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Case Report

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Middle Cerebral Artery Compromise Associated With Post-traumatic Hydrocephalus: A Case Report

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Conflict of Interest
The authors have no financial conflicts of interest.

ABSTRACT

Post-traumatic hydrocephalus (PTH) is a commonly encountered complication following decompressive craniectomy, and is usually characterized by symptoms including headache, nausea, vomiting, and papilledema. Extracranial herniation accompanied by hemiplegia is a rare complication in patients with PTH who underwent craniectomy after subdural hematoma removal. We report a case of PTH that presented with extracranial herniation within one month of decompressive craniectomy. Following ventriculoperitoneal shunt implantation, left hemiplegia improved dramatically with restoration of the left middle cerebral artery blood flow, which was evident on serial imaging. Vascular compromise is often overshadowed by increased intracranial pressure when clinicians are dealing with traumatic brain injury patients. Delicate neurological and radiological examinations and prompt early interventions could lead to optimal outcomes in patients receiving decompressive craniectomy.

Keywords: Post-traumatic hydrocephalus; Encephalocele; Ventriculoperitoneal shunt

INTRODUCTION

Extracranial herniation usually happens within one week from the decompressive craniectomy and is a sign of increased intracranial pressure (IICP). Post-traumatic hydrocephalus (PTH), on the other hand, happens mostly after one month from the surgery. In our case, a man with PTH and extracranial herniation within one month after decompressive craniectomy suffered from left hemiplegia. After ventriculoperitoneal (VP) shunt implantation, the neurological deficit improved and vascular compromise in the brain magnetic resonance imaging (MRI) wasn't observed in the followed-up images. The purpose of the case report is to emphasize the importance of early intervention.

CASE REPORT

This is an 82-year-old gentleman whose daily activity is totally independent but with underlying diseases inclusive of type 2 diabetes mellitus, hypertension, dyslipidemia, gouty arthritis, and atrial fibrillation. He was involved in a motor-vehicle accident and was sent

to hospital for immediate medical attention. Initial Glasgow Coma Scale (GCS) was 15. No initial loss of consciousness and retrograde amnesia were recorded, but with right side contusion, multiple rib fractures and pleural hematoma.

One day after initial trauma, altered mental status was noted. GCS dropped to 5 (E1V2M2). Emergent brain computed tomography (CT) revealed right fronto-temporo-parietal acute subdural hematoma (SDH) with subfalcine herniation. After explaining the condition to his family, we arranged an emergent right decompressive craniectomy with subdural hemorrhage removal as a life-saving procedure to manage a patient's IICP.

In the intensive care unit caring after operation, followed up brain CT showed resolution of SDH with mild brain edema. He regained full consciousness but failed to wean the mechanical ventilator. So tracheostomy procedure was performed.

After stabilizing, he was transferred to an ordinary ward with 4 limbs muscle power in 4 points in Medical Research Council (MRC) scales and under regular rehabilitation programs.

However, 30 days after the craniectomy, left hemiplegia was noted with muscle power dropping from 4 to 2 points in MRC scales, and scalp showed mild bulging without wound dehiscence. A brain CT was done showing no obvious sign of ischemic stroke.

Subsequent brain MRI showed mild extracranial herniation with subgaleal fluid accumulation and communicating hydrocephalus with Evans index exceeded 0.3 (**FIGURE 1**). Magnetic resonance angiogram (MRA) revealed decreased blood flow over the right middle cerebral artery (MCA), especially the disappearance distal to the M2 segment (**FIGURE 1**). A VP shunt was suggested to manage the patient's hydrocephalus and the patient consented to the operation. His muscle power improved gradually from 2 to 4 points in MRC scales within one week after VP shunt insertion. Followed up post-operative brain MRA depicted restoration of blood flow in the superior segment of M2 (**FIGURE 2**). GCS improved to 11 (E4VtM6). He was discharged after 10 days and received rehabilitation programs in our hospital with regular follow up at the neurosurgical outpatient clinic.

