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CORRESPONDENCE

Spontaneous Intracerebral and Intraventricular Hemorrhage

Hemostasis by Transarterial Glue Embolization

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Introduction

This communication concerns a case of severe intracerebral hematoma and intraventricular hemorrhage in a young man, with continuous hemorrhage shown on angiography that was controlled by transarterial occlusion of a bleeding thalamoperforating artery. The pathophysiology of continuous intracranial bleeding in this particular case is discussed.

Case Report

A 21-year-old man complained of headache and nausea, and presented progressively impaired consciousness. Ambulance personal reported that the patient was comatose and had spontaneous episodes of stereotyped extension of the extremities during transfer. On admission to the emergency department, he was comatose, presented stereotyped extension movements of arms and legs on pain stimulation (Glasgow Coma Scale score 4), the left pupil was dilated compared to the right with slight direct response to light on both sides, and blood pressure was 160/95 mmHg. The patient was intubated. A head computed tomography (CT) scan showed a hematoma in the left thalamus and massive intraventricular hemorrhage (Fig. 1). A right frontal external ventriculostomy was placed. Cerebral digital subtraction angiography (DSA) was done 6 h after symptom onset using a right transfemoral approach. DSA of the left vertebral artery (VA) showed leakage of contrast agent into a pseudoaneurysm coming from a thalamogeniculate branch of the left posterior cerebral artery (Fig. 2). The angiography catheter was withdrawn, and the left VA was catheterized with a 5-F guide catheter (Envoy®, Cordis Endovascular Systems, Miami Lakes, FL, USA). The pseudoaneurysm was catheterized and obliterated (Fig. 3). Pseudoaneurysm catheterization was done with a microcatheter Ultraflow 1.5 F with the help of a microwire Mirage (all ev3 Europe, Paris, France). The microcatheter was flushed very slowly with 0.3 ml 5% glucose, and embolized with 0.4 ml of a 75% solution of N-butyl-2-cyanoacrylate and methacryloxysulfolane (Glubran-2®, GEM Srl, Viareggio, Italy) in iodized poppy-seed oil (Lipiodol® Ultra Fluide, Laboratoire Gerbet, Aulnay-sous-Bois, France). The number of DSA runs was kept to a minimum to avoid increased contrast agent leakage, but an oblique view was considered to give a better working projection, and the run shown in Fig. 2 was done after guide catheter change. The time elapsed between the DSA run in the working projection, done just before introduction of the microcatheter, and successful catheterization of the pseudoaneurysm was 8 min. Glue injection was done to the extent that the embolic agent presumably formed a plug around the ruptured artery, and there was no resistance on pulling out the microcatheter. DSA of the left VA after embolization showed obliteration of the pseudoaneurysm (Fig. 4). Immediate postembolization CT scan showed an increase in hematoma size compared to the preembolization images (Fig. 5). Later the same day, small bilateral frontal craniotomies were done to evacuate intraventricular clots, and bilateral external ventriculostomies were placed. The patient was treated in a specialized neurosurgical intensive care unit (NSICU) using a standard management protocol for high intracranial pressure according to the Lund concept [1]. Three days later, control of the intracranial pressure was considered not optimal, thus bilateral decompressive craniectomies were done. The neurological condition did not

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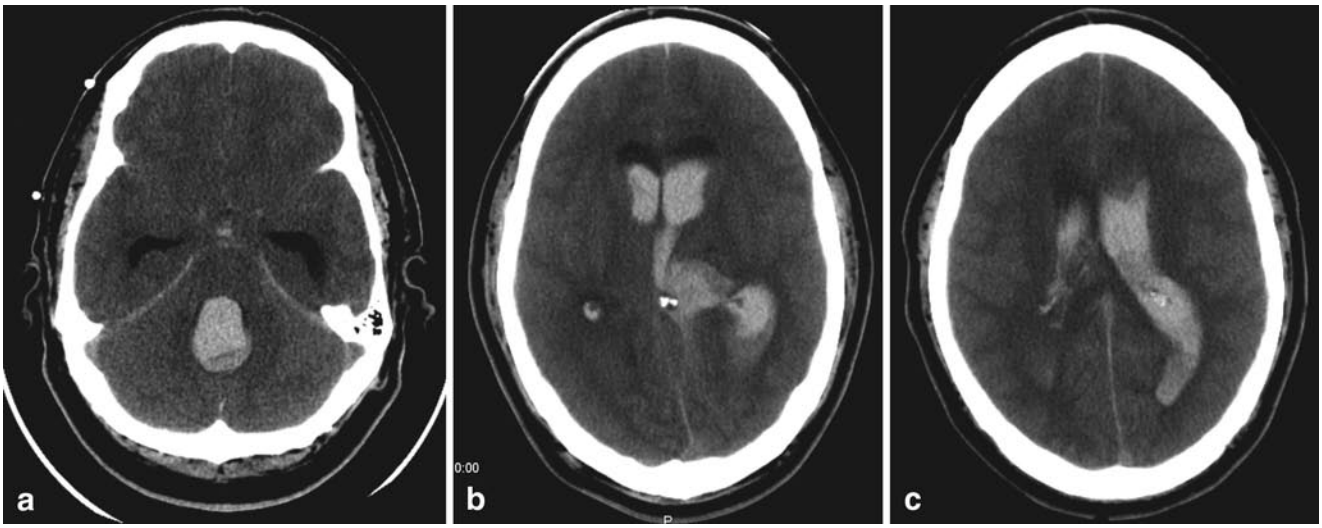

