

# Pre-hernia Intervention for Cascade Cerebral Hernia Caused by Supratentorial Traumatic Acute Subdural Hematoma

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**Abstract:** *Subject* To investigate the characteristics and diagnosis and treatment which were pre-hernia intervention of cascade hernia. Reduce rate of mortality and disability that is attention must be paid to prehospital intervention before the formation of a true cascade of brain herniation. clinical series of pre-hernia manifestations were rapid that from sequential brain tissue displacement in part patients, and by development. Leading to a clinical series of pre-hernia by formed. The effect of pre-hernia intervention on prognosis which from timely one. *methods* From June 2015 to December 2022, the 61 patients with unilateral acute frontotemporal parietal subdural hematoma were treated. From June 2015 to December 2022, 61 patients with unilateral frontotemporal acute subdural hematoma were evaluated according to GCS. Twenty-five patients with cascade cerebral hernia and 36 patients with cascade cerebral hernia before hernia underwent dynamic CT examination, and patients with pre-herniation of brain cascade hernia were treated with early intervention. *Results* The patients were followed up for 6 to 12 months. According to the Glasgow Outcome scale (GOS), there were 25 patients in the brain cascade hernia group; either with 3 cases of 1 point, 4 cases of 2 points, 5 cases 3 points, 4 cases 3 points, 2 cases of 4 points, and 9 cases of 5 points. There were 36 patients in the prehernia group: 1 case of 1 point, 3 cases of 2 points, 4 cases of 3 points, 11 cases of 4 points and 17 cases of 5 points. *Conclusions* Unilateral supratentorial acute frontotemporal parietal acute subdural hematoma leading to the cascade cerebral hernia is easy to lead to instability of clinical vital signs or respiratory and circulatory failure. Patients must be treated promptly at the early stage or stage I-III or in IV of cascade cerebral hernia in this group. Pre-hernia intervention is the key to reduce mortality and morbidity.

**Keywords:** Supratentorial Acute Subdural Hematoma, Cascade Cerebral Hernia, Pre-hernia Intervention, Curative Effect

## 1. Introduction

The incidence of acute subdural hematoma traumatic of frontotemporal parietal is highest. It is a common type of intracranial hematoma that is cerebral hernia may occur when not treated in time, also. The incidence of tentorial notch herniation is relatively highest, that among them many with hernias [1, 2] which is contusion and laceration of unilateral or bilateral frontal lobes, and hemorrhage of thalamus secondary to central cerebral hernia, and Hematoma of extra-intracerebellar secondary to cerebellar tonsil hernia, separately [3-5]. Sometime, It can also be found that were multiple hernias from no acute subdural hematoma by

scholars report [6]. The mortalities of brain hernias by the other author's found mortalities of 30% and 90% in patients with ASDH operated on  $\leq 4$  and  $>4$  hours after injury, respectively [7]. Leading causes of highest mortality is relationship with various types of brain hernia, but it's not the only factor which is closely related to the duration of cerebral hernia at admission. However, the morbidity and mortality caused by cascade brain hernia have not been reported.

But its basic pathological characteristic is sequential displacement of brain tissue which secondary to damages of unilateral or bilateral midbrain and pons, and even medulla oblongata. Then the lead of pupil dilation, instability of vital

signs, and severe function dysfunction of respiratory and circulatory or even failure. This process is considered to be the basic feature of the sequential herniation of the cerebral and is called the cascade herniation of the brain. And is significantly different from the concept of latest cerebral hernia. The mortality rate and disability are very highest. A different types of brain hernias caused by other intracranial hematoma or acute subdural hemotoma are not easy to distinguish. At present, There is no the whole world report of this cascade of cerebral hernia. Therefore, from June 2015 to the December 2022, our department diagnosed and treated 25 patients of cerebral cascade hernia and 36 patients of pre-hernia patients with cerebral cascade hernia by early intervention, and achieved good results, the report is as follows:

## 2. Clinical Materials and Methods

### 1) General information:

Sixty-one patients with acute subdural hematoma of unilateral in intracranial frontotemporal parietal region were selected in this group, including 47 males and 14 females (33cases on the right and 28 cases on the left). The age ranged from 24 to 55 years (mean 45 years). The causes of trauma were traffic accidents. The patients were vided into two groups that is the cerebral cascade hernia group and the pre-cerebral cascade hernia group: There were 25 patients that is early cerebral hernia upon when arrived ambulances at their destination in 10 cases, and 15 cases occurred during the in the hospital emergency medical service. 36 cases of cascade hernia were treated with pre-hernia interven. Other patients with non acute subdural hematomas of the intracranial hematoma were not included in this investigatived.

