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Transient Obstructive Hydrocephalus Associated with Cerebrospinal Fluid Dynamic: An Unusual Case of Early Neurological Deterioration in Intracerebral Hemorrhage Patient

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Abstract

Spontaneous Intra Cerebral Hemorrhage (ICH) is a subtype of stroke and has a high mortality and disability rate. Early Neurological Deterioration (END) is a common complication of ICH, that refers to the rapid deterioration of the disease in the early stages and is related to poor long-term prognosis. There are widely used rating scales and a large number of studies that analyze risk factors in order to identify those at the highest risk and measures to response. However, rare END caused by special mechanic cannot be predicted using routine clinical evaluations. We describe an old patient presented a sudden END, characterized by projectile vomiting and rapidly deteriorating state of consciousness 60 hours post-onset of ICH, who was initially classified as low risk during routine clinical evaluation. Through a detailed review of imaging data, a shrunk blood clot was found lodged in the midbrain aqueduct driven by Cerebro Spinal Fluid (CSF) circulation, causing acute obstructive hydrocephalus, and conservative treatments were administered after a thorough discussion of the underlying mechanism. The patient's consciousness gradually returned to awake within 48 hours when the blood clot got through the stenosis aided by the elevated pressure of CSF dynamic in turn of hydrocephalus. Follow-up results confirmed that this case of END, which manifested as transient obstructive hydrocephalus, had no effect on long-term prognosis, and this rare CSF dynamic factor has not been previously considered in predictive models for END and can't be overlooked.

Keywords: Intracerebral hemorrhage; Intraventricular hemorrhage; Obstructive hydrocephalus; Cerebrospinal fluid dynamic; Early neurological deterioration

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Introduction

Spontaneous Intracerebral Hemorrhage (ICH) is a subtype of stroke, accounting for approximately 27.9% of strokes, which increases significantly with age in the population and has a high mortality and disability rate, of which about 70% have a poor long-term prognosis [1]. A systematic review and meta-analysis have revealed that Early Neurological Deterioration (END) is a common complication and the pooled prevalence is 23% [2], among the various factors that impact the ultimate outcome of a patient following initial ICH. END is generally characterized by a significant increase in the National Institutes of Health Stroke Scale (NIHSS) score or a decrease in the Glasgow Coma Scale (GCS) score. Nevertheless, alternative definitions may also be employed, which has engendered inconsistency in the clinical studies [3-5].

END primarily reflects changes in intracranial pathophysiology that can be distinctly visualized through imaging examinations. However, continuous neuroimaging monitoring is difficult [6]. Combining clinical rating scales with baseline neuroimaging parameters such as hematoma volume, intraventricular hemorrhage, intraventricular expansion, hematoma expansion, and the spot sign can help create a predictive model to identify those at the highest risk [7], but rare factor related with cerebrospinal fluid dynamic may still be overlooked.

Case Presentation

A 70-year-old male patient was transported to the emergency care unit via ambulance after experiencing sudden dizziness and vertigo, which rendered him unable to walk for two hours. There

