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The use of routine imaging data in diagnosis of cerebral pseudoaneurysm prior to angiography

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ABSTRACT

Purpose: A false aneurysm is rare and underdiagnosed complication of intracranial hemorrhage. Objective of the study was to point out diagnostic imaging signs of false aneurysm and to determine frequency and diagnostic significance of these signs.

Materials and methods: Cerebral arteriography performed in our center from November 2007 to September 2010 revealed the false aneurysm in 8 patients (4 male, 4 female, mean age was 38 years). During the same angiographic procedure 6 patients were treated by endovascular embolization using coils, mixture of Histoacryl and Lipiodol or Onyx (liquid embolic material). Authors retrospectively analyzed preprocedural studies (computed tomography, magnetic resonance imaging) and angiographic findings to identify signs specific to false aneurysm.

Results: Computed tomographic findings that are not specific but should raise suspicion of the false aneurysm include: enlargement of parenchymal hematoma dimensions, unusual or delayed evolution of hematoma and spot sign associated with acute hematoma expansion. More specific signs can be revealed in digital subtraction angiography that shows a globular shaped neckless aneurysmal sac, delayed filling and emptying of contrast agent and stagnation of contrast with regard to the head position.

Conclusion: Although preangiographic imaging studies findings in patients with false aneurysms are not specific, they should lead to angiographic validation, especially enlarging parenchymal hematoma and atypical hematoma evolution. Digital subtraction angiography makes it possible to diagnose the lesion and to use endovascular embolization techniques, which are currently the method of choice for treatment of pseudoaneurysms.

1. Introduction

Various pathological or idiopathic states that may lead to disruption of the arterial wall are possible causes of intracranial false aneurysms (FAs). They include: trauma, rupture of true cerebral aneurysm or arteriovenous malformation (AVM) [1–6], surgical and endovascular procedures, neoplasm, Marfan's syndrome, fibromuscular dysplasia and vasculitis. FAs of cerebral arteries are reported very rarely [1,7]. Leading cause of intracranial FAs is penetrating trauma. Traumatic pseudoaneurysms occur in less than 1% of patients with cerebral aneurysms [8].

In cases of AVM- or aneurysm-related hemorrhage FAs are situated near the malformation or at the bifurcation of arteries, where usually congenital aneurysms grow. Traumatic pseudoaneurysms are commonly located peripherally or near the skull base [9].

The FA is extravascular blood containment. Few days after the extravasation, the clot undergoes fibrous organization, is excavated by the arterial flow and becomes gradually the sac of a pseudoaneurysm [9]. The fragile wall of FA carries high risk of bleeding. The FA communicates with the artery lumen what enables its growth. Sometimes only massive hemorrhage from pseudoaneurysm of cerebral artery makes the diagnosis possible [10].

Both surgical and endovascular methods are used in the treatment of cerebral FAs [1,6,11]. We applied endovascular therapy.

2. Materials and methods

Eight patients (4 male, 4 female) were diagnosed with the FA in our center from November 2007 to September 2010. The mean age

