CASE REPORT

Delayed contralateral epidural hematoma after decompressive craniectomy for a traumatic acute subdural hematoma

Chen-Hsing Su a,b,c, Jung-Tung Liu a,b,c, Cheng-Siu Chang a,b,c, Wen-Jui Liao a,b,c, Cho-Shun Li a,b,c,*

a School of Medicine, Chung-Shan Medical University, Taichung, Taiwan
b Department of Medical Education, Chung-Shan Medical University Hospital, Taichung, Taiwan
c Department of Neurosurgery, Chung-Shan Medical University Hospital, Taichung, Taiwan

Received 18 May 2015; received in revised form 6 July 2015; accepted 27 August 2015
Available online 25 December 2015

KEYWORDS
epidural hematoma; intracranial pressure; subdural hematoma; therapeutic hypothermia

Summary After falling from a height, a 29-year-old male patient developed a traumatic left subdural hematoma (SDH) with brain swelling and a midline shift to the right side, as well as a small epidural hematoma (EDH) (thickness: <1 cm) overlying a contralateral temporal linear fracture. A decompressive craniectomy for SDH evacuation and the placement of an intracranial pressure (ICP) monitoring device were performed. Because of uncontrollable ICP (>35 mmHg) 48 hours after surgery, a left, extended decompressive craniectomy was performed in combination with therapeutic hypothermia for 6 days, including rewarming for 3 days. The patient remained stable for several days. However, the patient developed sudden right pupil dilatation with an uncal herniation on Day 14. Computed tomography revealed a considerable enlargement of the contralateral EDH. An emergency craniectomy was performed for EDH evacuation. In this paper, we describe this rare case, in which the delayed expansion of the contralateral EDH occurred 14 days after the initial surgery, and discuss its clinical management and radiologic findings, in addition to reviewing the literature and presenting the possible mechanism of this complication.

Conflicts of interest: The authors declare no conflicts of interest.
* Corresponding author. Department of Neurosurgery, Chung-Shan Medical University Hospital, 110, Section 1, Chien-Kuo North Road, Taichung, Taiwan.
E-mail addresses: cshy1350@csh.org.tw, hkli@ms28.hinet.net (C.-S. Li).

http://dx.doi.org/10.1016/j.fjs.2015.08.008
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1. Introduction

Delayed expansion of a contralateral epidural hematoma (EDH) after decompressive surgery for an acute subdural hematoma (SDH) is rare. It is a life threatening condition requiring emergency evacuation surgery. The longest period for an occurrence of the enlargement of a delayed contralateral EDH following the initial surgery is 96 hours, as reported by Su et al. Here, we report a patient who developed delayed EDH expansion 14 days after the initial surgery, an extensive period never reported in the literature according to our research.

2. Case Report

A 29-year-old male patient developed a disturbance of consciousness after falling from a 4-m height. His initial Glasgow Coma Scale score at the emergency room was E2V1M4. Brain computed tomography (CT) revealed a left SDH, a traumatic subarachnoid hemorrhage with a mass effect and a midline shift, and a small right temporal EDH (thickness: <1 cm) overlying a fracture of the right temporal bone (Figure 1). An emergency decompressive craniectomy for SDH evacuation and the placement of an intracranial pressure (ICP) monitoring device (Camino; Integra Life Sciences Corporation, Plainsboro, NJ, USA) were performed. Two days following the craniectomy, Cushing’s triad, including considerable bradycardia and hypertension, and increased ICP up to 35 mmHg were observed. A follow-up CT scan revealed considerable brain swelling on the left side with a midline shift, whereas no enlargement of the contralateral temporal EDH was observed (Figure 2). An extended decompressive craniectomy was performed in combination with therapeutic hypothermia (TH). TH was maintained at a target temperature of 33°C for 6 days, including rewarming to 36°C at a rate of 1°C/d for 3 days. ICP was approximately 15 mmHg during the TH. After regaining a normal body temperature on Day 8, repeat CT revealed residual brain swelling without a midline shift and no obvious enlargement of the right temporal EDH (Figure 3). The osmotic agents used from Day 1 were tapered accordingly, and the patient remained stable for several days. However, 5 days following the completion of TH or Day 14 after his traumatic brain injury, the patient developed sudden right pupil dilatation, and his Glasgow Coma Scale score decreased from E2V1M4 to E1V1M3. A follow-up CT scan revealed a considerable