### 2) Clinic al manifestations

In the brain cascade hernia group, 25 patients had different degrees of disturbance of consciousness, among GCS score of 3-5 in 10 cases, and GCS score of 6-8 in 15 cases. Pupil diameter were dilated of unequal or progressively in the left and right sides respectively. Sometime, The pupillary were responded association with light weakene or disappeared in 20 case respectively, that is first left dilated, then right fixed dilated of pupil, with adiameter of 6mm in 5cases respectively. Howery pathological changes in all pupils diameter with 1 hour, with secondary had cheyne-stokes breathing in 15 cases, and had hyperbreathing in 5 cases, Limb hypertonia with pathological reflex was found in 20 cases and limb atonia in 5 cases.

There were 36 patients of pre-herniation of brain, early mild disturbance of consciousness, headache, repeated vomiting, no obvious change in pupil, 15 patients of reaction, mild coma, vomiting, agitation, one side of the pupil s lowly progressive dilation, one side of the pupil constriction, light response weakening in 15 cases, hallow coma, severe agitation, frequent vomiting, The diameter of bilateral pupils was 3.5mm and sometimes it from big to small, and in 6 cases had weak response tolight. Hypertonia on the affected side was found in 30 cases (extremities), and had flaid

paralysis in 6 cases.

### 3) Imaging date

Emergency CT examination was performed in both groups: Either, such has 28 patients and 33 patients with the acute subdural hematomas of left and right frontal, temporal and parietal in the brain cascade hernia group, respectively. In the brain cascade hernia group, the cingulate gyrus was shifted to the contralateral side through the subfalx foramen. After a short period of time, CT examination showed that the frontal angle of the affected side was narrowed on frotal horn, the third ventricle was narrowed and displaced, the cisterns were not clearly displayed, the mesencephalon and pons were low density and formed, and the midline was shifted  $\geq 1.0\text{cm}$ . Brain level CT examination of 36 patients in the pre-hernia group showed that in the early stage, the cingulate gyrus of the same hematoma side was lightly shifted to the contralateral side through the falx foramen, the cisterns of the lateral cleft were narrowed and the brain parenchyma slightly moved down. The reexamination of CT confirmed that the volume of hematoma increased, the third ventricle was shifte disappeared, the cisterns around the mesencephalon disappeared, the esencephalon and pons were low density and deformet the same, the medulla oblong was low density and the cerebellar tonsil was shifted down, and the midline was shifted 1.2cm.

### 4) Diagnosis criteria of cerebral cascade hernia.

Diagnostic criteria for cascade cerebral hernia of acute subdural hematoma in supratentorial region of intracranial:

(1) The neurological symptoms and signs after injury have typical characteristics of appearance from the intermittent;

(2) The main symptoms were progressive headache, frequent nausea and vomiting, and increased intracranial pressure with progressive deterioration of consciousness; GCS were all below 6-8 points;

(3) Unilateral pupillary change slowly or rapidly progressed to bilateral dilation, that is weakened or disappeared in response to light;

(4) Unstable vital signs with or without cardiopulmonary insufficiency;

(5) Acute sudural hematoma from supratentorial left or right frontotemporal parietal were accompanied to with or without brain contusion, and incomplete or complete disappearance of the cisterns ambiens, interpeduncular cisterns and prasellarcisterns, and mesencephalon and pons of deformation or cerebellar tonsil down shifting;

### 5) Occurrence time of brain cascade hernia and pre-hernia intervention:

The cerebral hernia of early were arrived at the scene in 15 cases that is occurred in 1.5 hours. Brain hernia were occurred between 1 and 1.5 hours after cerebral trauma in 10 cases. Operation time were used after prehospital treatment for 36 cases of pre-hernia: either 30cases within 2 hours of onset and 6 cases within 2.5 hours after the onset of hernia.

