, right homonymous hemianopsia, and mild aphasia of sudden onset (National Institutes of Health Stroke Scale [NIHSS] 13). Brain computed tomography was normal. IV recombinant tissue plasminogen activator was started 80 minutes after symptom onset. By the end of perfusion, NIHSS was 10. At 120 minutes after thrombolysis, deterioration occurred (NIHSS 17). Urgent brain computed tomography excluded hemorrhage, and endovascular approach was decided with cerebral angiography disclosing a left middle cerebral artery occlusion (Fig 1). Mechanical clot extraction was performed with partial recanalization of the left middle cerebral artery territory (Fig 1)—symptom recanalization time: 315 minutes. At the end of thrombectomy, NIHSS was 20. Brain magnetic resonance imaging showed only the ischemic infarct (Fig 1). Transesophageal echocardiogram revealed

From the *Department of Neurology, Hospital de Braga, Braga; †Department Cardiology, Hospital de Braga, Braga; ‡Department of Pathological Anatomy, Hospital de São João, Porto; and §Department of Neuroradiology, Hospital de Braga, Braga, Portugal.

Received August 7, 2013; accepted September 15, 2013.

The authors report no disclosures.

Authors contributions—A.F.S.: Study concept, analysis and interpretation of data, literature review, and final manuscript writing; J.P.: analysis and interpretation of data and manuscript review; V.R.: analysis and interpretation of data and manuscript review; J.P.: analysis of the histological specimens and manuscript review, J.R.: analysis and interpretation of data, important intellectual contribute, and manuscript review; and C.F.: analysis and interpretation of data, important intellectual contribute, and final manuscript writing.

Address correspondence to Ana F. Santos, MD, Department of Neurology, Hospital de Braga, Sete Fontes, São Victor, 4710-243 Braga, Portugal. E-mail: filipasantos@hotmail.com.

