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Case Presentation

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Post-Traumatic Cerebral Infarction in Subdural Hematoma and Hemorrhagic Transformation Following Burr-Hole Drainage: A Case Report

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Abstract

Background: Although post-traumatic cerebral infarction (PTCI) is a known complication of traumatic brain injury (TBI), to the best of our knowledge, postoperative hemorrhagic transformation (HT) after PTCI has not been reported previously.

Case description: We present the case of a 43-year-old woman with hemorrhagic cerebral infarction (HCI) following burr-hole drainage for a traumatic subdural hematoma (SDH). Computed tomography (CT) confirmed PTCI in the left occipital lobe 9 days after TBI. We performed burr-hole drainage for the left acute-on-subacute SDH. Two days after the surgery, head CT revealed hemorrhagic transformation (HT) in the ischemic cerebral infarction.

Conclusions: HT may occur in PTCI and burr-hole drainage may cause it. We suggest that neurosurgeons should consider the possible presence of postoperative HCI when operating for patients with PTCI.

Keywords: Burr-hole drainage; Hemorrhagic cerebral infarction; Hemorrhagic transformation; Post-traumatic cerebral infarction; Subdural hematoma; Traumatic brain injury

Introduction

Post-Traumatic Cerebral Infarction (PTCI) is a recognized complication of Traumatic Brain Injury (TBI), with a frequency from 1.9% to 10.4% **[1,2].** It is always associated with poor outcome and high mortality rate **[3,4].** Hemorrhagic Transformation (HT) is a complication of ischemic stroke **[5,6]** and can be triggered after reperfusion **[7].** However, because etiology and pathology of HT are very complex, whether it occurs in PTCI is unclear. We encountered a rare case with hemorrhagic transformation of PTCI following burr-hole drainage for a Subdural Hematoma (SDH).

Case Presentation

A 43-year-old woman presented after a fall with headache. Seven days prior to admission, she had fallen and struck her head. Initial head Computed Tomography (CT) showed an acute left frontotemporoparietal SDH with 4 mm Midline Shift (MLS) **(Figure 1A).** She was admitted to local hospital and underwent conservative therapy. Seven days later, her headache was aggravated so she was transferred to our hospital. The Glasgow Coma Score (GCS) on admission was 14 (E3V5E6) and her examination was without focal deficits. Head CT revealed an acute-on- subacute left frontotemporoparietal SDH with 8 mm MLS **(Figure 1B).** Labs revealed platelet count was 441 × 109/L and without coagulopathy. She was managed conservatively. Two days after admission, her GCS was 7 (E2V1E4), with anisocoria (left larger than right). Repeat head CT demonstrated enlargement of the SDH with 15 mm MLS, cerebral infarction in the left occipital lobe and descending tentorial herniation **(Figure 1C and D).** She was taken urgently for left burr-hole drainage. Anticoagulants were not given. Post-operative CT showed the subacute SDH had been removed and improvement in MLS **(Figure 1E).** Two days after the surgery, head CT showed hemorrhage in the ischemic cerebral infarction **(Figure 1F).** She was treated conservatively. Two weeks after the surgery, head CT revealed the left interhemispheric SDH and MLS significantly reduced but hyperintensity still existed in the infarction area **(Figure 1G)** and the patient was discharged with right homonymous hemianopia.

