Case Report

Delayed massive epistaxis from traumatic intracranial aneurysm after blunt facial injury

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Cases: Traumatic intracranial aneurysm following blunt head injury is uncommon but can be induced by extension of skull base fracture and causes unexpected hemorrhagic complications. We present two cases of traumatic intracranial aneurysm in the paraclinoid area that was revealed by delayed massive epistaxis. Lack of initial neurological deficits omitted screening for cerebrovascular injury.
Outcome: Internal trapping was carried out using endovascular techniques in both cases, with extracranial–intracranial bypass in one case. No recurrent bleeding occurred in either case.
Conclusion: To prevent unexpected delayed life-threatening hemorrhagic accidents, careful assessment of skull-base fracture is prerequisite, even in cases of mild facial injury.

Key words: Blunt facial injury, endovascular trapping, extracranial–intracranial bypass, massive epistaxis, traumatic intracranial aneurysm

INTRODUCTION

SCREENING FOR CEREBROVASCULAR injury is undertaken in cases showing neurological symptoms after severe head trauma.[1] However, little attention is generally paid in cases of mild facial trauma without neurological symptoms.

We encountered two cases of traumatic intracranial aneurysm (TICA) revealed as delayed massive epistaxis after blunt facial injury. Both patients were diagnosed with facial bone fracture in the primary care hospital and no additional examination was carried out. However, skull-base fractures extending to the paraclinoid area were detected later and thought to be the cause of the aneurysms.

These two cases illustrate the importance of careful assessment of skull-base fractures, even in cases of mild facial trauma. If a fracture extends to the paraclinoid area,

screening for cerebrovascular injury must be carried out.[2] Conversely, if a patient suffering massive epistaxis is encountered, history of facial trauma should be assessed.

CASES

Case 1

A 48-YEAR-OLD MAN was admitted to a local hospital after a motor vehicle accident without loss of consciousness. Zygomatic and maxillary bone fractures were diagnosed, but detailed assessment with additional radiological examination was not carried out. He was discharged on the 4th day after conservative treatment with no additional event.

Massive epistaxis from nostrils occurred 10 and 15 days after the accident, and he was brought to our hospital. His condition was a shock state with severe hypotension (blood pressure [BP], 59/42 mmHg; heart rate, 70 b.p.m.; hemoglobin, 6.5 g/dL; hematocrit, 19.6%), but he recovered with blood transfusion. Computed tomography (CT) and CT angiography (CTA) revealed a skull-base fracture extending to the superior wall of the ethmoidal sinus and sella turcica (Fig. 1A–D), and right internal carotid artery (ICA)
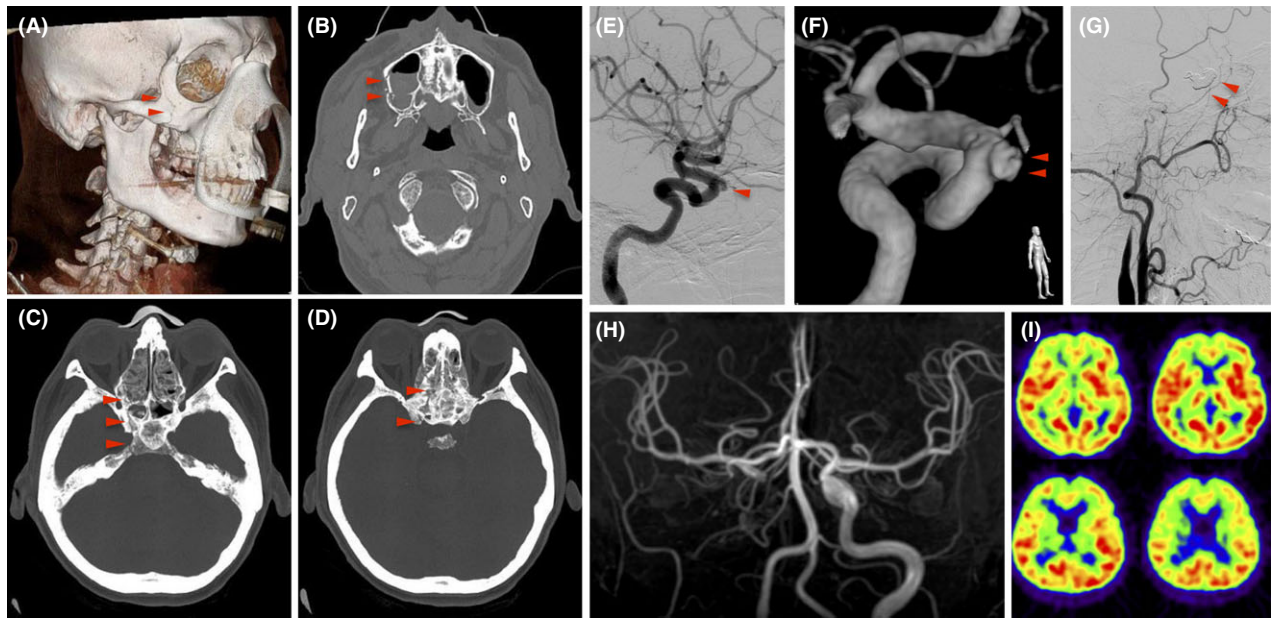
Fig. 1. Case 1 of delayed massive epistaxis from traumatic intracranial aneurysm after blunt facial injury. A, B, Three-dimensional image and axial-slice computed tomography illustrating zygomatic bone fracture (A, arrowheads) and maxillary bone fracture (B, arrowheads). C, D, Maximum intensity projection image of thin-slice computed tomography showing extension of the fracture line to the sella turcica and ethmoid bone (arrowheads). E, Angiography revealing aneurysmal formation on the internal carotid artery in the paraclinoid area (arrowhead). F, Three-dimensional image of the aneurysm acquired by 3D-rotational angiography (arrowheads). G, Internal trapping using detachable coils (arrowheads). H, Magnetic resonance angiography after endovascular treatment. Collateral flow is seen through the anterior communicating artery and posterior communicating artery. I, Positron emission tomography at 12 months postoperatively. No decline in cerebral blood flow is apparent.