### 6) Indications for prehernia tervention of cerebral cascade hernia

(1) Progressived changes in the state of consciousness accompanied by increased respiratory sputum and inability to

cough up;

- (2) Bilateral anisocoria with persistent light response;
- (3) eyelashes reflex and head-eye reflex were present;
- (4) unilateral limb motor weakness gradually involved bilateral;
- (5) The progressively increases of intracranial pressure.

#### 7) Pre-treatment of hernia

Pre-hernia treatment, such as the were used to lateral position hyperventilation, prevention of vomit aspiration, cleaning respiratory secretions, rapid establishment of artificial airway, tracheal intubation or minimally invasive tracheotomy, emergency high-flow oxygen supply, i.e., and which appropriate 20% mannitol dehydrating agent and appropriate fluid replacement. Vital signs were closely monitored to prevent the rapid aggravation of turbulence during the hospital transportation examination. After CT examination in the green area, the patients were hospitalized craniotomy as soon as possible, and the complicated fatal injuries were temporarily treated at the same time.

#### 8) Surgical treatment

The patients with cascaded cerebral hernia were transferred to surgical therapy immediately of active treated. Receive surgical cured that were within 1.5-2.5 hours after admission in 36 cases. Sometime, intraoperative drainage were performed in cisterns of interpeduncular or annular that is used replacement catheterization of cisterns. Diagnosed cerebral cascade hernia were underwent craniotomy of immediately after hospitalization in 25 cases.

## 3. Results

The patients were followed up for 6-12 months. According to Glasgow Outcome Scale (GOS), there were 25 cases in the brain cascade hernia group, with 4 cases of 1 score, 3 cases of 2 score, 4 cases of 3 score, 5 cases of 4 score, and 9 cases of 5 score. Sometime, there were 36 cases in the pre-hernia group, with 2 case of 1 score, 3 cases of 2 score, 3 cases of 3 score, 11 cases of 4 score, and 17 cases of 5 score.

Characteristics of different sequential brain tissue displacement have been exhibited by found in two groups is patients. Among them, 25 patients of cascading cerebral hernia were all found to have I-V stages manifestations. Surgically treated for all emergencies.

However, Symptoms and signs of I-III stage were manifested for the pre-hernia which is cascade cerebral hernia in 36 cases. Sometime, And the performance in stage IV before hernia is not as significant as that in patients after with cascade cerebral hernia. Only symptoms and signs of cerebral hernia stimulation in stage V. Above all of these for changes are opportunities which early active intervention on time that is gradually increasing GOS during clinical recovery which improved survival and reduced mortality rate and the mortality rate is only in 6 cases.

## 4. Discussion

### 1) Definition of pre-hernia of cerebral cascade hernia and

### concept of pre-hernia treatment

The brain hernia cascade referred to by the patients in this group is due to the progressive of brain edema caused by the supratentorial acute subdural hematoma and by occupying effect of hematoma the progressive increase of intracranial pressure, and the gradual formation of intracranial hypertension, resulting in brain tissue displacement through the supratentorial hiatus, fissure, or tentorial foramen, respectively, and protruding from the opposite side contralateral or supratentorial or upper cervical spinal canal, and forming a sequential early cascade cerebral hernia formation characteristics. This article all its traumatic brain cascade cerebral hernia. while The brain hernia cascade referred to by the patients in this group is due to the progressing of brain edema caused by the supratentorial acute subdural hematoma, and by occipital effect of hematoma, the progressive increase of intracranial pressure, and the gradual formation of intracranial hypertension, resulting in brain tissue displacement through the supratentorial hiatus, fissure, or tentorial foramen, respectively, and protruding into the contralateral or supratentorial or upper cervical spinal canal, and forming a sequential early multiple cerebral hernia formation characteristics. This article all its traumatic brain cascade cerebral hernia. while During this period, emergency measures must be taken to avoid secondary compression and downward displacement of brain stem caused by further development. Patients in this group strictly followed the time of pre-hernia treatment, and first adopted measures to reduce intracranial hypertension and maintain cardiopulmonary function and support vital signs before hernia. Therefore, it is reported that pre-hernia treatment can significantly reduce the mortality and morbidity of the patients.

