Contralateral Acute Subdural Hematoma Following Traumatic Acute Subdural Hematoma Evacuation

—Case Report—

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Abstract

Contralateral acute subdural hematoma (ASDH) occurring after removal of traumatic ASDH is a rare, but nearly devastating postoperative complication. We treated a 26-year-old male who developed a contralateral ASDH shortly after craniectomy for evacuation of a traumatic ASDH. Burr-hole craniotomy was performed before decompressive craniectomy, and the bleeding source was a cortex artery within the frontal lobe contusion. Despite supportive therapy with barbiturate and mild hypothermia he expired 3 days later of brain death. Literature review suggests that the old are more susceptible to contralateral ASDH following evacuation of traumatic ASDH. Contralateral ASDH following evacuation of traumatic ASDH is a rare but potentially lethal complication, so neurosurgeons should try to detect such contralateral hematoma formation and prevent clinical deterioration.

Key words: acute subdural hematoma, decompressive craniectomy, postoperative hemorrhage

Introduction

Patients with acute subdural hematoma (ASDH) suffer high mortality rates, despite modern surgical, medical, and technological advances.^{14,15,19} Decompressive craniectomy in the management of patients with ASDH have been justified by various reports,^{3,8,20} but complications secondary to surgical decompression are likely to adversely affect outcome.^{9,16,20} Contralateral ASDH occurring after removal of traumatic ASDH have been reported recently as a rare but potentially lethal postoperative complication which requires consecutive operations. Delayed contralateral ASDH may manifest as intraoperative brain swelling, postoperative neurological deterioration, or intractably elevated intracranial pressure (ICP). If unrecognized, this disorder can cause devastating consequences. We report a case of contralateral ASDH occurring after hematoma evacuation in a patient with traumatic ASDH, and discuss this rare phenomenon.

Case Report

A 26-year-old male suffered severe left temporo-occipital brain injury in a car accident, and was admitted to our hospital with progressive headache and irritability. On admission, clinical examination demonstrated poor orientation in time and place, pupils of equal size with normal light response, Glasgow Coma Scale (GCS) score of 14, and

a left temporo-occipital scalp hematoma. Specific hematological investigations, liver function tests, and coagulation profile were all within the normal ranges. In particular, the platelet count was demonstrated as 316/mm³ (normal 100–300/mm³), whereas coagulation studies revealed prothrombin time (PT) of 10.4 sec (normal 10.0–13.5 sec), activated partial thromboplastin time of 29.5 sec (normal 22.0–36.0 sec), and thrombin time of 17.2 sec (normal 14.5–21.5 sec). Tests found D-dimer of 405 µg/l (normal 0–500 µg/l), fibrinogen of 3.4 g/l (normal 2.0–4.0 g/l), and PT-international normalized ratio (INR) of 0.95 (normal 0.80–1.15). Furthermore, the patient was not receiving any antiplatelet or anticoagulant agents, and had no family history of bleeding diathesis. His medical history was free of severe systematic disease. Emergent brain computed tomography (CT) revealed a right frontal small contusion and subarachnoid hemorrhage without midline shift (Fig. 1A, D). Mannitol infusion to control the increased ICP was not directly recommended, and observations were performed carefully. However, his level of consciousness decreased rapidly to GCS of 8 within half an hour, and his left and right pupils were 3 mm and 5 mm in diameter without light response. Primary resuscitation and stabilization therapy were performed. Repeat brain CT demonstrated a right fronto-temporal ASDH (4 mm maximum width) and a left frontal small contusion associated with severe brain swelling and midline shift of 6 mm (Fig. 1B, E).

Burr-hole craniotomy and dural incision in the right temporal region followed by decompressive craniectomy

and hematoma evacuation were performed within 3 hours of the accident. The brain showed considerable swelling and dural grafting was used. Postoperative CT revealed contralateral fronto-temporo-parietal ASDH (10 mm maximum width) with a midline shift of 15 mm (Fig. 1C, F). The patient was rapidly returned to the operating room within 5 minutes. Burr-hole craniotomy was also performed rapidly before decompressive craniectomy, and the bleeding source was a cortex artery within the frontal lobe contusion. Fresh frozen plasma was administered in view of resuscitation and blood volume loss. The intraoperative check coagulation profile did not demonstrate trauma-in-

duced coagulopathy except for a slight increase of fibrinogen. After surgery, the pupils were 5 mm in diameter respectively, and unresponsive to light. Despite supportive therapy with barbiturate and mild hypothermia, postoperative ICP remained about 21 mmHg, and cerebral perfusion pressure was around 68 mmHg. He expired 3 days later of brain death.

Discussion

Decompressive craniectomy is an effective and favorable technique for rapidly reducing ICP in patients with ASDH,²⁰ but decompressive craniectomy may release the tamponade effect and result in brain shift which exposes these patients to the risk of complications caused by developing distant intracranial hemorrhage.^{4,9,16,18,20} Contralateral epidural and intraparenchymal hematoma have received considerable attention in the literature,^{1,11,16} but contralateral ASDH is worthy of more attention. This rare complication may lead to potentially life-threatening disorders, if unrecognized. We reviewed the literature to identify cases of contralateral ASDH occurring immediately after evacuation of traumatic ASDH, as summarized in Table 1.^{1,4,9,18} The present case was 1 of 126 cases (0.8%) treated at our facility for ASDH.