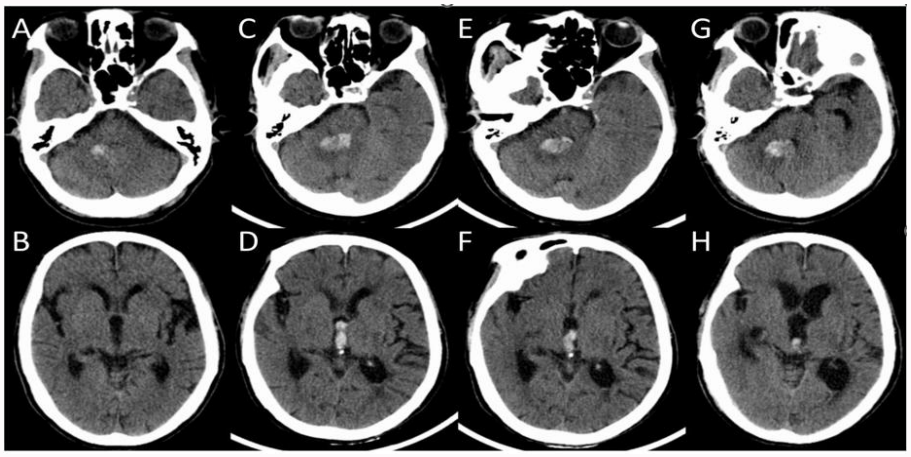

Figure 1: CT scan of the baseline and followed-up till the time of END.
A/B depicted the emergency Computed Tomography (CT) scan of the patient who was admitted to our NICU on the initial day, and the Modified Graeb Score (MGS) score was 0; C/D revealed cerebellar hematoma expansion and rupture into the fourth ventricle, inverting into the third ventricle via the midbrain aqueduct on the second day of admission, leading to an increased MGS score of 8 without positive clinical change; E/F showed that on the third day of admission, the volume of hematomas in the ventricles had decreased, and the MGS score dropped to 6; G/H illustrated the CT scan taken immediately after the patient's consciousness deteriorated, with the Glascow Coma Scale (GCS) score declining from 14 to 7, and the MGS score increased to 11.

were no accompanying symptoms of nausea, vomiting, or tinnitus. The patient's consciousness was stable and a brain Computed Tomography (CT) scan revealed a right cerebellar hemorrhage of about 0.6 ml. The Computed Tomography Angiography (CTA) scan detected mild to moderate cerebral vessel stenosis, with no aneurysms or arteriovenous malformations. Following consultation, the patient was admitted to the Neuro Intensive Care Unit (NICU) for further treatment. The patient has had Grade 2 hypertension for more than 20 years, managed daily with Amlodipine maleate. He also has chronic non-atrophic gastritis for over two decades and a history of duodenal coccitis. The history of an old cerebral infarction is unknown.

At the initial inspection, the patient had a blood pressure of 186/112 mmHg and a pulse rate of 96 beats per minute. His Glasgow Coma Scale (GCS) was 14(E3V5M6), NIHSS score was 2, and Modified Graeb Score (MGS) score was 0. He exhibited no dysarthria, nystagmus, or diplopia. Cranial nerve examination was normal, as were the reflexes, strength, and peripheral extremities.

The laboratory examination results were as follows: fasting blood glucose, 8.06 mmol/L; White blood cell count, 12.63 × 10^9/L; total number of platelets, 416 × 10^9/L; neutrophil percentage, 82%; serum potassium, 3.01 mmol/L; blood chloride, 98.75 mmol/L; serum calcium, 2.07 mmol/L. No other abnormalities were found in routine blood and urine analyses. Subsequent Magnetic Resonance (MR) showed a few old cerebral infarctions and moderate cerebral atrophy, but no significant parenchymal lesions at the site of bleeding were found.

Intensive antihypertensive therapy, routine follow-up CT scan, and supportive care such as replenish electrolytes were utilized in NICU and his dizziness was significantly relieved. Approximately 24 h later, head CT revealed that the cerebellar hematoma had expanded, ruptured into the fourth ventricle, and inverted into the third ventricle via the midbrain aqueduct, increasing the MGS score from 0 to 8, with no change in GCS and NISHH scores. At 48 hours, head CT showed a reduction in hematoma volume in the ventricles, lowering the MGS score from 8 to 6. Unexpectedly, around 60 h post-onset, the patient's consciousness shifted from alertness to light coma (GCS score of 7) accompanied by violent vomiting. An immediate head CT indicated supratentorial obstructive hydrocephalus was progressed rapidly and the MGS score increased to 11. A punctate hyperintensity at posterior region of third ventricle in the axial position, indicating a blood clot stuck at the midbrain aqueduct (Figure 1).

Following a thorough discussion of the underlying mechanism, conservative dehydration drugs and anti-infective treatments were administered. After 48 hours, a CT scan showed the stuck blood clot at the midbrain aqueduct was disappear, the obstruction was relieved, and the patient's consciousness gradually returned to awake (Figure 2). Following successful treatment of aspiration-induced pneumonia, the patient's neurological symptoms completely subsided and blood pressure was well controlled during a 20-day hospital stay. No progression to communicating hydrocephalus was observed over the subsequent year.

