



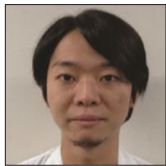
Case Report

# Paramedian thalamic infarction caused by cisternal drain placement in open clipping for aneurysmal subarachnoid hemorrhage: Two case reports

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## ABSTRACT

**Background:** Some complications associated with cisternal drainage have been reported; however, there are few reports on direct vascular injury caused by cisternal drain. We experienced two rare cases of thalamic infarction caused by cisternal drain placement during open clipping for a ruptured anterior communicating artery (AcomA) aneurysm through an anterior interhemispheric approach.

**Case Description:** Two cases of ruptured AcomA aneurysm were treated by surgical clipping through an anterior interhemispheric approach, and then a cisternal drain was inserted from opticocarotid space toward prepontine cistern. Postoperatively, the magnetic resonance imaging showed unilateral anterior-medial thalamic infarction in both two cases. By reviewing the postoperative computed tomography and digital subtraction angiography, it was suspected that the cisternal drain, which was inserted slightly deep, obstructed the P1 perforator because of an anatomical variation involving a lowered basilar bifurcation and caused postoperative unilateral paramedian thalamic infarction.

**Conclusion:** To avoid these complications, neurosurgeons should consider the potential for P1 perforator injury related to cisternal drain placement.

**Keywords:** Cisternal drainage, Complication, Subarachnoid hemorrhage, Thalamic infarction

## BACKGROUND

Cerebral vasospasm often occurs in subarachnoid hemorrhage (SAH) patients secondary to a ruptured intracranial aneurysm and is a known prognostic factor for poor outcome. The spasmogenic substances released from the subarachnoid blood clots, and vascular hyperreactivity, are considered the leading causes of cerebral vasospasm.<sup>[6,12,14]</sup> Cisternal drains are often placed during open clipping for aneurysmal SAH patients, to provide a continuous draining passage and administration route for anti-clot drugs (e.g., Urokinase, and tissue plasminogen activator). This can aid in rapid removal of subarachnoid clots from the cerebrovascular cistern, and thus help prevent complications associated with cerebral vasospasm.<sup>[5,7,11,16,17]</sup> On the other hands, there is an association of long-term cisternal drain use with meningitis and delayed

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hydrocephalus.<sup>[10]</sup> However, there are few reports on direct vascular injury associated with cisternal drain placement. Herein, we present two rare cases of thalamic infarction caused by cisternal drain placement during open clipping for a ruptured anterior communicating artery (AcomA) aneurysm.

## CLINICAL PRESENTATION

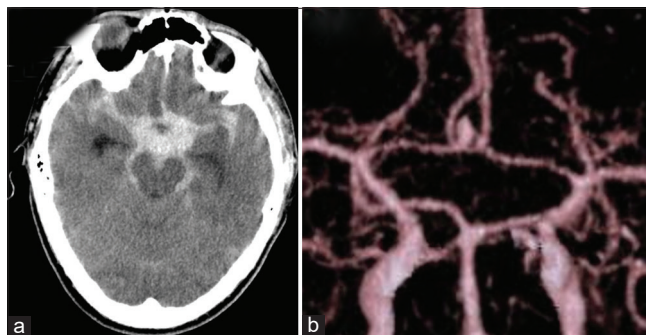
### Case 1

A 44-year-old man presenting with severe headache and disturbance in consciousness was admitted to our hospital with a Glasgow Coma Scale of 14 (E3V5M6), and no focal symptoms. Three-dimensional computed tomography (CT) scan revealed diffuse thickened SAH [Figure 1a] and an upward-protruding AcomA aneurysm [Figure 1b]. We diagnosed as SAH, World Federation of Neurological Surgeons Grading System for Subarachnoid Hemorrhage (WFNS) Grade 2. We performed open clipping through an anterior interhemispheric approach, and the aneurysm was completely obliterated. We subsequently gently retracted the right frontal lobe posteriorly, opened the chiasmatic and carotid cisterns, and placed a cisternal drainage tube (2.5 mm×100 cm, SILASCON, E-3L-12, Kaneka Medix Corporation, Osaka, Japan) from the opticocarotid space toward the prepontine cistern.