angiogram illustrated a small irregular-shaped aneurysm (3 mm) at the paraclinoid area (Fig. 1E, F). As it was thought to be a pseudoaneurysm, we planned to perform internal trapping.

Balloon occlusion test (BOT) was undertaken before the treatment under local anesthesia. The right ICA was occluded with a balloon catheter for 20 min, and stump pressure was measured continuously. The mean stump pressure decreased by 50% of the mean systemic BP, but no neurological symptom appeared during BOT.

Following the BOT, internal trapping of the ICA from the proximal site of the origin of the ophthalmic artery to the cavernous portion was carried out using electrical detachable coils (Fig. 1G). Magnetic resonance angiography and positron emission tomography revealed no declines in cerebral blood flow in the ipsilateral hemisphere after 12 months (Fig. 1H, I). No adverse events have been observed for 36 months after treatment.
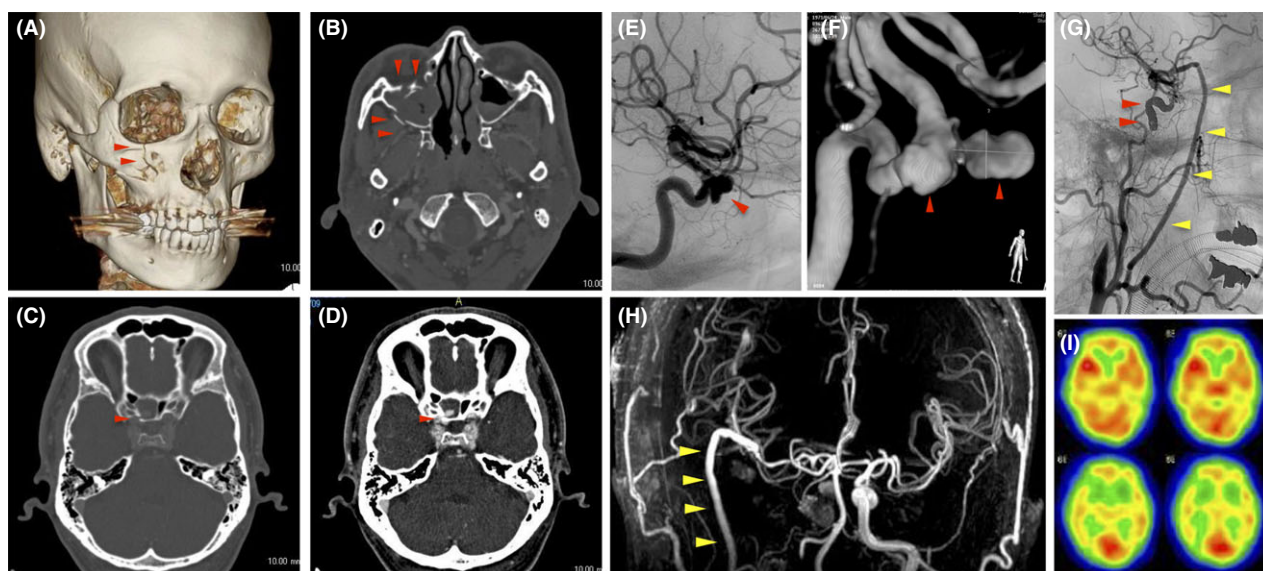
Case 2

A 38-year-old man was admitted to a local hospital after a motor vehicle accident without loss of consciousness.

Zygomatic and maxillary bone fractures were diagnosed, and he was discharged on the 5th day after conservative treatment.