1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2013.09.018

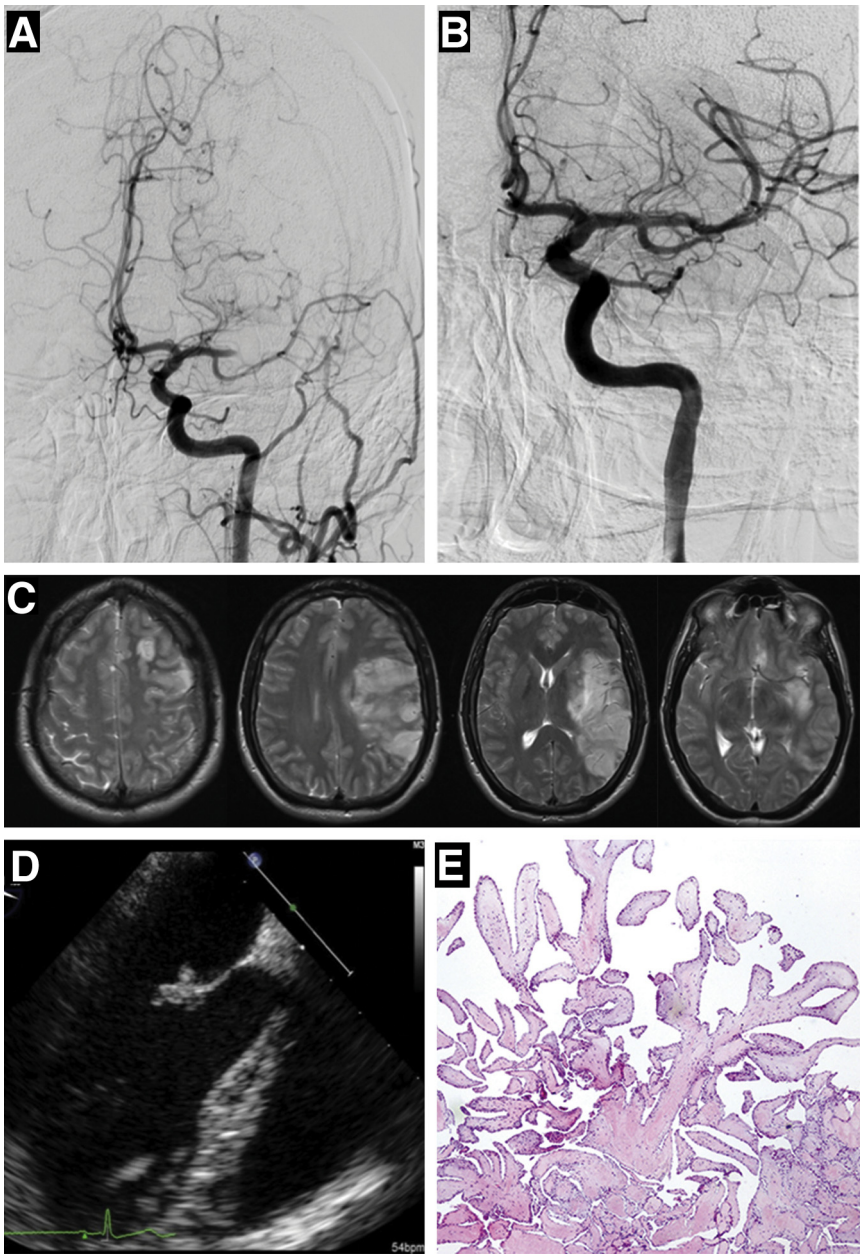

Figure 1. *Cerebral angiography, brain magnetic resonance imaging, transesophageal echocardiogram view, and histological examination. Cerebral angiography showing left M1 middle cerebral artery occlusion (A). After mechanical clot extraction, there was partial recanalization of the left middle cerebral artery territory (B). Axial T2-weighted turbo spin-echo image showing an extensive left cortical–subcortical frontotemporoparietal hypodensity with extension to the insula and striatocapsular region (C). Transesophageal echocardiogram revealing a small mass on the anterior leaflet of the mitral valve, suggestive of cardiac papillary fibroelastoma (D). Histological examination (hematoxylin–eosin stain, ×40) showing a lesion with a papillary configuration lined by flat endocardial cells and with a densely hyalinized central core (E).*

a small mass on the *anterior leaflet* of the mitral valve, suggestive of CPF (Fig 1). Twenty-eight days after stroke, he was submitted to cardiac surgery. Histological examination was consistent with CPF (Fig 1). Clinical evolution was favorable; however, he remained aphasic, with preserved comprehension, and hemiparetic (NIHSS 14).

Discussion

CPF is a potentially treatable cause of stroke. Transesophageal echocardiogram is frequently required for accurate assessment.[2] Definite diagnosis is based on histopathological features.[1] Because of its rarity, there are few data regarding safety and efficacy of thrombolytic therapy in acute ischemic strokes caused by CPF. Only 3 previous reports presented patients with acute ischemic stroke and associated CPF treated with thrombolytic therapy—1 received alteplase intravenously[3] and 2 intra-arterially[4,5]—in one of them intra-arterially thrombolysis was combined with mechanical clot disruption.[5] When embolization is from the thrombotic material surrounding the neoplasm, it could be successfully treated with alteplase; if the embolizing material is a fragment from the tumor, fibrinolytic therapy is probably futile. Patients with known CPF should not be excluded from receiving alteplase but as the embolization could originate in the tumor itself, mechanical thrombectomy might represent a safe and effective treatment option. In our case, the clot removed

during thrombectomy was not submitted to histological examination. But, as its composition can explain the differences in treatment outcome, we suggest routine histological examination for all clots or masses removed during thrombectomy procedures in acute ischemic stroke.

References

1. Gowda RM, Khan IA, Nair CK, et al. Cardiac papillary fibroelastoma: a comprehensive analysis of 725 cases. Am Heart J 2003;146:404-410.
2. Sun JP, Asher CR, Yang XS, et al. Clinical and echocardiographic characteristics of papillary fibroelastomas: a retrospective and prospective study in 162 patients. Circulation 2001;103:2687-2693.
3. Matijevic V, Poljakovic Z, Ilic I, et al. Cardiac papillary fibroelastoma: source of cerebral embolism treated with intravenous thrombolysis. J Stroke Cerebrovasc Dis 2011; 20:485-487.
4. Palazzuoli A, Bruni F, Pasqui A, et al. Cardiac papillary fibroelastoma: an unusual source of cerebral embolism solved with local fibrinolysis. Neurol Sci 2002; 22:469-472.
5. Ljevak J, Mišmaš A, Bazina A, et al. An infrequent type of stroke with an unusual cause and successful therapy: basilar artery occlusion caused by a cardiac papillary fibroelastoma recanalized 12 hours after onset. Intern Med 2013;52:277-279.