

Immediate Postoperative Epidural Hematomas Adjacent to the Craniotomy Site

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Objective : The authors present eight cases of immediate post-operative epidural hematomas(EDHs) adjacent to the craniotomy site, describe clinical details of them, and discuss their pathogenesis.

Methods : Medical records of eight cases were retrospectively reviewed and their clinical data, operation records, and radiological findings analyzed. Any risk factors of the EDHs were searched.

Results : In 5 of 8 cases, adjacent EDHs developed after craniotomies for the surgical removal of brain tumors. Three cases of adjacent EDHs developed after a pterional approach and neck clipping of a ruptured anterior communicating artery aneurysm, a ventriculoperitoneal shunt, and a craniotomy for a post-traumatic EDH, respectively. In all eight cases, brain computed tomography (CT) scans checked immediately or a few hours after the surgery, revealed large EDHs adjacent to the previous craniotomy site, but there was no EDH beneath the previous craniotomy flap. After emergent surgical removal of the EDHs, 7 cases demonstrated good clinical outcomes, with one case yielding a poor result.

Conclusion : Rapid drainage of a large volume of cerebrospinal fluid or intra-operative severe brain collapse may separate the dura from the calvarium and cause postoperative EDH adjacent to the previous craniotomy site. A high-pressure suction drain left in the epidural space may contribute to the pathogenesis. After the craniotomy for brain tumors or intracranial aneurysms, when remarkable brain collapse occurs, an immediate postoperative brain CT is mandatory to detect and adequately manage such unexpected events as adjacent EDHs.

KEY WORDS : Postoperative hematomas · Adjacent epidural hematoma.

Introduction

The computed tomography(CT) scan has played a significant role in the early detection and adequate management of post-craniotomy intracranial hematomas⁷⁾. However, postoperative epidural hematomas(EDHs) still remain an embarrassing complication in neurosurgical practice. Fukamachi and coworkers reported that the incidence of postoperative EDHs was 1.0%⁴⁾.

Postoperative EDHs can develop regionally, adjacent to, or distant from the operative locus⁴⁾. There have been some reports of distant EDHs^{4,10,14)}. Adjacent EDHs, however, have rarely been described in detail in the literature^{4,12)}.

The authors present eight cases of immediate postoperative EDH adjacent to the previous craniotomy site, discuss their

mechanisms of development and suggest methods to prevent them.

Materials and Methods

Postoperative EDHs were classified into regional, adjacent, and distant types according to their relationship to the craniotomy site. In adjacent EDH, the hematoma was located outside the craniotomy or burr hole site, but in contact with the edge of the previous craniotomy⁴⁾.

The authors collected eight cases of post-craniotomy adjacent EDHs they had managed between March 2002 and April 2004. There were 4 men and 4 women, ranging from 15 to 66 years of age. Their clinical data, operation records, and radiological findings were reviewed and possible risk factors analyzed. The

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Table 1. Summarized clinical data of 8 patients with post-craniotomy adjacent epidural hematomas

Case	Age/ sex	Underlying pathology	Site of craniotomy	Operative procedure	Site of EDH	Systemic HTN	Bleeding tendency	Ventricular enlargement	GOS
1	19/F	Low grade astrocytoma Rt. LV	Rt. P	GTR of tumor	Rt. FTP	-	-	+	5
2	34/M	Central neurocytoma Lt. LV	Lt. F	GTR of tumor	Lt. TP	-	-	+	5
3	42/F	Meningioma Rt. F convexity	Rt. F	GTR of tumor	Rt. FTO/ Lt. O	-	-*	-	5
4	35/F	ACoA Aneurysm	Lt. FT	Neck clipping	Lt. TP	-	-	-	5
5	61/F	Meningioma Lt TP convexity	Lt. FTP	GTR of tumor	Lt. PO	-	-	-	5
6	66/M	Hydrocephalus	Rt. PO	VP shunt	Rt. TP	-	-	+	2
7	45/M	Meningioma Lt. sphenoid wing	Lt. FTP	GTR of tumor	Lt. P	-	-	-	5
8	15/M	Post-traumatic EDH Rt. F	Rt. F	ROH	Rt. TP	-	-*	-	5

EDH : epidural hematoma, HTN : hypertension, GOS : Glasgow outcome scale, M : male, F : female, Rt : right, Lt : left, LV : lateral ventricle, ACoA : anterior communicating artery, GTR : gross total removal, VP : ventriculoperitoneal, ROH : removal of hematoma, F : frontal, FT : frontotemporal, FTP : frontotemporoparietal, O : occipital, P : parietal, PO : parieto-occipital, TP : temporo-parietal, * : postoperative INR high (1.56 in case 3 & 1.41 in case 8, respectively)

