Case Report

Fatal Traumatic Subarachnoid Hemorrhage due to Acute Rebleeding of a Pseudoaneurysm Arising from the Distal Basilar Artery

Byung Chul Kim, M.D., Jae Il Lee, M.D., Won Ho Cho, M.D., Kyoung Hyup Nam, M.D.
Department of Neurosurgery, Medical Research Institute, Pusan National University Hospital, Pusan National University School of Medicine, Busan, Korea

Isolated traumatic pseudoaneurysms of the basilar artery are extremely rare but often fatal resulting in a mortality rate as high as 50%. A 51-year-old man presented with craniofacial injury after blunt trauma. A brain computed tomography (CT) scan showed thick basal subarachnoid hemorrhage associated with multiple craniofacial fractures, while CT angiography revealed contrast extravasation at the distal basilar artery with pseudoaneurysm formation. After this primary survey, the condition of the patient suddenly deteriorated. Conventional angiography confirmed the contrast extravasation resulted from pseudoaneurysm formation, which was successfully treated with endovascular coil embolization. Decompressive craniectomy and coma therapy with propofol were also performed. However, the patient died on the 7th hospital day because of the poor initial clinical condition. The current case is the first report of acute pseudoaneurysm rupture arising from the basilar artery within the first day after trauma. Our findings suggest the possibility that pseudoaneurysm rupture should be considered if brain CT shows thick traumatic subarachnoid hemorrhage on the basal cistern with a basal skull fracture.

Key Words: Traumatic brain injury · Subarachnoid hemorrhage · Basilar artery · Pseudoaneurysm.

INTRODUCTION

Traumatic aneurysms arise primarily from the anterior circulation and less frequently from the posterior circulation at a reported incidence of approximately 10% (1,6). Correspondingly, isolated traumatic pseudoaneurysms of the basilar artery are extremely rare because of the secure location of this artery (1), but are associated with a high mortality, presumably because of pseudoaneurysms growing faster and showing higher rebleeding than true aneurysms (2). Therefore, the majority of traumatic basilar artery injuries have been diagnosed in forensic studies. Indeed, to our knowledge, traumatic basilar artery pseudoaneurysm has been reported in only seven clinical cases (1,3,5,6,8,9). We report here an extremely rare case of traumatic pseudoaneurysm arising from the basilar artery. The traumatic pseudoaneurysm presented with acute pseudoaneurysm formation and rebleeding within the first day after trauma, and was managed with endovascular coil embolization.

CASE REPORT

A 51-year-old man was presented to the emergency room after ground-level fall in a state of inebriation. The patient had craniofacial trauma with multiple abrasions, ecchymosis and heavy nose bleeding. On arrival, he was somnolent but was able to obey simple orders (Glasgow Coma Scale: 12). The initial vital signs were stable (blood pressure: 140/100 mm Hg, pulse rate: 80/min). Brain computed tomography (CT) showed thick subarachnoid hemorrhage (SAH) on the basal cistern with clival fracture and traumatic intracranial hemorrhages. Diffuse SAH was predominant in the basal cistern, ambient cistern, left sylvian fissure, preoptic cistern, both cerebellopontine cistern and premedullary cistern (Fig. 1). CT angiography revealed contrast extravasation on the distal basilar artery (Fig. 2). Based on this finding, we decided to conduct conventional angiography for confirming the aneurysm and performing subsequent endovascular management. However, during the preparation of the angiography, the

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* Received: April 21, 2014  •  Revised: July 25, 2014  •  Accepted: August 18, 2014  •  Address for reprints: Kyoung Hyup Nam, M.D.  •  Department of Neurosurgery, Medical Research Institute, Pusan National University Hospital, Pusan National University School of Medicine, 179 Gudeok-ro, Seo-gu, Busan, Korea  •  Tel: +82-51-240-7257, Fax: +82-51-244-0282, E-mail: namdark@daum.net  •  This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/3.0) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.
A patient fell rapidly into a coma and showed bilateral pupil dilatation (Glasgow Coma Scale: 3). Because of hemodynamic and respiratory instability (blood pressure: 60/40 mm Hg, pulse rate: 120/min), we made the decision to perform conventional angiography after resuscitation. Repeated brain CT revealed the SAH had increased in size (Fig. 3).