### 2) Pathological changes of cerebral hernia cascade

Traumatic cerebral hernia is caused by the progressive of brain edema caused by intracranial hematoma or brain contusion and laceration, which there were no hematoma volume of significant increase. First once, Through clinical observation were irritated symptoms of cascade cerebral hernia occur at this time by the group. when supratentorial of hematoma side with compartment cavity were changed which causes the pressure of the ipsilateral compartment cavity to be higher than that of the adjacent cavity. Therefore, the compartment cavity with high pressure is compressed to the part with low pressure, especially the tentorial area with low pressure difference which causes the displacement of the brain tissue. The formation of cascade cerebral hernia in this group was mainly caused by unilateral supratentorial hematoma and brain edema, which were increased the pressure in the cavity of the affected from disease side, resulting in the displacement of the cuneate gyrus that through inferior edge of the falx to the falx to the corresponding side, which was the key factor for the early formation of cascade hernia. The pressure then transmits to the gyrus of sulcus or hippocampal of and makes it protrude to between the tentorium fissure and the brain stem, which is blocked the cerebrospinal fluid channel from the intratentorial cavity to the supratentorial cavity. However,

when due to increased of intracranial pressure in the supratentorial, it must transmit to the lower pressure of the tentorial foramen area, so that the midbrain is compressed into a pee-like shape and displaced downward through the tentorial foramen, compressing the medulla oblongata. As a result, the pressure difference between the supratentorial and to tentorial cavity by increased, and even the water fall effect occurred, resulting in the rapid downward displacement of the cerebellar tonsils, and even into the superior cervical spinal canal, resulting in a rapid sequential brain multi-stage displacement process in a short period of time. This is the patients of in this group which were main clinical pathological feature of the cascade cerebral herniation. However, the progressive deterioration of the cascade hernia resulted in progressive fixation of the bilateral foramens, weakened response to light, unstable vital signs and heart failure or pulmonary insufficiency and other clinical manifestations. By observing the occurrence and development process and corresponding clinical manifestations of the cascade cerebral hernia in this group of patients, especially. Brain pathological morphology were observed for characteristic of cascading cerebral hernia by imaging, it was found that there was an intervaracteristic change from the cerebral falx hernia to the foramen magnum hernia. This is should be distinguished from the central hernia by the axial downward displacement of the midline structure of the brain [8], and also from the multiple cerebral hernias forme by the off-center structure and its axial downward discement [9]. Sometime, Suthalamus is accompanied with changes of inferior displacement. However, In this group of patients, the formation process of cascade cerebral hernia has obvious sequential characteristics and the occurrence interval performance is completely different. To improve the quality of life and reduce the mortality, clinicians should carefully the evolution characteristics, actively strive for early intervention treatment, block the sequential brain damage, shorten the time of preoperative examination and preparation as far as possible, and perform surgical treatment as soon as possible.

### 3) Characteristics of clinical manifestations

Hematoma, brain contusion and other space-occupying lesions were caused by craniocerebral injury often cause single cerebral hernia, among which tentorial notch hernia is the most common, followed by cerebral falx hernia, etc., and may also occur in combination with each other [10]. The occurrence characteristics of these cerebral hernias are different from those of cerebral hernia cascade in this group. Thcharacteristics of these cerebral hernias are different from the of the cerebral hernia cascade in this group. The typical cerebral herina cascade is usually caused by unilateral supratentorial traumatic acute subdural hematoma with hyprogressive mass caused by brain contusio and laceration, but patenits with acute subdural hemetoma of bilateral is realled rare. In the treated of this patients, it was found that the progresived of cascade cerebral hernia could be divided into five stages:

*stage I: Stimulation stage:*

Secoary brain edema due to supratentorial hematoma and cerebral contusion and laceration; The progressive increase of intracranial pressure is the key factor leading to the cascade of cerebral hernia, so the patient's state of consciousness deteriorates slightly, the pupil of the affected side is relatively narrow, and the patient is sens it to light.