Figure 1 Initial computed tomography showing (A) a small right temporal epidural hematoma (thickness: <1 cm), (B) left temporal subdural hematoma with a midline shift, (C) and (D) a right temporal bone fracture (arrows).
expansion of the right temporal EDH (Figure 4). An emergency craniectomy with an EDH evacuation was performed. At discharge 2 months later, the patient remained in a vegetative state.

3. Discussion

Delayed expansion of a contralateral EDH after decompressive surgery for an acute SDH is rare, with an incidence ranging from 1.3% to 5.7%. In the literature, the longest period for an occurrence of the enlargement of a delayed contralateral EDH following the initial surgery is 4 days, as reported by Su et al. Our patient developed a delayed expansion of the EDH 14 days after the initial surgery, an extensive period never reported in the literature according to our research. Intraoperative brain swelling, postoperative neurologic deterioration, and uncontrollable, elevated ICP may imply a delayed expansion of the contralateral EDH. A decompressive craniectomy may be a predisposing factor for a delayed expansion of the EDH. An immediate postoperative CT scan is recommended for patients with an acute SDH and a contralateral skull fracture to detect this rare but life threatening delayed expansion of the contralateral EDH, which has devastating consequences such as neurologic deficits and even death.

Shen et al. reviewed 37 patients with an expansion of a contralateral acute EDH following acute SDH evacuation, of whom 81% (30/37) had contralateral skull fractures and only one was aged > 60 years. The low prevalence of EDH formation among elderly patients may be attributed to

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Figure 2  Computed tomography on Day 2 after decompressive surgery showing considerable left brain swelling with a midline shift but no enlargement of the right temporal epidural hematoma.

Figure 3  Repeat computed tomography on Day 8 after rewarming showing residual brain swelling without a midline shift and no obvious enlargement of the right temporal epidural hematoma.
increased adherence between the dura and the inner table of the skull.

The precise mechanism of the delayed formation of a contralateral EDH following a decompressive craniectomy is unclear. It has been hypothesized that the initial impact causes a contrecoup injury with SDH and intracerebral hematoma formation, in addition to a coup injury with a skull fracture and bleeding from the fracture site or the dura mater. The SDH with mass effect probably increases the ICP and produces a tamponade effect on the contralateral epidural bleeding source, which subsequently induces delayed EDH enlargement after the mass effect has subsided or the SDH is removed. Therefore, measures such as decompressive surgery, the use of hyperosmolar agents, and TH are undertaken to reduce the elevated ICP, which may decrease the tamponade effect and promote delayed formation or expansion of a contralateral EDH.

In our reported case, the uncontrollable ICP was frequently >35 mmHg with considerable Cushing’s triad despite treatment with various osmotic agents. Repeat CT (Figure 2) confirmed that the intracranial hypertension was induced by brain swelling rather than by the rare but life threatening delayed expansion of the contralateral EDH. Sadaka and Veremakis et al reviewed the most recent 18 studies using TH for ICP management in patients with traumatic brain injury, concluding that ICP was always significantly lower in the TH group than in the normothermia group. We started TH as a second-line therapeutic option; the ICP was <20 mmHg, and no TH-induced coagulopathy or other side effects were observed. A decompressive craniectomy is a predisposing factor for the delayed expansion of an EDH. The prolonged use of hyperosmolar agents and TH may further reduce the ICP and gradually decrease the tamponade effect, which is attributed to the etiology of this devastating delayed expansion of the contralateral EDH. Based on our research, no other study has reported the delayed expansion of a contralateral EDH occurring up to 14 days after the initial surgery. Neurosurgeons should be aware that the rare but life threatening delayed expansion of a contralateral EDH can occur within an extensive period after the initial surgery. Additional studies should be conducted to determine whether contralateral EDHs should be managed during the initial surgery.

References