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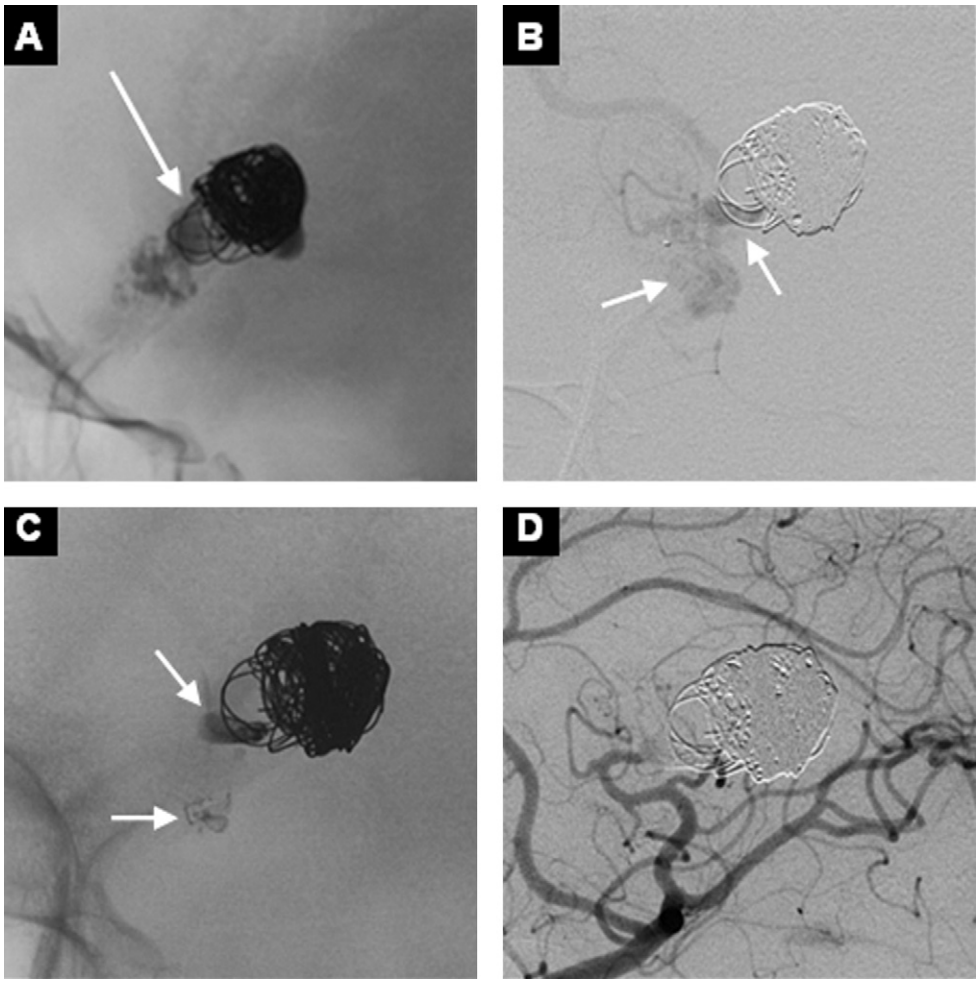

Fig. 1. (A) DSA image taken after complete embolization of AVM and FA with 11 coils and mixture of Histoacryl and Lipiodol (arrow). (B) DSA 17 months after first embolization procedure. Selective angiography shows restored blood flow through part of the AVM and residual inflow to one of the FA chambers (arrows). (C and D) DSA after second embolization with Onyx (arrows). Complete obliteration of FA, 90% AVM size reduction.

was years 38 (age range 19–67 years). On admission all patients presented with meningism signs of various intensities (nuchal rigidity, photophobia and headache) due to subarachnoid hemorrhage (SAH). Six out of eight patients were conscious, in good condition and two of eight patients had Glasgow Coma Scale range from 4 to 5.

Patients underwent non-enhanced computed tomography (NECT) and computed tomography angiography (CTA), which showed intracranial hemorrhage and suggested underlying etiology (true aneurusm or AVM). Magnetic resonance imaging (MRI) was performed before digital subtraction angiography (DSA) only in one patient because of the necessity of urgent endovascular treatment in other cases. FAs were diagnosed by DSA. Two patients underwent postembolization MRI study.

In our study group in five cases FAs were caused by hemorrhage from AVM (5 of 8 patients) and in three cases by rupture of an aneurysm (3 of 8 patients).

Embolization of FA and primary vascular malformation was performed during the same intravascular procedure in 6 out of 8 patients: 4 with AVM-related FA and 2 patients with pseudoaneurysm caused by true aneurysm rupture. In 2 out of 8 cases endovascular treatment of pseudoaneurysm was impossible: in one case because of an acute angle of the origin of the AVM feeding artery and in the other case – small size and vasospasm of the artery at the aneurysm formation. Two patients with ruptured true aneurysms (2 out of 8 patients, Glasgow Coma Scale range from 4 to 5) died in consequence of massive SAH and ischemic stroke secondary to vasospasm.

3. Results

The authors analyzed available literature and designated signs that can indicate the presence of FA. These signs and their frequency in our study group are shown in Table 2. Based on results of imaging studies we found two radiological signs that might suggest FA before DSA procedure: enlarging size of the hematoma and unusual or delayed hematoma evolution on CT or MRI.

The malformations, its locations, embolization materials and results of embolization are shown in Table 1. We applied minimally invasive endovascular treatment of FA in 6 out of 8 cases. In each case the goal was embolization of FA and underlying vascular malformation during one procedure. We used three embolic materials: embolization coils (2 patients), mixture of Histoacryl and Lipiodol (2 patients) and Onyx (3 patients).

In one of the AVM patients we achieved complete occlusion of AVM and large FA (maximum dimension of approximately 3 cm) using simultaneously embolization coils (11 detachable coils with total length of 324 cm) and tissue adhesive (mixture of Histoacryl and Lipiodol) [12]. To the best of our knowledge the strategy of combined coil and Histoacryl embolization has not yet been discussed in the literature. After 6 months the patient was again admitted to our hospital because of SAH. DSA showed