DISCUSSION

Complications secondary to decompressive craniectomy in patients with traumatic brain injury (TBI) are important and not frequently discussed in the literature. These complications occur in a sequential fashion at specific times after decompressive craniectomy. In the early stage after decompressive craniectomy, the most frequent complications are inclusive of progressive hemorrhagic injury (within 3 days), epilepsy (within 3 days), extracranial herniation (within 5 days).[3] After 7 days, infection and subdural hygromas could develop between one week and one month; delayed complications are mainly hydrocephalus and syndrome of the trephined, which happens after one month.[3,4]

Extracranial herniation mostly happens in the setting of IICP with a skull defect, and it appears frequently in the first week after operation of craniectomy.[4] When extracranial herniation is accompanied with hydrocephalus, it could be deciphered that the underlying process of IICP induces both situations, so aggressive evaluation and management of the IICP should be considered.

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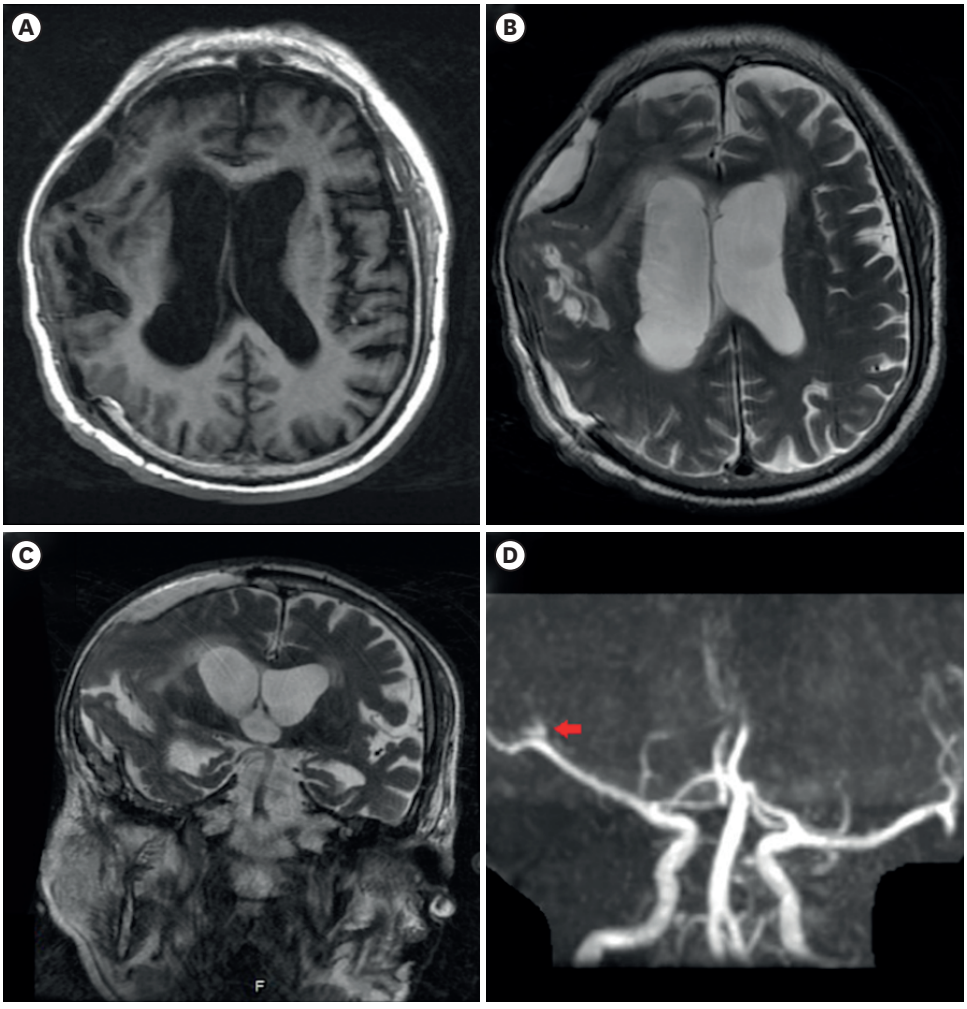