Table 2. Blood coagulation tests

Case	Prothrombin time (10.8~13.9 sec)	Prothrombin percent(68~120%)	Prothrombin INR(0.91~1.25)	a PTT (24~38 second)
	Pre-op / post-op	ppre-op / post-op	pre-op / post-op	pre-op / post-op
1	11.5 / 13.6	101.7 / 71.3	0.99 / 1.18	25.1 / 27.7
2	11.0 / 12.7	112.1 / 81.9	0.95 / 1.10	27.9 / 24.8
3	11.7 / 11.8	98.9 / 49.1	1.01 / 1.56	27.2 / 49.4
4	10.4 / 13.1	116.1 / 80.1	0.92 / 1.13	22.5 / 26.4
5	11.3 / 10.5	110 / 108	0.96 / 0.96	25.2 / 28.7
6	11.8 / 12.1	95 / 89	1.03 / 1.06	33.8 / 37.5
7	11.7 / 13.9	97.8 / 70.1	1.01 / 1.20	26.2 / 25.6
8	13.3 / 16.4	77.4 / 55.6	1.14 / 1.41	27.5 / 34.0

INR : international normalized ratio, aPTT : activated partial thromboplastin time, pre-op : preoperative, post-op : postoperative

clinical outcome was evaluated at the time of discharge using the Glasgow Outcome Scale.

Results

In 5 of 8 cases, adjacent EDHs developed after craniotomies for the surgical removal of brain tumors (a low grade astrocytoma, a central neurocytoma, and 3 meningiomas). Three cases developed after a pterional approach and neck clipping of a ruptured ACoA aneurysm, a ventriculoperitoneal(VP) shunt, and a craniotomy for a post-traumatic EDH, respectively. Their clinical data are summarized in Table 1. No case had systemic hypertension or preoperative coagulopathy.

Three patients had precraniotomy ventricular enlargement (cases 1, 2, 6) and one of them (case 6) underwent extraventricular drainage(EVD) of CSF. In case 4, about 30cc of CSF was removed

through a lumbar drainage catheter during the operation for the ACoA aneurysm. A remarkable elevation of the systemic blood pressure during recovery from anesthesia or immediate postoperative period was not seen in any of the patients.

In six patients, the prothrombin time(PT), the activated partial thromboplastin time(aPTT), and international normalized ratio(INR) were within normal range during the preoperative and postoperative periods. In two patients (cases 3 and 8), postoperative INR and PT were prolonged and normalized after the transfusion of fresh frozen plasma (Table 2).

Brain CT scans were checked immediately after the surgery in 6 patients, 2 hours after the surgery in case 4, and 5 hours after the surgery in case 3. Disturbance of consciousness developed in all patients when EDHs were diagnosed with brain CT scans. In all patients, EDHs were large and exerted such a severe mass effect that emergent surgical removal was mandatory (Fig. 1, 2, 3). The hematomas were mixed density on the brain CT.

In all cases, the EDHs were located at the site adjacent to and lower than the previous craniotomy site (at dependent areas). However, there

was no significant EDH beneath the previous craniotomy bone flap. For removal of the EDHs, the previous craniotomies were extended. No definite bleeding foci, except for oozing from the dura, were found in seven cases. A pin of the head rest perforated the calvarium and probably caused the EDH in case 7.

After the surgery, case 2 showed transient motor dysphasia and right-sided hemiparesis, resulting in mild slurred speech and memory disturbance on discharge. Case 6 underwent three operations for recurrent hematomas, and his outcome was poor. All other cases demonstrated good recovery.

Illustrative Cases

Case 1

A 19 year-old girl complained of sudden headache on the day of admission. She showed nystagmus on her left eye, and no other

deficits were evident upon neurologic examination. A brain magnetic resonance image(MRI) revealed a large mass lesion medial to the atrium of the right lateral ventricle with strong contrast enhancement and cystic components (Fig. 1A). With the patient placed in the prone position and her head fixed with a Mayfield headrest, a medial posterior parietal craniotomy was performed. Prior to dural opening, dural tack-up sutures were made along the margin of the craniotomy. After the gross total removal of the

tumor, the brain was remarkably sunken down. Bleeding from the dura mater and the bone was meticulously controlled. Two tack-up sutures were made to attach the dura closely to the replaced bone flap. An epidural Hemovac[®] drain was then placed.