Discussion

END primarily indicates intracranial pathophysiological exacerbation in a relatively short period after the initial ICH and is often accompanied by positive changes on followed-up imaging. END is a common complication defined as a significant increase

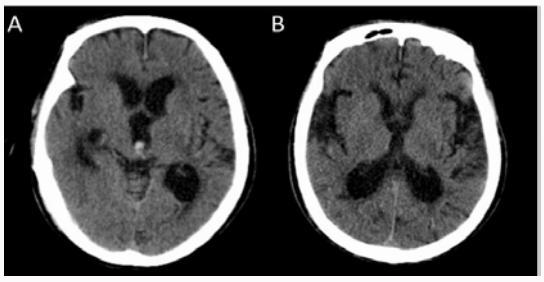

Figure 2: CT scan at the time of END and 48h later.
A: The axial image of the head Computed Tomography (CT) scan displayed punctate hyperintensity in the posterior region of the third ventricle, which may indicate that the blood clot is obstructing the midbrain aqueduct, consequently leading to impaired circulation of cerebrospinal fluid; B: CT scan 48 hours later showed that the abnormal signal disappeared and the ventricular system returned to its normal state.

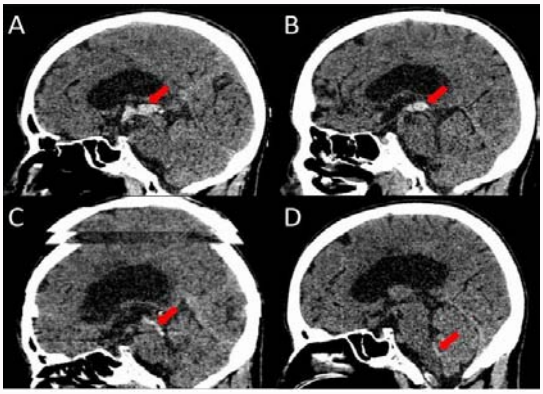

Figure 3: The exacerbation and remission of END were reviewed in the sagittal position.
A: The cerebellar hematoma expanded and ruptured into the fourth ventricle and flowed backward into the third ventricle(red arrow); B: The hematoma diminished, softened, and moved with the CSF circulation to the posterior third ventricle; C: The hematoma was stucked when descending through the midbrain aqueduct, leading to a sudden END in this patient, appearing as an elongated strip; D: Driven by elevated cerebrospinal fluid circulation, the hematoma passed through the midbrain aqueduct and the clinical symptoms resolved within 48 hours.

in the National Institutes of Health Stroke Scale (NIHSS) score or remarkable decrease in the Glasgow Coma Scale (GCS) score from baseline to 24 h [3]. A recent systematic review found that the pooled prevalence of END was 23% [2]. But the validity of this conclusion is subject to debate due to varying timeframes for defining END, and our study focuses on the first 72 hours following the onset of ICH, attempting to explore the causes of END from another rare perspective.

It was reported that the initial NIHSS score, GCS score, as well as hematoma volume, intraventricular hemorrhage, intraventricular expansion, hematoma expansion, and the spot sign were associated with an increased probability of END [2]. In this case, although the hematoma in the right cerebellum expanded, it did not exceed 1 ml after all and the spot sign was negative. Clinical rating scales are fairly stable, so we classify him as low-risk patient.

Approximately one-third of intraparenchymal hemorrhage patients experience early-stage hematoma expansion, defined as an increase in hematoma volume by over 33% or greater than 12.5 mL on follow-up CT [8]. In this case, we used rapid, intensive, and sustained blood pressure lowing therapy within the first 24 hours of admission, which was thought to minimize the tendency of the hematoma expansion [9], but subsequent CT revealed that this measure was not effective in achieving the desired outcome. Increased hematoma mass effect and accompanying edema may compress adjacent brain tissue and may cause elevation of Intra Cranial Pressure (ICP) and potential fatal herniation syndromes and the complication rate arises significantly when an intraparenchymal hematoma mass breaks into the ventricular system, which is called Secondary Intra Ventricular Hemorrhage (SIVH). In this case, despite the cerebellar hematoma's expansion, no significant mass effect was observed due to its small size. The patient's condition deteriorated primarily because of the blood mass in the ventricular system. Therefore, we employed the Modified Graeb score (MGS) to quantify the portion ruptured into the ventricular system, which demonstrates its efficacy as a relatively accurate and convenient tool while changes in clinical observations remain minimal [10,11].

Compared with SIVH, the prognosis of Primary Intra Ventricular Hemorrhage (PIVH) is more dependent on whether the cause of the bleeding is adequately managed. The etiologies of primary intraventricular hemorrhage can be attributed to hypertension, Arterio Venous Malformation (AVM), aneurysm, Moyamoya disease, coagulopathy, and other rare factors such as hematologic disorders, intraventricular tumors, thrombocytopenia and so on [12]. In this case, subsequent non-contrast Magnetic Resonance (MR), 3D time-of-flight Magnetic Resonance Angiography (MRA), and contrast-enhanced MRA [13] during hospitalization revealed no further hemorrhage-related lesions.