FIGURE 1. Brain magnetic resonance imaging series in our patient status post craniectomy. (A, B) The upper column showed contrast enhanced axial T1 and T2 weighted images before ventriculoperitoneal shunt implantation. They revealed post-traumatic hydrocephalus and extracranial herniation. (C, D) The lower column showed contrast enhanced coronal T2 weighted and 3-dimensional time of flight MRA image. The MRA showed the superior division of M2 of right MCA being trapped, causing the following blood flow to be compromised (arrow). MRA: magnetic resonance angiogram.

PTH is a subtype of hydrocephalus and its incidence is variable, ranging from 0.7% to 45%.[2] PTH happens when blood clots bled into the ventricles leading to obstruction at the site where cerebrospinal fluid (CSF) flows into the convexities, and thus induce the following non-communicating hydrocephalus.[2] Classic symptoms of PTH are mostly non-specific and could be overlooked and ignored unintentionally since the symptoms of PTH may mimic the sequelae after TBI. What's more is that if the patient remained unconscious, it's impossible to tell PTH from sequelae of TBI according to clinical manifestation, unless imaging examinations could make the final diagnosis.[2]

In our case, the patient suffered from TBI with right SDH and he received operation of right decompressive craniectomy and SDH removal. After the operation, his consciousness improved to clear and 4 limbs muscle power were in the MRC grade 4. However, one month after operation, extracranial herniation from the skull defect and left hemiplegia were noted, with muscle power deteriorating from MRC grade 4 to grade 2. For suspicion of a new

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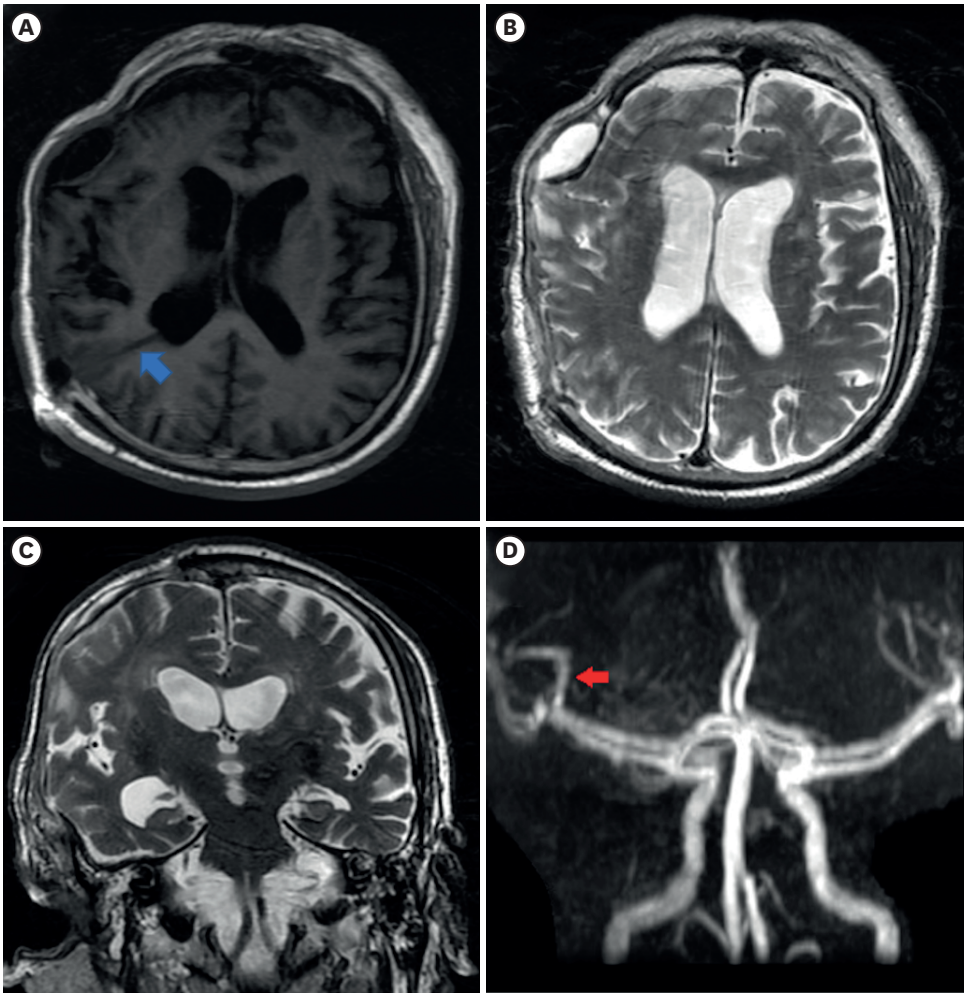