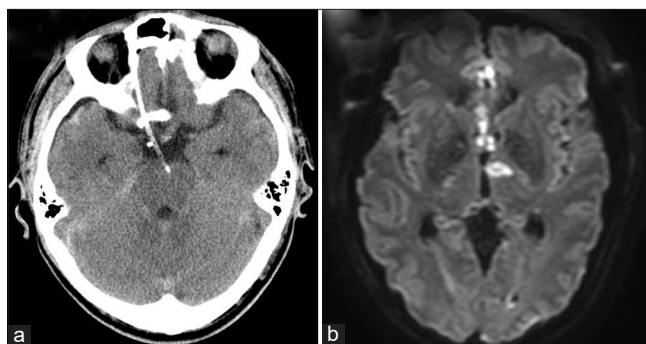
Postoperatively, CT scan demonstrated that tip of the cisternal drain was placed at the interpeduncular fossa [Figure 2a], and diffusion-weighted magnetic resonance imaging (MRI) revealed a left paramedian thalamic infarction [Figure 2b]. However, because of the cisternal drain, the subarachnoid clots were quickly removed, with no evidence of cerebral vasospasm. Postoperative CT and digital subtraction angiography (DSA) showed the tip of cisternal drain placed above the basilar bifurcation [Figure 3]. Thus, we suspected that perforator injury arising from the P1 segment of the posterior cerebral artery caused the thalamic infarction. Fortunately, the patient did not present any noticeable symptoms associated with the infarction and is now transitioning to convalescent rehabilitation.

### Case 2

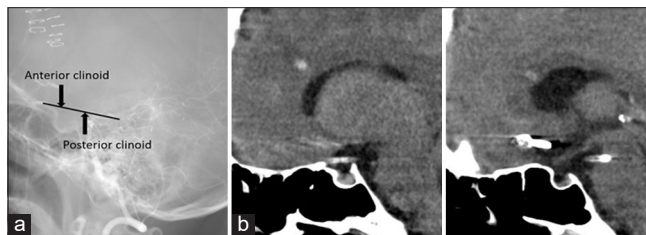
A 41-year-old woman presented with WFNS Grade 2 SAH as same as Case 1. The image findings revealed diffuse SAH [Figure 4a] because of a ruptured AcomA aneurysm, which was high-positioned relative to the tuberculum sellae, and somewhat posteriorly projected [Figure 4b]. Thus, we performed clipping through an anterior interhemispheric approach, and then inserted the same cisternal drain as Case 1 between the right optic nerve and right carotid artery by a subfrontal route in the same manner as Case 1. On day



**Figure 1:** Preoperative (a) plain computed tomography (CT) and (b) CT angiography of case 1.

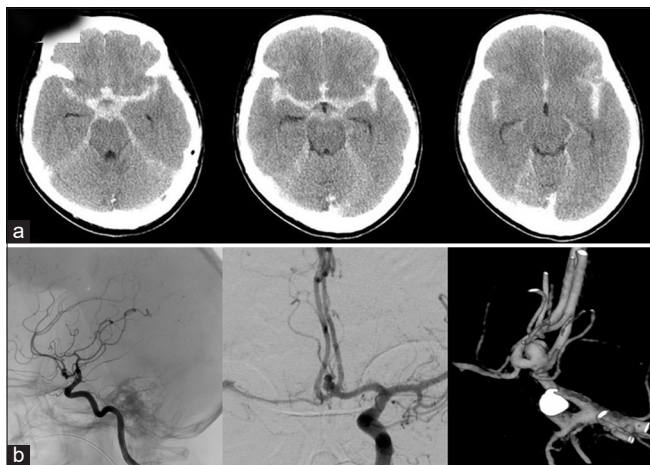


**Figure 2:** Postoperative (a) plain computed tomography and (b) diffusion weighted-magnetic resonance imaging of case 1.