The pathophysiologic mechanisms of contralateral ASDH occurring after ASDH evacuation are not clear. There is probably no single etiologic factor. Sudden increase in cerebral blood flow (CBF) with defective autoregulation, damage to cerebral vasculature secondary to perioperative parenchymal shift, previously undetected contusion, and bleeding secondary to decompression and coagulopathy have all been proposed as causative factors of delayed intracranial hematomas.^{6,10,12,17} Reviewing the reported cases, the average age was 52 years, half of the patients were older than 60 years, and two patients were older than 80 years, indicating that the old are more susceptible to contralateral ASDH than the young. In our

Fig. 1 A, D: Axial computed tomography (CT) scans on admission showing a right frontal small contusion and subarachnoid hemorrhage without midline shift. B, E: Preoperative CT scans revealing a 4-mm thick, right fronto-temporal acute subdural hematoma (ASDH) and a left frontal small contusion associated with severe brain swelling and midline shift. C, F: Postoperative CT scans revealing a 10-mm thick, contralateral ASDH with midline shift and mass effect.

Table 1 Characteristics of reported cases of contralateral acute subdural hematoma following traumatic acute subdural hematoma evacuation

Case No.	Author (Year)	Age (yrs)/ Sex	Injury mechanism	Preoperative GCS score	Pupils	Brain shift* (mm)	Brain swelling	Coagulopathy	Time to surgery (hrs)		GOS
									First	Second	
1	Ban et al. (1991) ¹⁾	62/F	traffic	7	bi-mydriasis	ND	yes	no	3	IM	D
2		72/M	traffic	5	bi-mydriasis	ND	yes	no	0.5	IM	D
3	Matsuno et al. (2003) ⁹⁾	31/M	fall	6	bi-mydriasis	24	yes	no	1	IM	SD
4		40/M	traffic	3	bi-mydriasis	10.3	yes	no	3	IM	SD
5		19/M	fall	4	uni-mydriasis	29	yes	no	5	IM	VS
6	Tomycz et al. (2010) ¹⁸⁾	81/F	fall	15	normal	23	no	yes	ND	IM	GR
7	Fridley et al. (2011) ⁴⁾	85/F	fall	15	normal	16	no	no	ND	IM	GR
8	Present case	26/M	traffic	8	bi-mydriasis	21	yes	no	3	IM	D

*Together with bilateral brain shift. bi-mydriasis: bilateral mydriasis, D: dead, F: female, GCS: Glasgow Coma Scale, GOS: Glasgow Outcome Scale, GR: good recovery, IM: immediate, M: male, ND: not described, SD: severe disability, uni-mydriasis: unilateral mydriasis, VS: vegetative state.

opinion, this may be explained by the fact that the brain parenchyma severely compressed by large hematoma had lost structural elasticity, that is, significant re-expansion of brain did not occur immediately after operation due to increasing brain atrophy and subdural space with age, caused mechanical parenchymal shift and subdural space detachment from the cerebral parenchyma opposite to the craniectomy, so the contralateral bridging veins consequently ruptured and bled resulting in ASDH formation. An 81-year-old woman with coagulopathy of INR of 4.2 developed a contralateral ASDH with midline shift shortly after craniotomy for evacuation of a traumatic right ASDH.¹⁸⁾ In addition, older patients have dura that is more adherent to the skull inner table,^{4,13)} which increases the effects of vacuum pressure and may predispose older patients to tear bridging veins after hematoma evacuation. In our case, bleeding from a cortex artery in the frontal lobe contusion was demonstrated during the second operation, which caused hemorrhage into the contralateral contusion secondary to sudden increase in CBF associated with defective autoregulation or tamponade effect removal, and contributed to the formation of ASDH. These are the most likely causative factors in young patients.

Intraoperative brain swelling was found after evacuation of traumatic ASDH in 6 of the 8 reported cases (75%) with poor prognosis. Intraoperative brain swelling is a warning sign of contralateral ASDH formation in traumatic patients,¹⁶⁾ so neurosurgeons should try to detect the development of contralateral hematoma and prevent clinical deterioration, particularly in the absence of clinical evidence of brain swelling. Postoperative routine CT is a reliable way to evaluate this potential complication for a comprehensive strategy of prompt treatment.^{4,16)} This need is demonstrated clearly by two cases with the development of contralateral hematoma without intraoperative brain swelling. Any unexplained increase in ICP warrants immediate CT, which is the best choice to establish the diagnosis for intervention for this evolving pathology. Contralateral hematomas can be easily and precisely identified using intraoperative sonography, which could lead to early amelioration of the life-threatening mass effect caused by contralateral expansion,^{1,7)} but comparison with CT findings is necessary to prospectively evaluate the efficiency of intraoperative sonography.

How can we prevent this devastating complication? We suggest that early and gradual decompression could prevent the sudden brain shift and decreased tamponade effect. Decompression with burr-hole craniotomy rapidly followed by decompressive craniectomy may help to decrease the risk of contralateral hemorrhage in our experience, but there are several other favorable measures as follows. Dural netted incision to prevent severe brain swelling involves incision of the dura in a netted shape to evacuate hematoma and reduce brain shift.⁵⁾ Dural incision step by step to remove the hematoma so as to minimize the changes in pressure. Correct head position to aggravate brain shift, especially in the presence of no significant re-expansion of brain tissue.¹²⁾

In conclusion, contralateral ASDH following evacua-

tion of traumatic ASDH is a rare but potentially lethal complication, so neurosurgeons should try to detect such contralateral hematoma formation and prevent clinical deterioration. Various measures may prevent this devastating complication. More experience is needed for further understanding of the pathophysiology and treatment.

Conflicts of Interest Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices in the article.

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