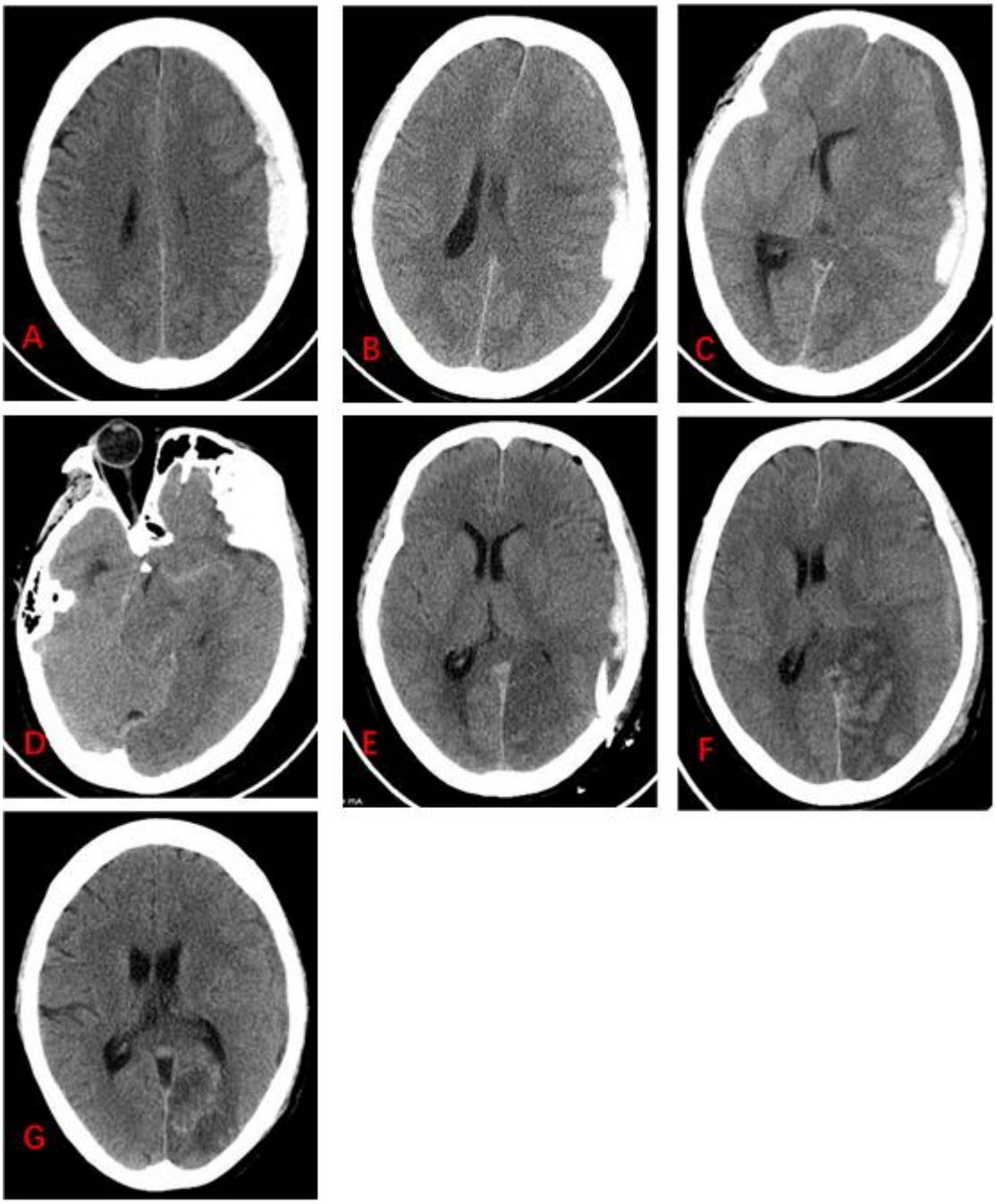

Figure 1: Serial Computed Tomography (CT) scans. A) Initial CT after Traumatic Brain Injury (TBI) showed an acute Subdural Hematoma (SDH) in the left hemisphere with 4 mm Midline Shift (MLS). B) CT 7 days after TBI revealed a left acute-on-subacute SDH with 8 mm MLS. C, D) CT 9 days after TBI demonstrated expansion of the acute-on-subacute SDH with 15 mm MLS, cerebral infarction in the left posterior cerebral artery territory and descending tentorial herniation. E) CT 2 hours after burr-hole drainage showed subacute SDH removed and improvement in MLS. F) CT 2 days after burr-hole drainage demonstrated Hemorrhagic Transformation (HT) without mass effect in left occipital lobe infarction. G CT 1 week after HT revealed significant reduction of SDH and MLS but existence of hypertensity in infarct area.

Discussion

PTCI is a well-known complication of moderate or severe TBI. Possible mechanisms include vascular compression, cerebral vasospasm, direct vascular injury, embolization and systemic hypoperfusion **[1-4].** Among these mechanisms, mechanical compression or displacement of major cerebral arteries was thought to be the most common cause of PTCI **[3,8].** Compression of the posterior cerebral artery by the tentorial herniation mainly due to subdural hematomas or epidural hematomas is recognized as the cause of infarction of the occipital lobe and is perhaps the most well-recognized mechanism leading to PTCI **[1,2,9,10].** A CT scan showed evidence of transtentorial herniation and left occipital infarction in our patient with an acute-on-chronic SDH which produced significant mass effect.

PTCI can be diagnosed by CT scans. However, CT scans are limited in detecting early PTCI and revealing etiologies of PTCI. Head CT Angiography (CTA) or Digital Subtraction Angiography (DSA) can be used to reveal vascular spasm and vascular injury. Unfortunately, our patient refused to accept CTA or DSA examination because of economic reason, so the role of vascular spasm or vascular injury in producing cerebral infarction in our patient is unknown. Ischemic stroke can occur HT. HT may be related to reperfusion injury, oxidative stress, neuroinflammation and neurovascular activation **[7].** The risk factors of HT include old age, hypertension, hyperglycemia, lowered platelet count, large infarcts, reperfusion time, and thrombolytic, antithrombotic or anticoagulant treatments **[11].** Although HT is usually associated with embolic stroke and intra-arterial or intravenous thrombolysis treatments **[12,13],** it is likely a multifactorial phenomenon **[14].** In our case, it occurred secondary to PTCI which is extremely rare. Furthermore, the burr-hole drainage surgery for the acute-on-chronic SDH may be the etiology of HT. The medium size of infarct was the major risk factor for HT **[15]** and the postoperative HT can be explained in our patient. The burr-hole drainage successfully reduced the compression of brain tissue and reperfused the ischemic left occipital lobe. The perfusion injury as a potential trigger of HT undermined the integrity of Blood-Brain Barrier (BBB) leading to blood extravasation **[16].** On the basis of the radiologic anatomy of the lesions, HT is divided into two types: Hemorrhagic Infarction (HI) and Parenchymal Hematoma (PH) **[13].** HI is a punctate or heterogeneous hyperdensity in an ischemic infarct area, whereas PH refers to a typical homogeneous, hyperdense lesion with or without mass effect **[13,17].** Previous studies suggest that HI indicates early recanalization and better perfusion related to favorable early outcome but PH may cause neurological deterioration and is associated with adverse outcome **[5,13,17,18].** In our case, the type of HT was HI and the effect of it on the prognosis will be found in our follow-up of this patient.

Conclusion

We represented a case with PTCI and its HT after burr-hole drainage. From our case, we can see that HT may occur in PTCI and burr-hole drainage may cause it. So, we suggest that neurosurgeons should be aware of the possible occurrence of HCI after TBI, especially when operating for patients with PTCI.

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