*Stage II: The retraction to the contralateral shift stage:*

Due to the progressive aggravation of brain edema induced by hematoma or concomitant brain contusion and lacerationthat but slightly, the intracranial pressure was slightly unbalanced, resulting in a small pressure difference, only leading to the cingulate gyrus to the contralateral shift, and through the foramen below the falx cerebri, to the contralateral c avity, resulting in clinic aldisturbance of consciousness and slow progress There was no progressive change in pupils, vital signs were stable, and the state of consciousness was progressively aggravated. The cerebral falx changed in a linear arc, the mid d le cerebral artery and temporal lobe shifted backward, and the anterior horn and ody of the latera entricle crossed the midline and shifted to the opposit side.

*Stage III: downward shift of the sulci gyrus or hippocampal gyrus:*

Due to the samall pressure dince in the notch area of the tentorium, under the condition of progressive and gradual increase of cerebral pressure, the brain tissue contions to shift downward, resulting in the temporal lobe of the sulci gyrus or hippocampal gyrus from fissure of the tentorium downward and the mesencephalon compressed, so that the clinic al appearance of disturbance of consciousness progress rapid ly, headache, nausea and vomiting frequent; The irritation of the pupil became smaller and became progressive dilated on one side, and the response to light decreased gradually. The muscle strength of the contralateral upper and lower limbs decreased progressively, the muscle tension increased, and pathological signs appeared, and the instability of vital signs began to occur. Imaging examination showed that the space-occupying effect extended more than before, resulting in the displacement of the temporal lobes ulcus gyrus or hippocampal gyrus, the disappearance of the cisterns, and significant compression deformation of the midbrain. At the same time, it was shown that the brain edema was obvious due to the effect of the two, and the obstruction of the hippocampal gyrus within the fissure obstructed the supratentorial and infratentorial cerebrospinal fluid circulation, and the continuous increase of intracranial pressure was more obvious.

*Stage IV: Diencephalic shift stage:*

Intracranial hematoma and with brain edema were occupied space secondary to shift of diencephalic downward. The Serious damage were caused the midbrain or lower brainstem which is occurred to dysfunction of metabolile, endocrine, and autonomic nerve system. That is exacerbates coma of progressive deterioration in traumatic condition. Sometime, dysfunction manifestation of psychanopsia and nervs after still diencephalic leion.

*Stage V: Cerebellar tonsillar translocation stage:*

Displacement of the cerebellar tonsil or medulla oblongata were cascaded which from supratentorial and infratentorial brain tissue displacement. There were the brain tissue of posterior displacement which were through physiological gaps that is pushed into for the spinal canal of the cervical vertebral which Causing compression were important structures of brain tissue, vessels, or nerves, et al. The divided into two types were primary and secondary of the cerebellar tonsillar translocation. The secondary the cerebellar tonsillar translocation is referred for in this group. There were supratentorial brain tissue to sequential displacement that is a main factor by formatted.