FIGURE 2. Brain magnetic resonance imaging series in our patient status post VP shunt management. (A, B) The upper column showed contrast enhanced axial T1 and T2 weighted images after VP shunt implantation. They revealed improvement of hydrocephalus and extracranial herniation. (C, D) The lower column showed enhanced axial T1 and T2 weighted images before VP shunt implantation. The follow-up 3-dimensional time of flight magnetic resonance angiogram revealed the restoration of blood flow in the superior segment of M2 of right middle cerebral artery (arrow).
VP: ventriculoperitoneal.

onset of stroke, brain MRI showed no infarction but hydrocephalus (**FIGURE 1**), extracranial herniation, and decreased blood flow over right MCA. Under normal circumstances, the insular segment (M2) of the MCA commonly divides into 2 or 3 branches. However, in our case, the MRA showed the superior division of M2 of the right MCA being trapped, causing the following blood flow to be compromised (**FIGURE 1**). This could be explained by the concomitant PTH and extracranial herniation, since these 2 conditions lead to the trapping of the M2, causing it being avulsed outward by internal expansive force from PTH and external stretching force from the skull defect. After the examination of brain MRI, with suspicion of temporary brain ischemia inducing left hemiplegia, we then arranged VP shunt. Motor deficit improved markedly within one week of the shunt operation, with left limbs muscle power improved from MRC grade 2 to grade 4. Repeated brain MRI scan was arranged 2 weeks later and the MRA demonstrated restoration of cerebral blood flow in the superior segment of M2 (**FIGURE 2**) commensurate with return of motor strength.

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Hydrocephalus is an excessive accumulation of CSF and is often in both sides. It's known to be related to symptoms including conscious disturbance, unsteady gait, and general weakness. Our patient presented with left hemiplegia and muscle powers of his right limbs were still 4. So it's less likely to be mainly caused by hydrocephalus.

For patients with TBI post decompressive craniectomy, it is believed that PTH shall increase the risks of complications and also cause further clinical deterioration. However, it remains challenging to all physicians to detect PTH early and arrange prompt management, such as VP shunt and so on. As long as these symptoms induced by PTH are reversible, physicians should try to diagnose and manage it as early as possible. Though there still remains no consensus on the timing of VP shunt post PTH, we believe the operation of VP shunt should be performed once the neurological deficit induced by PTH appears.[5)]

Aside from above mentioned issues, we also consider that PTH and syndrome of the trephined may share some clinical similarities, such as skull defect and hemiplegia.[1)] In our case, the symptoms are not induced by the atmospheric pressure, but the internal force of PTH. This may imply that the underlying mechanism of these 2 diseases could not be simply interpreted in the presence of the skull or not, but also other factors should be taken into account, such as the blood flow and other factors. The underlying mechanism still needs further discussion and investigation, with the advance of multiple model technologies in the future. We would like to share our case, though with PTH and extracranial herniation as complications after craniectomy, it is imperative to know that PTH cases with vascular compromise as the prominent etiology is treatable with CSF shunting. Patients could recover well with early diagnosis and timely interventions.

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