Delayed Brainstem Hemorrhage Secondary to Mild Traumatic Head Injury: Report of Case with Good Recovery

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Key words
- Duret hemorrhage
- Mild traumatic head injury
- Secondary traumatic brainstem hemorrhage

Abbreviations and Acronyms
CT: Computed tomography
GCS: Glasgow Coma Scale
SAH: Subarachnoid hemorrhage

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Supplementary digital content available online.
Journal homepage: www.WORLDNEUROSURGERY.org
Available online: www.sciencedirect.com
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INTRODUCTION
Traumatic brainstem hemorrhage, which occurs in 1%–10% of closed head injuries, is commonly classified as primary type and secondary type. While primary traumatic brainstem hemorrhage results from injury at the moment of impact, secondary traumatic brainstem hemorrhage occurs at a later stage after the primary head injury. In clinical practice, secondary traumatic brainstem hemorrhage often develops during descending transtentorial herniation due to raised intracranial pressure, which is known as Duret hemorrhage. 1-4 Duret hemorrhage is always considered a fatal and irreversible event. However, in rare circumstances, victims of Duret hemorrhage could gain favorable outcomes. 1,4-5 To our knowledge, secondary brainstem hemorrhage due to mild traumatic head injury without descending transtentorial herniation has never been reported. In this report, we present a case of delayed brainstem hemorrhage secondary to a relatively mild traumatic head injury that experienced a rapid and favorable recovery.

CASE DESCRIPTION: A 48-year-old man was admitted for a motorcycle accident. Head computed tomography 2 hours after the accident revealed mild subarachnoid hemorrhage at the interpeduncular cistern. In the following hospital days, he experienced 2 episodes of mental state deterioration and increase of the SAH and hematoma extension to the brainstem. A digital subtraction angiography was performed with no positive finding of vascular anomaly and evident cerebral vasospasm. He experienced a rapid and favorable recovery. His Glasgow Outcome Scale score was 5 at 3 months’ follow-up.

CONCLUSIONS: We present a rare case of secondary traumatic brainstem hemorrhage that experienced a rapid and good recovery process. The mechanism is still obscure to us and needs to be further studied. Although traumatic brainstem hemorrhage usually means a fatal event to most of the patients, some patients may experience a favorable recovery. This rare circumstance should be stressed in prognosis consultation and clinical management of these kinds of patients.

BACKGROUND: In clinical practice, secondary traumatic brainstem hemorrhage often develops during descending transtentorial herniation due to raised intracranial pressure, which is known as Duret hemorrhage. Although usually considered a fatal and irreversible event, in rare circumstances, victims of Duret hemorrhage could gain favorable outcomes. To our knowledge, secondary brainstem hemorrhage due to mild traumatic head injury without descending transtentorial herniation has never been reported. In this report, we present a case of delayed brainstem hemorrhage secondary to a relatively mild traumatic brain injury that experienced a rapid and favorable recovery.

CASE REPORT: A 48-year-old man was admitted for a motorcycle accident. He was a smoker and had abused alcohol for >20 years. Head computed tomography (CT) 2 hours after the accident revealed mild subarachnoid hemorrhage (SAH) at the interpeduncular cistern (Figure 1A). Physical examination on admission showed a Glasgow Coma Scale (GCS) score of 14 (E4, V4, M6). Nine hours after the accident, his mental state began to decline. His GCS score declined to 10 (E2, V3, M5). Head CT revealed brainstem hemorrhage adjacent to the previous SAH and increase of the primary SAH (Figure 1B). A digital subtraction angiography was performed with no positive finding of vascular anomaly and evident cerebral vasospasm. CT performed 1 day after the accident showed no extension of the brainstem hemorrhage. Two days later, his mental state gradually recovered. He could obey commands and correctly answer some easy questions until the fourth day after the accident, when his mental state declined once again. Physical examination showed a GCS score of 10 (E2, V3, M5). An emergent CT revealed further increase of the brainstem hemorrhage (Figure 1C). His mental state never deteriorated anymore, and further
CT showed no increase of the brainstem hemorrhage and SAH. He experienced a rapid and favorable recovery. His Glasgow Outcome Scale score was 5 at 3 months' follow-up.

**DISCUSSION**

As nearly all of the reported cases of secondary brainstem hemorrhage were secondary to descending transtentorial herniation, Duret hemorrhage was always considered the synonym of secondary brainstem hemorrhage. The pathogenesis of secondary brainstem hemorrhage is still obscure to us, although it has been noticed for decades. Generally speaking, there was no consensus on the origin (arterial or venous) of Duret hemorrhage. Some authors believed that stretching and disruption of the paramedian pontine perforating arteries during brainstem downward movement led to hemorrhaging. Other authors insisted that veins may be compressed more easily than arteries, and hemorrhagic transformation of an area of venous ischemia may also contribute to Duret hemorrhage. Besides, surgical decompression may promote Duret hemorrhage by way of reperfusion injury.

However, our patient represents a unique case because the hypotheses mentioned earlier could not perfectly explain the things that occurred in our case. Firstly, there was no descending transtentorial herniation and persistent vascular compression and stretching. And then reperfusion injury was not present due to decompressive craniectomy for intracranial hematoma and increased intracranial pressure. This case represents another type of secondary brainstem hemorrhage that is due to mild head injury and without the presence of brain herniation. On the basis of the progressive nature of mental state deterioration and increase of brainstem hematoma in the early stage of hospitalization, continuous injury to the local brainstem vasculature may exist. The primary injury might be due to the instantaneous stretching of brainstem vessels at the moment of upward and downward movement during motor cycle accident. The momentary injury may lead to a cascade of local vascular responses, which causes endothelial cell damage and increase of vascular permeability or even microvascular rupture. Blood components, especially red blood cells, gradually leaked to the subarachnoid space and brainstem.

Generally speaking, traumatic brainstem hemorrhage, regardless of the type of injury (primary or secondary), usually means a fatal event to most of the victims. However, traumatic brainstem hemorrhage does not readily warrant poor outcome and withdrawal of aggressive treatment. In rare circumstances, some patients might survive from secondary traumatic brainstem hemorrhage and experience a favorable recovery. In our opinion, those patients with good outcome might, in fact, have experienced a relatively focal and mild secondary brainstem injury. And nearly all of the cases of Duret hemorrhage with good outcome had experienced a rapid postoperative recovery, which implies a mild and focal brainstem injury.

In conclusion, we present a rare case of secondary traumatic brainstem hemorrhage that experienced a rapid and good recovery process. The mechanism is still obscure to us and needs to be further studied. Although traumatic brainstem hemorrhage usually means a fatal event to most patients, some patients may experience a favorable recovery. This rare circumstance should be stressed in prognosis consultation and clinical management of these kinds of patients.

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Conflict of interest statement: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The authors declare that they have no conflict of interest.