Formosan Journal of Surgery (2016) 49, 157–160

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.e-fjs.com

CASE REPORT

Delayed cerebral venous infarction after transarterial embolization of intracranial dural arteriovenous fistula: A rare complication

Chee-Tat Lam [a], Yong-Hui Liu [a,b,*], Chen-Yu Hsiao [c], Hang-Wei Liu [a], Ming-Dar Tsai [a,b]

[a] *Department of Neurosurgery, Shin Kong Wu Ho-Su Memorial Hospital, Taipei, Taiwan*
[b] *Fu Jen Catholic University School of Medicine, Taipei, Taiwan*
[c] *Department of Radiology, Shin Kong Wu Ho-Su Memorial Hospital, Taipei, Taiwan*

Received 23 December 2015; received in revised form 5 February 2016; accepted 21 March 2016
Available online 5 July 2016

KEYWORDS
embolization;
intracranial arteriovenous fistula;
venous infarction

Summary A 34-year-old male patient presented with seizure and was finally diagnosed with intracerebral hematoma because of dural arteriovenous fistula bleeding. Acute onset of hemiplegia developed after 9 days of endovascular therapy. Angiography confirmed venous thrombosis.

1. Introduction

Intracranial dural arteriovenous fistula (DAVF) cases usually present with hemorrhage and neurological deficits at the time of diagnosis. Endovascular therapy is one of the treatment options. Cerebral venous thrombosis (CVT) is one of the most serious complications associated with endovascular techniques. Delayed cerebral venous infarction is a rare complication in the literature.

2. Case Report

A 34-year-old male presented with complex partial seizure. Brain computed tomography (CT), CT angiography (CTA) (Figure 1) and magnetic resonance imaging revealed

Conflicts of interest: The authors have no conflicts of interest relevant to this article.

* Corresponding author. Department of Neurosurgery, Shin Kong Wu Ho-Su Memorial Hospital, Number 95, Wen Chang Road, Shih Lin District, Taipei City, 11101, Taiwan.
E-mail address: nsnicklao@gmail.com (Y.-H. Liu).