#### 4) Clinical efficacy and prognosis

The mortality were declined at 39 to 27% from severe head injury in the United States [11]. However, in this article, the cascade cerebral hernia were explored by the author belongs for the category of severe brain injury which is with higher mortality rate. So, pre-hernia treatment of cerebral hernia as become a topic of concern for scholars. However, we believed that it is important to maintain airway patent for to prevent cerebral hypoxia and maintain stable vital signs and appropriate reduction of intracranial pressure before operation, which is get a recovery of good very important after brain injury surgery. This is because the secondary brain injury caused by the cascade brain hernia is often more serious than the primary injury, which is the key to prevent the respiratory and circulatory failure of the cascade brain hernia. Patients of Thirty-six were with prehernia stage of cascade hernia that intervention measures were taken before hernia, which belonged to I-III or IV stage changes in this group. Therefore, early diagnosis should be made after cerebral hernia occurs treatment [12]. Therefore, the treatment of experienced doctors in the early stage or on the way to lose time is very important for the curative effect of surgery after hospitalization. we should strive to actively adopt various rescue measures within 30 minutes to 1 hour, first of all, prevent and remove the state of cerebral anoxia as early as possible, remove the obstruction of the respiratory tract and the need for emergency intubation should be timely, and the need for tracheotomy should be implemented quickly, so to relieve the paralytic obstruction of the respiratory tract, keep the respiratory tract unobstructed, and achieve the pure of adequate oxygen supply. It is beneficial to protective effective function of cardiopulmonary function and circulation, avoid the occurrence of hypotension and cerebral hypoperfusion pressure, and reduce the factors of uncontrolled intracranial pressure during operation, which is one of the key factors to reduce the mortality. Therefore, when respiratory and circulatory function is impaired, acute respiratory central paralysis is easy to occur, resulting in expectoration weakness or difficulty, with secondary to severe hypoxia of brain cells. Hypoxia lasts for a long time, which can cause to damage in the molecular structure of nerve cells under hypoxic conditions, aggravate the progress of brain edema, and cause hypoxia to have a greater impact on the myocardium. At the same time, a catheter was placed in the cisterns around of midbrain during the operation before

the occurrence of cascade cerebral hernia, and saline irrigation was used for the supratentorial and infratentorial cerebrospinal fluid circulation and relieve the compression damage of the brain stem by promoted [13]. Also, better results were achieved by using drainage tubes placed in the cerebral the cisterns in this group. Yet, Decompressive of minimally invasive (tentorial edge of uncut) and thorough of hematoma evacuation were reduced of intracranial pressure which during surgery is crucial for the therapeutic effect [14]. Therefore, we found that the best time for pre-hospital treatment of cascade hernia is the appropriate amount of fluid in the clinical stage I-III or IV stage, and an important factor to ischemia and hypoxia of improved by sufficient effective cerebral circulation. Pre-hospital maintenance or cardiopulmonary resuscitation as soon as possible can no to eliminate the inducing factors of increasing brain water, but also reduce brain edema and alleviate the factors from intracranial pressure of increase. It can also remove the obstacles of venous return, which is conducive to venous return, thereby by indirectly reducing intracranial pressure. Appropriate application of mannitol dehydration can effectively reduce intracranial pressure, reduce the degree of secondary brain edema in brain tissue, alleviate the secondary damage before the herniation of the cascade brain hernia, and win the best time for hospital treatment. The pre-hospital intervention can significantly reduce the mortality and disability rate of the patients with pre-hospital hernia, while the mortality and disability rate of the patients with hospital hernia after operation and related treatment are lower.

This is because pre-hospital intervention can not only prevent the progression of the disease, so as to prevent the secondary damage of the brain and life support neural structure, but also acquire the excellent therapy also, which is the basis for better prognosis. The patients were with cerebral cascade hernia in hospital and appropriate methods of transport, create a better recovery after surgical therapy. This is not entirely the same as the treatment of simple tentorial hernia [15]. It was observed that the vital signs of the cascade hernia were unstable before the hernia, among which the increased rate and rhythm of respiratory, and disordered most easily occurred, followed by the change of the heart rate. Adequate oxygen supply, increase oxygen tension, rapidly of airway ventilation, appropriate amount of dehydration in the pre-hospital by adopt of first method. In short, The imaging changes of cerebral cascade hernia and pre-hernia are of great significance for the pre-hospital treatment measures and prognosis.

## 5. Conclusion

Cascade cerebral hernia is formed from the traumatic acute subdural hematoma and cerebral edema which on one side of the supratentorial region. Progressive deterioration can not bring a patient out of danger of some patients after the onset of the disease that is related to late treatment and severe injuries. However, the sequential displacement of

brain tissue were occurred as result from hematoma occupying and brain edema. Pre-hospital intervention extremely important that stage of I- III or IVstage of hernia is golden time. Sometime also found a relationship of outcome with time from onset of coma to craniotomy, with patients operated on at  $\leq 2$  hours after clinical deterioration having significantly better outcomes than those operated on at  $>2$  hours, and which is concern pre-hospital of intervention. however, Death is occurred in 6 cases at  $\geq 2$  hours.

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