Fig. 1 Head CT scan showing an intraparenchymal hematoma in the left thalamus (**b**) with massive rupture into the ventricular system (**a**, **c**)

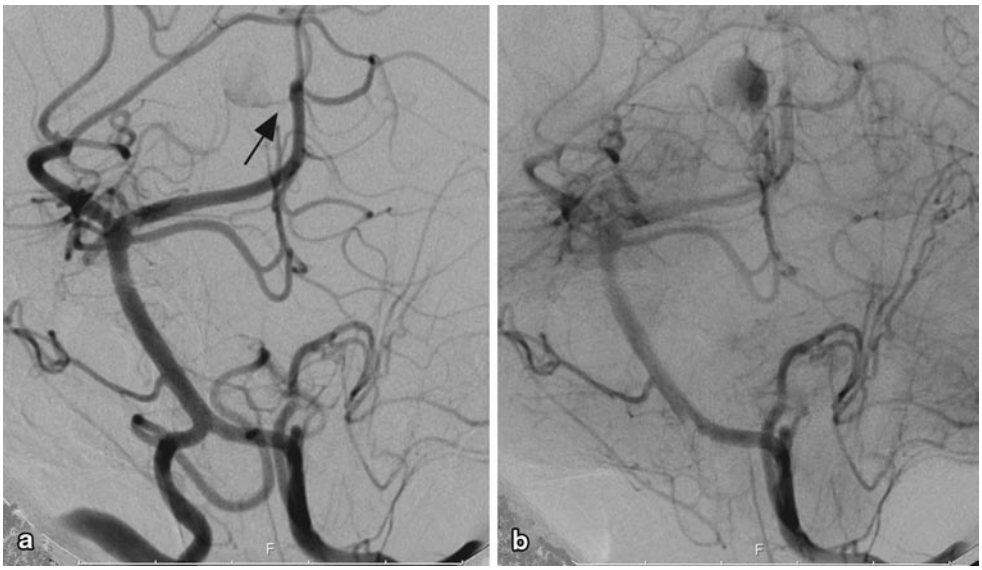

Fig. 2 DSA of the left VA, oblique view, in early (**a**) and late (**b**) arterial phases. The *arrow* in **a** points to leakage of contrast agent from a thalamogeniculate branch that came from the left posterior cerebral artery at P2–P3 segment junction

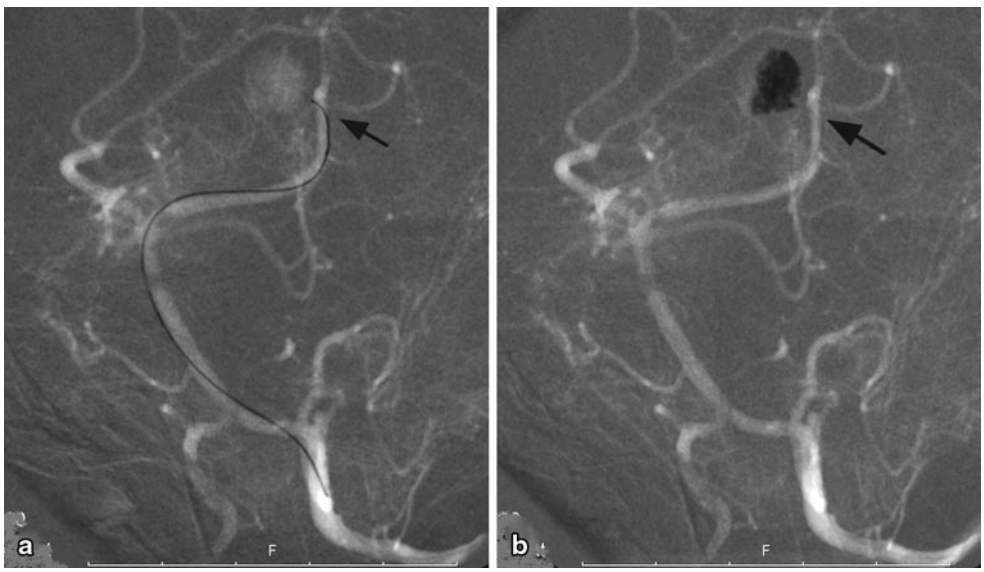

Fig. 3 Roadmap in the same projection as Fig. 2: **a** during catheterization of the pseudoaneurysm (*arrow* points to the guidewire tip); **b** after glue injection and just before removal of the microcatheter (*arrow* points to the microcatheter tip)