http://dx.doi.org/10.1016/j.fjs.2016.03.007

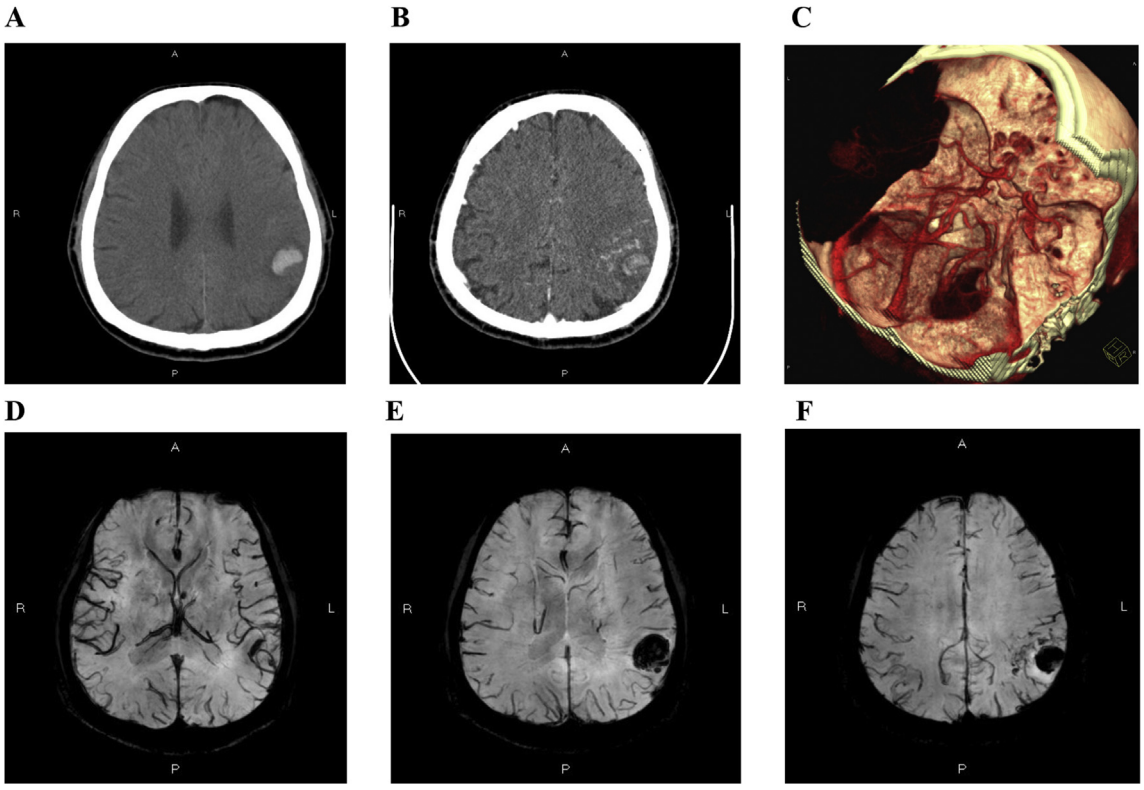

Figure 1 (A) Brain CT shows left parietal ICH; (B) and (C) brain CTA; (D), (E) and (F) SWI. Multiple surrounding engorged vessels are revealed.

a 2.3 cm × 1.4 cm hematoma with multiple surrounding engorged vessels in the left parietal region. Subsequent digital subtraction angiography (DSA, Figure 2) confirmed a Cognard type III DAVF. The DAVF was embolized with Onyx from the left occipital artery through the external carotid artery. Angiography after transarterial embolization (TAE) revealed complete obliteration of the lesion and no occlusion of any other arterial or venous structures. Headache and confusion developed 6 hours after embolization and repeat brain CT (Figure 3C) showed progressive left parietal hematoma with marked perifocal edema and adjacent congested vein. Osmotic agent therapy with mannitol improved his symptoms. Nine days after endovascular therapy, acute onset of right hemiplegia developed. Brain CT (Figure 3D) findings were consistent with left frontal-parietal infarction and brain herniation. DSA (Figures 3E and 3F) confirmed left transverse sinus to sigmoid sinus thrombosis. Revascularization with endovascular angioplasty and thrombolysis were attempted but failed. Emergent left decompressive craniectomy and hematoma evacuation were performed. The patient continued to have right upper-limb weakness after the operation. His disseminated intravascular coagulation profile was checked on the first day after surgery. Fibrinogen, D-dimer, and fibrin degradation product levels were elevated; protein S and antithrombin were reduced, whereas platelet count, global clotting times (i.e., activated partial thromboplastin time and prothrombin time), and protein C were within normal range. Only the protein S was persistently low; the other test results were unremarkable on repeated laboratory studies 2 weeks after surgery.

3. Discussion

The vein of Labbé and sigmoid sinus of the patient were patent before and immediately post-TAE of the DAVF. Consciousness disturbance after the endovascular therapy with new hematoma could be explained by the compromise of the cortical draining vein because of previous hematoma enlargement with perifocal edema. Although osmotherapy improved the initial post-TAE brain edema in our patient, it simultaneously created dehydration status and potentiated the development of silent CVT over 9 days post-TAE. Other than dehydration status, our patient also had risk factors for CVT such as coagulation disorders and hematological conditions. Protein S deficiency was noted on the follow-up laboratory test. Redistribution of the venous drainage pathways and subsequent venous hypertension were suggested as causing the pathophysiological change. However, extensive collateral circulation within the cerebral venous system enables a considerable degree of compensation in the early stages of venous occlusion.[1] This insidious process of CVT could lead clinicians to delay diagnosis. In addition, patients with CVT always present with syndromes clinically indistinguishable from isolated headache and intracranial hypertension.[2]