The conventional cerebral angiography confirmed that a pseudoaneurysm arising from the top of the basilar artery corresponded with the contrast extravasation. The size of the aneurysm was 8.5×7.4×3.9 mm. Transarterial coil embolization of the pseudoaneurysm sac and the rupture point of the distal basilar artery was achieved using a double-microcatheter technique (Fig. 4). Following the embolization, bilateral decompressive craniectomy with duraplasty was performed. Although the patient was in intensive medical care, his consciousness did not improve, and he died on the 7th hospital day because of deterioration of his initially poor clinical condition.

**DISCUSSION**

Traumatic pseudoaneurysms may occur following penetrating or blunt head trauma. For penetrating injury, the mechanism of traumatic aneurysms is direct injury from the bone fragments of skull fractures; in blunt injury, the mechanism is overstretching, torsion, or compression against the dura or bony prominences. Traumatic aneurysms of the basilar artery are almost associated with basal skull fractures, and the arterial wall can be lacerated by a fractured clivus or tentorium secondary to rapid deceleration. Traumatic intracranial aneurysms usually become symptomatic after a symptom-free interval following the traumatic episode. In many cases, clinical symptoms or neurologic deterioration of traumatic aneurysms may present within several weeks after the traumatic events. Although cerebral angiography cannot reveal a pseudoaneurysm within 1 or 2 days after trauma, a follow-up angiography obtained several weeks later may identify an aneurysm.

Unlike previously reported cases, the present one showed acute formation of a pseudoaneurysm and deterioration within several hours after trauma due to rebleeding. The authors presumed that overstretching of the perforating artery formed the pseudoaneurysm and that the acute bleeding was arrested temporarily by the adjacent thick hemorrhage. The unexpected rebleeding might have led to serious deterioration because of the increased blood pressure or clot lysis. Because the time interval seemed to be short for clot lysis, sudden increased blood pressure was more reliable cause of the rebleeding.

Because of the high mortality associated with traumatic pseudoaneurysms, early diagnosis and prompt treatment are highly important. The several reported treatment options include surgical repair and endovascular embolization. An examination of the recent literature suggests that endovascular treatment has become preferable to surgical repair, and the preference for this treatment option is likely due to traumatic pseudoaneurysms hav-
ing friable walls and poorly defined neck. In our case, endo-
vascular coil embolization was used for the management of the
pseudoaneurysm in consideration of the patient's poor clinical
condition and the deep location of the pseudoaneurysm. Al-
though occlusion of the parent artery was considered, coil em-
bolization was performed because of the risk of a fatal condition
secondary to occlusion of the basilar artery.

The long-term prognosis is heavily influenced by the extent of
other primary and secondary brain injuries. Thus, prompt treat-
ment could prevent massive hemorrhage and secondary brain
injury, such as brain swelling. Endovascular embolization can be
an effective method for the management of pseudoaneurysms,
especially posterior circulation aneurysm with poor clinical grade.
Furthermore, brain CT angiography should be performed in all
patients with traumatic SAH for the identification of possible
vascular injury, especially thick SAH around brain stem or SAH
with basal skull fractures.

CONCLUSION

This report describes a rare case of traumatic basilar pseudoa-
neurysm caused by severe SAH within 1 day after trauma. Early
detection and prompt management could prevent catastrophic
rupture of a traumatic pseudoaneurysm. Therefore, brain CT
angiography and endovascular management should be consid-
ered for identifying vascular injury in severe traumatic SAH with
a basal skull fracture.

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Fig. 4. A: Conventional angiography demonstrating pseudoaneurysm arising from distal basilar artery. B: Angiography confirming complete occlusion of the pseudoaneurysm sac and rupture point of distal basilar artery. C: Left internal cerebral artery angiography demonstrating posterior cerebral artery with collateral flow through the posterior communicating artery.