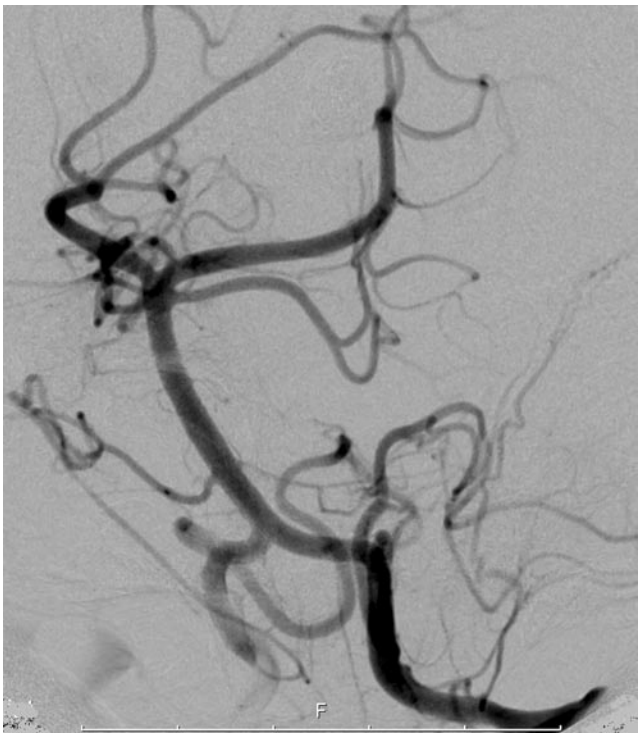

Fig. 4 DSA of the left VA after embolization showing obliteration of the pseudoaneurysm

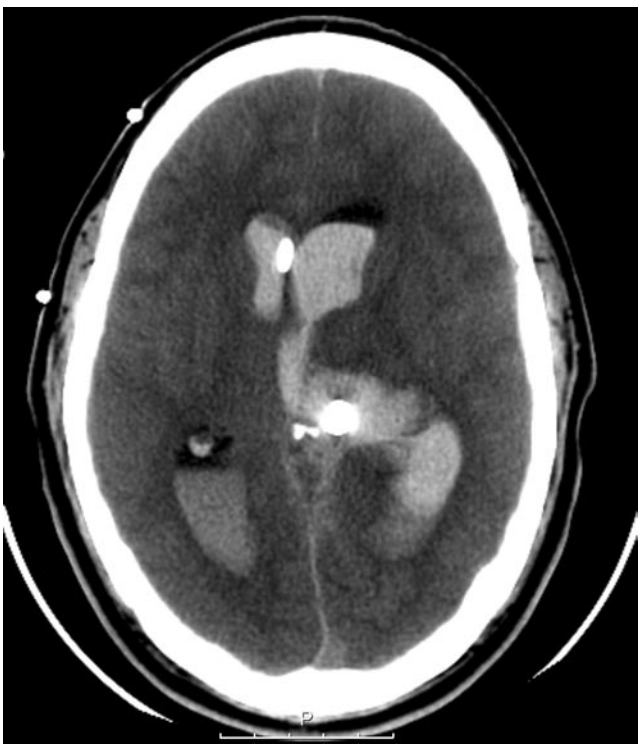

Fig. 5 CT scan after external ventriculostomy (the tip of the intraventricular catheter is seen in the right frontal horn) and embolization of the pseudoaneurysm (dense glue is seen in the thalamic hematoma). The hematoma, as well as the intraventricular blood clot, is larger than in the preembolization CT scan

improve, and the patient had a fatal outcome after 40 days of NSICU care. Head CT scans were repeated several times during this period, and there was no evidence of rebleeding of the thalamic lesion.