With regard to SIVH, numerous predictive models in clinical prognosis studies in the early period have incorporated it as a binary variable [14,15], suggesting its association with poorer long-term outcomes; however, this is very rudimentary and unreliable. Further research has disclosed that if hydrocephalus is not caused, SIVH do not significantly affect the long-term prognosis [11,16]. However, some scholars argue that the harm of ependymal and subependymal structures and inflammatory intraventricular processes may lead to a worse prognosis due to the stimulation of blood components and interference with CSF circulation over time [17].

Accurate calculation of hematoma volume in the ventricles is neither difficult nor necessary. The Graeb Score (GS) is a reliable, simple, quick, and meaningful approximation of IVH volume that based on the distribution of hematomas in the ventricles to perform semi-quantitative score ranging from 0 to 12. It is generally believed that the Graeb score of ≥3 was related to both increased mortality and worse functional outcome. The MGS is an improvement over GS because it is more accurate ranging from 0 to 32, weighing subcompartments of the ventricular system and hematoma volume and thus improved description of the patient's condition. Although it is well established that the original GS [18], the MGS [19], LeRoux score [20], Hallevi Score [21], and The slice score [22] all can achieve a good evaluation in IVH in certain aspects, their predictive value for END and long-term prognosis is still inconclusive.

The initial management of SIVH begins with close monitoring of the patient's clinical status, including regular clinical rating scales and frequent imaging studies to assess intracranial pathophysiological changes. Conservative measures rely primarily on medications, such as the use of osmotic agents (e.g., mannitol or hypertonic saline) to reduce intracranial pressure, and diuretics (e.g., acetazolamide or furosemide) to reduce cerebrospinal fluid production [23].

Once obstructive hydrocephalus occurs, which is the most serious complication of SIVH, External Ventricular Drainage (EVD) surgery is the most classic countermeasure [24], sometimes followed by Intra Ventricular Fibrinolysis (IVF), which involves intraventricular injection of recombinant tissue plasminogen activator (rt-PA). EVD has shown effectiveness in reducing intracranial pressure and stabilizing patients while awaiting definitive treatment options. Other modalities have been reported, including direct physical removal of intraventricular blood clots through a specific approach craniotomy, endoscopic removal of intraventricular hematoma with or without Endoscopic Third Ventriculostomy (ETV), Lumbar Drainage (LD), and an appropriate combination of these. Despite the best efforts, some patients may still require definitive treatment such as a Ventriculo Peritoneal (VP) shunt or endoscopic third ventriculostomy for delayed communicating hydrocephalus [25].

Our clinical management failed to predict the sudden deterioration of the patient's condition due to the rarity of the exacerbating factors: transient obstructive hydrocephalus linked to CSF dynamic. The final treatment plan hinges on the duration of obstructive hydrocephalus, as prolonged cases may cause irreversible brain damage. The cerebellar hematoma expanded after the initial Intra Parenchymal Haemorrhage (IPH) and broke into the third and fourth ventricle, passing through the midbrain aqueduct twice: at the first time, the blood clot strived against the stream of CSF through the midbrain aqueduct to the third ventricle without causing significant intracranial pressure changes; but the second time, its moving downstream under the driving force of CSF from the supratentorial to the infratentorial region through the stenosis caused a sudden clinical decline due to a rapidly onset of obstructive hydrocephalus. We had an intense discussion and finally reached a unanimous hypothesize that this hydrocephalus is transient and will resolve immediately once the stuck hematoma passes through the midbrain aqueduct aided by the elevated pressure of acute obstructive hydrocephalus. The favorable prognosis following conservative treatment confirm our conjecture. In the sagittal view of the CT scan clearly shows the dynamic trajectory of the blood clot (Figure 3). Despite the occurrence of aspiration-induced pneumonia, we have fortified our patients to avoid aggressive surgical interventions such as EVD.

Conclusion

The present case highlights the limitation of predictors in accurately forecasting END and the importance of dynamic continuous imaging and clinical evaluation. A comprehensive knowledge of the mechanism of transient obstructive hydrocephalus in relation to CSF dynamic can help avoiding excessive surgical intervention and ensuring patient's prognosis.

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