Massive epistaxis from nostrils occurred on the 14th day and he was taken to the same hospital. Physiological data and blood examination (BP, 80/50 mmHg; heart rate, 96 b.p.m.; hemoglobin, 10.2 g/dl; hematocrit, 30.2%) suggested arterial injury and he was transferred to our hospital. Computed tomography and CTA revealed a skull-base fracture extending to the lateral wall of the sphenoid sinus and pooling of contrast medium in the sphenoid sinus near the fracture (Fig. 2A–D). Right ICA angiogram revealed an irregular-shaped aneurysm (5 mm to anterior and 7 mm to medial side) at the paraclinoid area (Fig. 2E, F). As the injured vessel was thought to be unstable, we planned to perform internal trapping.

Balloon occlusion test was carried out under local anesthesia. As soon as the right ICA was occluded, the mean stump pressure decreased by 20% of the mean systemic BP with left hemiparesis and disturbance of consciousness. This result implied that bypass surgery was essential to preserve the cerebral blood flow before internal trapping.

Fig. 2. Case 2 of delayed massive epistaxis from traumatic intracranial aneurysm after blunt facial injury. A, B, Three-dimensional image and axial-slice computed tomography showing maxillary bone fracture (arrowheads). C, Axial-slice CT angiography showing the fracture line at the lateral wall of the sphenoid sinus. D, Pooling of contrast medium in the sphenoid sinus, suggesting traumatic pseudoaneurysm. E, Angiography revealing aneurysm formation on the internal carotid artery in the paraclinoid area (arrowhead). F, Three-dimensional image of aneurysm acquired by 3D-rotational angiography. G, Extracranial–intracranial bypass using radial artery graft (yellow arrowheads) and internal trapping using detachable coils (red arrowheads). H, Post-procedural magnetic resonance angiography showing collateral flow through radial artery graft (arrowheads). I, Single photon emission computed tomography at 1 month after procedures, showing no decline in cerebral blood flow.

Following the BOT, extracranial–intracranial bypass surgery was carried out under general anesthesia by using an autologous radial artery graft from the cervical external carotid artery to the right middle cerebral artery. Then internal trapping from the proximal site of the ophthalmic artery to the petrous portion was carried out on the same day while continuing general anesthesia (Fig. 2G, H). Although the embolization of the distal portion was rather rough because of the irregularity of the injured vessel, complete occlusion of the pseudoaneurysm was confirmed (Fig. 2G). No neurological symptom appeared after the procedure and single-photon emission CT revealed no decline in cerebral blood flow in the ipsilateral hemisphere on the 17th day postoperatively (Fig. 2I). No adverse events have been observed for 27 months after treatment.

DISCUSSION

Screening for blunt cerebrovascular injury

THE INCIDENCE OF blunt cerebrovascular injury (BCVI) among all blunt trauma patients was reported as <1% in the 1990s,[2] but has recently increased to 2–3% with aggressive screening protocols and advances in imaging technologies.[1]

The screening for BCVI has been usually undertaken for patients who have suffered severe head trauma with neurological symptoms.[1] However, even in cases with no neurological findings, skull-base fractures extending to the paraclinoid area can cause arterial injuries.[2] Careful assessment of images to detect skull-base fractures and BCVI is thus important to prevent life-threatening hemorrhagic complications in all cases of blunt facial injury.

Traumatic intracranial aneurysm

Traumatic intracranial aneurysm is a type of BCVI diagnosed angiographically, based on irregular shape of the arterial wall and delayed filling of the sac at a non-branching portion.[3,4] The incidence of TICA was reported to be approximately 1% among intracranial aneurysms, and 0.65% in blunt traumatic brain injury patients.[5,6]

Interestingly, Komiyama *et al.*[4] reviewed previous reports and revealed that the rate of positive detection of TICA on day 0 was only 54%. This suggests that some period might be needed for "maturation" of TICA in almost half of cases,

and implies the difficulty of identifying these lesions with initial images. As bleeding from traumatic aneurysms often occurs within 2 weeks after onset,[4,7] repeated screening should be considered in cases in which skull-base fracture near the major vessels or vessel wall irregularity was detected.[5]

Treatment for TICA

Surgical intervention should be planned for TICA, because mortality rates with conservative and surgical treatment are 41% and 18%, respectively.[7] Parent artery occlusion is desirable for radical cure, and in cases involving the skull-base region, internal trapping is reasonable rather than open surgery because of the accessibility.[8]

Balloon occlusion test might be useful to clarify collateral flow from other vascular territories. In addition to neurological assessment, stump pressure (>50%) might help to judge ischemic tolerance.[9] If the collateral flow was not enough, bypass surgery should be undertaken in advance of internal trapping.

Stent placement was reported as a new technique for treating TICA.[8,10] This represents an attractive procedure, because the patency of the parent artery can be maintained. However, the indications should be discussed with precise assessment of coexisting injuries as antithrombotic therapy is essential postoperatively.[8]

CONCLUSION

WE REPORTED TWO cases of TICA with delayed massive epistaxis after blunt facial injury without any neurological deficits. Careful evaluation of skull-base fractures and BCVI is important to prevent unexpected delayed life-threatening hemorrhage.

CONFLICTS OF INTEREST

NONE.

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