A routine immediate post-operative CT scan showed a large right frontotemporoparietal mixed density EDH of 3.5-cm maximum thickness (Fig. 1B). Her right pupil was dilated and non-reactive. An emergency right-sided frontotemporoparietal craniotomy was performed. When a burr hole was trephined at the temporal squama, a large amount of liquid hematoma gushed out. The EDH was located anterior to the previous craniotomy site, and there was no EDH at all under the previous craniotomy flap. The hematoma was totally evacuated (Fig. 1C). No definite bleeding source could be found.

The histologic diagnosis of the tumor was a low-grade astrocytoma with gemistocytic components. Twenty days after the surgery, the patient was discharged without any neurologic deficits.

Case 2

A 34-year-old man was admitted due to headache, nausea, and vomiting that had started 7 days earlier. He showed no neurologic deficits upon neurologic examination. A brain MRI revealed enlarged lateral ventricles and an approximately 4 × 2.5cm-sized mixed signal intensity mass lesion in the frontal horn and body of the left lateral ventricle (Fig. 2A). After the patient was placed in the supine position, a left frontal craniotomy was performed. Gross total removal of the tumor was achieved through a transcortical approach.

In the immediate postoperative period, his post-anesthetic recovery was delayed. A brain CT showed a large mixed density EDH of 3-cm maximum thickness (Fig. 2B); the hematoma was located at the left temporoparietal convexity posterior

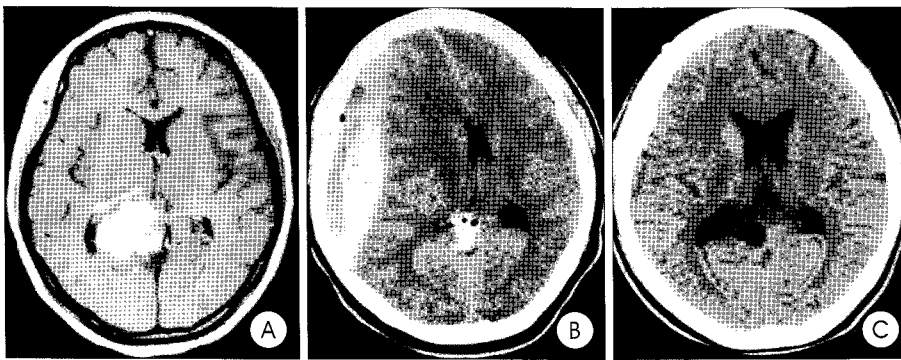


Fig. 1. A : Preoperative brain magnetic resonance image shows a large mass lesion medial to the atrium of the right lateral ventricle with strong contrast enhancement and cystic components. B : Immediate routine postoperative brain computed tomography scan shows a large right frontotemporoparietal epidural hematoma with mixed density. C : Postoperative brain computed tomography scan shows no residual hematoma.

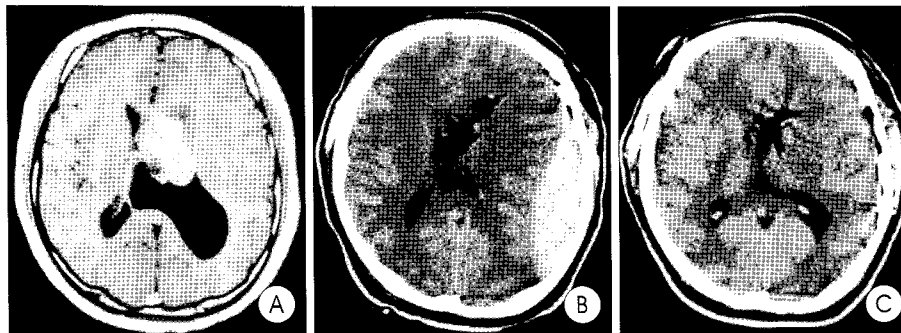


Fig. 2. A : Preoperative brain magnetic resonance image shows a large mass lesion in the frontal horn and body of the left lateral ventricle. B : Immediate routine postoperative brain computed tomography scan shows a large left temporoparietal mixed density epidural hematoma. C : Postoperative brain computed tomography scan shows no residual hematoma.