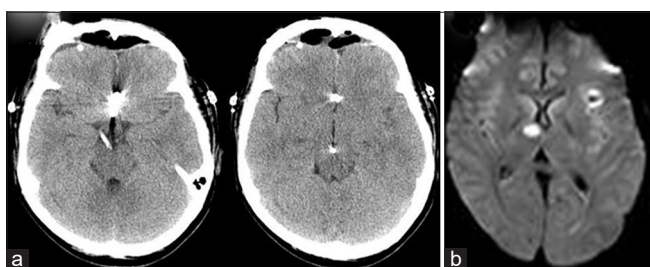


**Figure 3:** Postoperative (a) digital subtraction angiography and (b) plain computed tomography (sagittal image) of case 1.

1, MRI demonstrated acute ischemia in the right anterior-medial thalamus [Figure 5]. By reviewing the preoperative lateral VAG in DSA, the location of the basilar bifurcation was estimated as below the anterior clinoid-posterior clinoid line [Figure 6]. The tip of the cisternal drain was estimated to penetrate just above the basilar top on the right, thus potentially disturbing the right P1 perforator. On day 4, postoperative intermittent Urokinase injection and cerebrospinal fluid drainage almost completely removed the cisternal clot [Figure 5]. Her postoperative course was uneventful without spasm, and she was discharged with a modified Rankin scale of 0. Postoperative 14 months T2-weighted MRI demonstrated only a tiny residual chronic ischemic lesion in the corresponding right thalamic area [Figure 7].



**Figure 4:** Preoperative (a) plain computed tomography and (b) digital subtraction angiography of case 2.

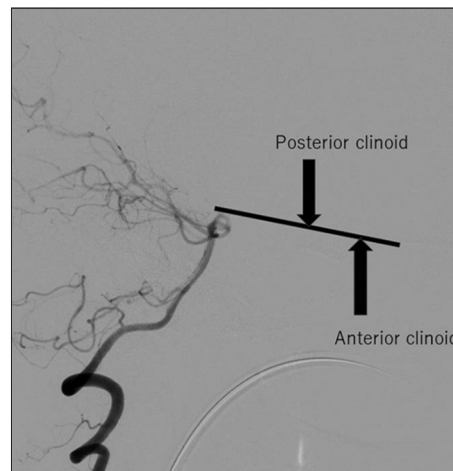


**Figure 5:** Postoperative (a) plain computed tomography and (b) diffusion weighted-magnetic resonance imaging of case 2.

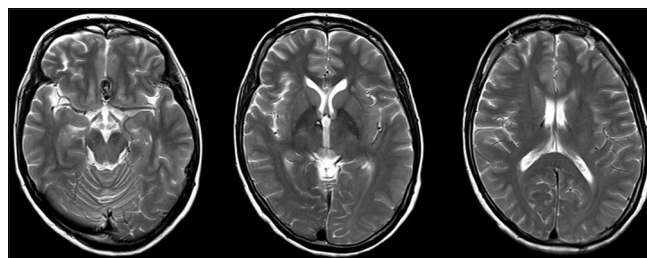
## DISCUSSION

Cisternal drainage has great value in the management of SAH, despite the risks of meningitis and delayed hydrocephalus. Indeed, in our two cases, cisternal drainage allowed rapid removal of the subarachnoid clots. Nevertheless, our cases show the potential for direct vascular injury following cisternal drain placement. Horiuchi *et al.* reported medial branch injury of the basilar artery caused by cisternal drain placement.<sup>[9]</sup> However, to the best of our knowledge, there are no other equivalent studies, and no prior evidence of thalamic infarction.