Discussion

Spontaneous intracerebral hemorrhage (ICH) represents 10–15% of first-ever strokes, with a high mortality (35–50% overall mortality at 30 days, half of the deaths occurring in the first 2 days), and severe functional impairment in survivors (in the USA, only 20% of patients are expected to be functionally independent at 6 months). For deep hemorrhages, mortality at 1 year is 51% [2]. Chronic arterial hypertension is the most common etiology, and other well-known etiologies include cerebral amyloid angiopathy, vascular malformations, tumors, use of recreational drugs, vasculitis, coagulopathies, and cerebral venous thrombosis [2, 3]. In spontaneous ICH. the bleeding event is acute, and the symptoms are rapidly progressive during the first hours. Factors affecting the size of the hematoma and severity of neurological damage are blood pressure, hemorrhage site, size and physical condition of the ruptured vessel, previous integrity of the brain tissue, state of autoregulation of the cerebral blood flow, and state of the hemostatic system. The amount of brain tissue that is primarily destroyed is usually small since the hematoma progresses by dissection along the path of least resistance between fiber tracts, pushing aside and compressing the adjacent brain tissue. Provided the hemostatic mechanisms are intact, clot formation and cessation of hemorrhage occur within a few minutes from ictus, but the volume of the hematoma has been shown to increase by more than 33% in serial CT scans in 38% of patients initially scanned within 3 h after symptom onset [2], indicating that these patients present rebleedings or continuous bleeding. In the early angiographic literature [4], leakage of contrast material into the hematoma during angiography had been observed in some cases up to several hours after ictus, and it was recognized as an indicator of poor prognosis. In the recent literature, ongoing hemorrhage has been recognized as contrast agent extravasation into an ICH at multidetector CT angiography (MDCTA). This image finding, which has been called the spot sign, is a strong predictor of hematoma enlargement [5]. In a recent study [6], the spot sign was detected in 133 of 573 consecutive patients with ICH examined with MDCTA (23.2%), and it was a predictor of in-hospital mortality and poor outcome among survivors in nontraumatic ICH.

In the present case, the patient had no previous history of arterial hypertension, and any other etiology for nontraumatic ICH [2, 3] could not be established. A potential etiology for the hemorrhage is a microarteriovenous malformation in

the left thalamus, but the imaging findings do not support this hypothesis, since the bleeding artery presented a normal (small) caliber and no early-filling vein was demonstrated on DSA. The bleeding artery could be identified on DSA as a thalamogeniculate perforating artery that originated from the left posterior cerebral artery at the P2–P3 junction (vessel nomenclature and angiographic topography according to Margolis et al. [7]). The primary thalamic hemorrhage ruptured massively into the 3rd ventricle, resulting in severe hydrocephalus and increased intracranial pressure. Even if opening of the hematoma into the ventricles in a first period decompressed the blood clot (in this way minimizing the primary thalamic lesion), the bleeding could not be stopped by physiological hemostatic mechanisms because of a continuous passage of noncoagulated blood to the cerebrospinal fluid (CSF) space. A pressure gradient between the bleeding artery and the extravascular pressure (in this case the CSF pressure) was thus maintained and, theoretically, the "bleeding pressure" was roughly equivalent to the cerebral perfusion pressure. The dynamics of this type of intracranial bleeding has been well described in experimental conditions [8]. The sequential course of events could also be compared to the mechanism of rapid volume increase of traumatic hyperacute epidural bleeding, when arteriovenous shunting occurs to diploic or meningeal veins [9]. It is noteworthy that despite unrestricted distribution of the blood in the ventricles the bleeding spot on the DSA images as well as the liquid embolic agent were conformed to an almost spherical shape. The steady bleeding rate was surely lower than the rate of contrast agent extravasation during angiography, since a raise of the intravascular pressure is a phenomenon that is intrinsic to the procedure. The injection of contrast agent (a viscous liquid) at the beginning of the DSA run caused an instantaneous pulse of liquid extravasation through the ruptured artery that entered a cavity (the extravascular space, in this case a ventricle-communicating hematoma), where the incoming liquid was first subject to forces opposing its entry that were approximately equal in all directions (i.e., isotropic), explaining the spherical shape of the contrast agent "drop".

Endovascular embolization of the bleeding artery was unproblematic in the present case, and it could unquestionably have been performed by all neurointerventionists with experience in embolization of cerebral arteriovenous malformations. Since patients with primary ICH that have an ongoing hemorrhage can be reasonably identified by the presence of the spot sign at MDCTA, it is appealing to speculate that these patients should undergo an emergency DSA and, if feasible, embolization of the bleeding artery. For these patients one such procedure would be potentially lifesaving. However, endovascular embolization will surely not be possible in all cases, since MDCTA can show multiple spots of contrast agent extravasation [5], and bleeding perforating arteries may not always be as evident on DSA images or as accessible to catheterization as in the present case. A promising therapy to stop this kind of bleeding is intravenous treatment with recombinant activated factor VII (rFVIIa), but the available evidence on efficacy and safety does not seem to motivate the use of rFVIIa for ICH outside of a clinical trial [2, 10].

Conflict of Interest Statement The author declares that there is no actual or potential conflict of interest in relation to this article.

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