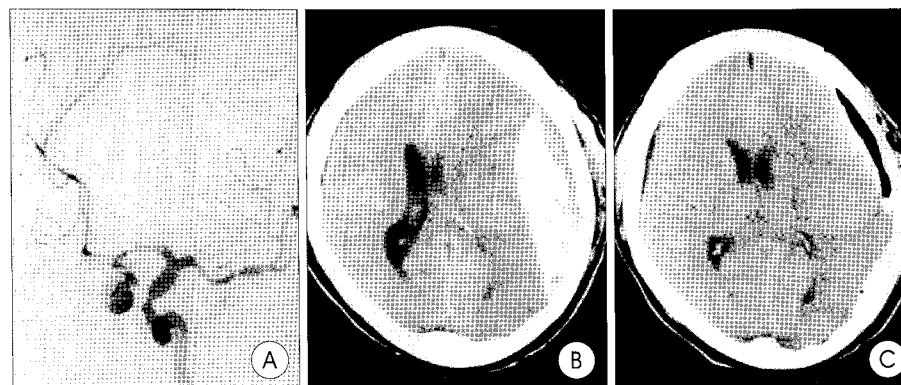


Fig. 3. A : Digital subtraction cerebral angiography shows an anteriorly directing anterior communication artery aneurysm. B : Immediate routine postoperative brain computed tomography scan shows a large left temporoparietal epidural hematoma with mixed density. C : Postoperative brain computed tomography scan shows some residual epidural hematoma.

to the previous craniotomy site. In the emergent re-operation, there was no EDH beneath the previous craniotomy flap. As the left temporoparietal craniotomy was made, a large volume of liquid hematoma gushed out. No causative bleeding source could be found. The EDH was totally evacuated (Fig. 2C).

After the surgery, the patient showed transient motor dysphasia and right-sided hemiparesis. The tumor was diagnosed as a central neurocytoma on histologic examination. He was discharged on post-operative day 27 with residual mild slurred speech and recent memory disturbance.

Case 3

A 35-year-old woman was admitted with severe headache that had developed after falling down on the day of admission. She showed no neurologic deficits, but her brain CT revealed a diffuse high density subdural hematoma (SDH) at the right temporoparietal convexity. The SDH had disappeared on a follow-up brain CT after conservative management.

The cerebral angiography checked on hospital day 21 demonstrated an anteroinferiorly directing anterior communicating artery (ACoA) aneurysm (Fig. 3A). With the patient placed in the supine position and her head fixed with a Mayfield head rest, a left pterional craniotomy was performed. After the dural opening, about 30cc of cerebrospinal fluid (CSF) was removed through a lumbar drain. The aneurysm was exposed by the usual pterional approach, and its neck was clipped uneventfully. Meticulous hemostasis was performed.

Two hours after the surgery, the patient was still stuporous and her left pupil was dilated. A brain CT showed a large mixed density EDH of 3-cm maximum thickness (Fig. 3B); the hematoma was located at the left temporoparietal convexity posterior to the previous craniotomy site without any EDH beneath the previous craniotomy flap. An emergent re-operation was performed to remove the adjacent EDH (Fig. 3C). The patient was discharged on postoperative day 17 with mild mental confusion.

Discussion

Postoperative intracranial hematomas are occasional urgent complications following craniotomies for various neurosurgical procedures^{4,8,12}. CT scan has made it easy to detect them⁷. The overall incidence of post-craniotomy intracranial hematomas that needed surgical evacuation was reported as 1.1%, and the hematomas were intraparenchymal in 43%, subdural in 5%, and extradural in 33%⁸. Fukamachi et al. reported that the incidence of postoperative EDHs was 1.0%, and 10 of the 16 hematomas were operated upon (0.9%)⁴. There have been reports of postoperative epidural hematomas after the CSF shunt¹⁴, the extraventricular drainage of CSF^{6,9} and the ventriculography¹.

Post-craniotomy epidural hematomas can be classified as regional, adjacent, or distant according to their relationship to the previous craniotomy site⁴. There are reports describing epidural hematomas that are remote from the craniotomy site^{4,10,14}. Wolfsberger reported one case of multiple supratentorial EDHs after posterior fossa surgery¹⁴. However, EDHs that develop adjacent to the previous craniotomy site have rarely been described in detail in the literature^{4,12}.

It is curious why the EDH developed adjacent to the craniotomy site while there was no hematoma beneath the previous craniotomy flap. Pathogenetic mechanisms of the post-craniotomy adjacent EDHs have been discussed in the literature. Sudden lowering of intracranial pressure (ICP) or rapid drainage of ventricular CSF result in brain collapse. This may exert a suction force on the structures between the dura and the calvarium^{4,12}. Losing support of the brain, the dura mater contracts by its own elasticity and is separated from the skull⁴. Detaching the collagenous fixations of the dura from the inner table of the skull may initially cause dural and diploic veins to bleed into the epidural space. As the hematoma enlarges and the distance between the dura and bony arterial channels increases, dural arteries may also tear^{3,9}. Collecting blood further promotes dural separation, and forms a larger EDH. Finally, the EDH may extend downward from the craniotomy site by gravity to the dependant area.