In our two cases, the sites of infarction involved the paramedian thalamic region, and we suspected perforator injury arising from the P1 segment of the posterior cerebral artery. Pedroza *et al.* classified P1 perforator injury into that involving the paramedian thalamic artery, superior paramedian mesencephalic artery, and inferior paramedian mesencephalic artery locations.<sup>[13]</sup> Thus, the infarction region in our two cases corresponded to the dominant area of the paramedian thalamic artery. This artery passes through the posterior perforating substance occupying the anterior one-third of the interpeduncular fossa and has a thickness of  $0.57 \pm 0.11$  mm.<sup>[13]</sup> In our two cases, the cisternal drains



**Figure 6:** Postoperative digital subtraction angiography of case 2.



**Figure 7:** Postoperative 14 months T2-weighted magnetic resonance imaging of case 2.

were inserted slightly too deep, and the tip was placed at the interpeduncular fossa. Thus, the paramedian thalamic artery may have been obstructed by direct injury or vessel kinking, resulting in thalamic infarction.

Cisternal drains are generally placed from the opticocarotid or carotidtentorial (retrocarotid) space along the clinoid line connecting the anterior and posterior processes, as in our two cases [Figure 3 and Figure 6]. A basilar bifurcation usually exists at the cranial side of the dorsum sellae.<sup>[3,8,18]</sup> Thus, the P1 perforator arising from the superior surface of the basilar bifurcation is typically not injured, even with deep cisternal drain insertion. However, in our two cases, the P1 perforator ran just behind the dorsum sellae because of anatomical variation of the lowered basilar bifurcation and may have been disturbed by the cisternal drain insertion. In a previous study examining the location of the basilar bifurcation with high-resolution CT in 126 patients, a lowered basilar bifurcation (lower than the dorsum sellae) was seen in 45 (35.7%) patients.<sup>[15]</sup> Thus, in these cases, the potential for P1 perforator injury by deep insertion of the cisternal drain should be considered. The senior author experienced total of 30 cases of open clipping cases by anterior interhemispheric approach for the SAH due to AcomA aneurysm so far and encountered this complication in 2 cases (6.7%) of them.



The premammillary artery, which is a perforating branch of posterior communicating artery (PcomA), also supplies the anterior medial thalamus. This artery normally branches from the anterior half of PcomA, which is close to the internal carotid artery, and runs outward. Therefore, there is little risk of injury unless the drain is inserted quite laterally toward the dorsal side of the internal carotid artery.

The infarctions in our two cases did not cause severe prognostic symptoms because they involved the unilateral medial thalamus. Nevertheless, the paramedian thalamic artery has a varied branching pattern, and originates from the unilateral P1 segment and terminates in the bilateral medial thalamus in 50% of cases.<sup>[2]</sup> In such cases, bilateral thalamic infarction may occur following unilateral paramedian thalamic artery occlusion, causing severe complications. The medial thalamus has two nucleus groups — the nuclei of midline and the dorsomedial nucleus (DM) — and is almost occupied by the DM. The presence of three frontal lobe circuits was previously reported, which all involve the DM.<sup>[1]</sup> The dorsolateral circuit is considered responsible for executive function including composition, intention, and attention. The orbitofrontal circuit is thought to mediate socially-appropriate behavior and empathy, and injury to this region can cause impulsiveness, explosiveness, dysthymia, and loss of sensitivity. The frontocingulate circuit generates motivation by maintaining the balance between inhibitory input to the supplemental motor cortex and arousal maintenance stimulus, and when damaged can result in mutism, apathy, and lack of spontaneity. These symptoms may not appear in cases of unilateral injury, but are unavoidable in bilateral injury.<sup>[4]</sup>

## CONCLUSION

To avoid the potential for thalamic injury, neurosurgeons should consider the potential for P1 perforator injury related to cisternal drain placement. Thus, it is important to confirm the position of the basilar bifurcation preoperatively, and to not insert the drain too deeply.

## Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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