Yacubian et al., explained that not only mechanical shifting of the brain resulting from CSF drainage but also replacement of the brain to its normal position rectifying the disturbed venous circulation may cause hemorrhage¹⁵. Haft et al. suggested that the upright position on the operating table may promote stripping of the dura mater from the skull⁶. Thiagarajah suggested that hypertension can cause hemorrhage at the operative site by disrupting hemostasis and autoregulation, and by damaging the blood-brain barrier¹¹. Gerlach et al. reported that decreased Factor XIII activity was associated with the increased risk of postoperative hemorrhage after intracranial surgery⁵.

The dural separation from the calvarial skull due to the abrupt brain collapse is thought to have contributed to the development of adjacent EDHs in the 7 patients of this report. Preoperative ventricular dilatation was seen in 3 of them. In another one, a VP shunt for hydrocephalus was complicated by recurrent adjacent EDHs. Accidental perforation of the cranial vault by a pin of the head rest caused an adjacent EDH in case 7. Although postoperative coagulopathy was found in cases 3 and 8, their INR was normalized after a transfusion of FFP. It is uncertain whether their increased INR played any causative role in the EDH formation. A factor XIII assay was not performed in our cases. All hematomas were located at the dependant areas and were mostly comprised of a fluid component. This suggests that the epidural blood clots extended downward

by gravity. Dandy introduced dural tenting sutures in the early 1930s²⁾. Dural tenting helps hemostasis by attaching the dura tightly against the overlying craniotomy flap and by occluding the dural vessels at the margin of the craniotomy. Winston however, suggested that dural tenting has risks, albeit small, of causing extradural, subdural, and subpial bleeding, and of concealing epidural bleeding at the time of operative wound closure¹³⁾. According to Fukamachi et al., they encountered postoperative EDHs, although they had performed tight dural tenting sutures⁴⁾. In spite of dural tenting, the authors think, bleeding from the craniotomy margin proceeded between the dura and the calvarium, forming adjacent EDHs.

We routinely used epidural suction drain (Hemovac[®]) in most craniotomies. The negative pressure of the Hemovac[®] varies between 85 to 95mmHg. Strong negative pressure suction through the closed drainage system may be a factor that promoted stripping of the dura mater and tearing of epidural vessels at areas adjacent to the craniotomy sites. Interestingly, although such large EDHs were located adjacent to the craniotomy site, there was no or only a scanty hematoma under the actual craniotomy flap. Dural tack-up sutures along the margin and at the center of the craniotomy flap as well as the epidural suction drain most probably prevented EDH collection beneath the craniotomy flaps in our cases. For the prevention of the adjacent EDH, the authors suggest the dural separation under the craniotomy edge be as narrow as possible at the time of the craniotomy. Abrupt brain collapse should be avoided, particularly in patients with pre-operative ventricular enlargement. It is recommended to fill the subdural space with normal saline to prevent remarkable brain displacement and the development of EDHs. The authors advise avoiding too strong negative pressure when an epidural suction drains are used. The authors also suggest that immediate post-craniotomy brain CT scanning be performed routinely. In our cases, brain CT scans were checked immediately or within a few hours after craniotomies. The unanticipated EDHs were diagnosed and managed without much delay and most patients showed good results.

Conclusion

Intra-operative rapid drainage of ventricular CSF or severe brain collapse can cause postoperative EDH adjacent to the previous craniotomy site. Accidental perforation of the skull by a head rest pin can also cause postoperative EDH. Excessively strong negative pressure of the epidural suction drain may contribute to the development of the adjacent EDH. After the craniotomies for brain tumors or intracranial aneurysms, when remarkable brain collapse occurs, immediate postoperative brain CT is mandatory to detect and manage such an unexpected event as the adjacent EDH.

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Commentary

I've read an interesting article about postoperative epidural hematomas adjacent to the craniotomy site. Authors reported pathogenetic mechanisms of the post-craniotomy adjacent epidural hematomas. First, sudden lowering of intracranial pressure(ICP) or rapid drainage of ventricular CSF result in brain collapse. Second, detaching the collagenous fixation of the dura from the inner table of the skull may initially cause dural and diploic veins to bleed into the epidural space. Finally, the epidural hematomas may extend downward from the craniotomy site by gravity to the dependant area. I agree with this opinion because I experienced several similar cases and deliberated on the reason why it happened. In our opinion, I think that two factors participate in mechanism and pathogenesis of postoperative hematoma. The first is the hemodynamic factors like preop. administration of anticoagulant, HTN, effect of operative position to venous outflow. the second is mechanical factors like vessel tearing, dural detachment due to brain parenchyme displacement induced by CSF overdrainage. But I agree with the opinion that mechanical factor is more important than hemodynamic factor in the cases of postoperative epidural hematoma.

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