



Successful Management of Hydrocephalus Following SAH with Early Surgical Intervention

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Abstract

Subarachnoid hemorrhage (SAH) is more than a simple vascular event; it is a systemic pathological condition of the brain. While surgical advances have increased survival rates, survivors continue to face a wide range of cognitive deficits and personality changes. This article reviews the mechanisms of early injury (EBI), delayed ischemic stroke (DCI), and the specific profile of neuropsychological deficits in these patients.

Introduction

In 2011, I was sitting in the office of the hospital where I worked. Through the window, I saw an elderly man, who appeared to be about 55 years old. His gait, which was crooked and wobbly, struck me as unusual. I immediately visited him and admitted him to the hospital as a patient with balance problems. After the necessary examinations, it was determined that the patient had hydrocephalus. Without any prior background, which I and my other colleagues who examined the issue, finally decided to put the patient on a cerebral shunt. This was done, which was very successful. Since then, I have not personally encountered a case of hydrocephalus. After 17 years, when I saw this man in the inpatient ward of the hospital where I am, I only realized from the severity of the headache and the condition of his face that it was an emergency. Immediately, the initial examinations, which included a CT scan of the brain and then a CT scan with contrast and MRI of the brain, were performed. To my surprise, I found that the patient had a subarachnoid hemorrhage (SAH). Initial supportive measures were started for him. After ten days, when the patient's condition was stable, he was discharged from the central hospital and transferred to our hospital for further treatment and recovery. After three days, the patient's relatives said that the patient had encountered three new problems:

- Incontinence of urine,
- Inability to stand,
- Inability to walk,
- And most importantly, symptoms of psychological disorders.

For me, who have known the patient for more than three decades, all these symptoms were new. Even when the subarachnoid hemorrhage began, his only main complaint was headache, and no other brain symptoms. And all these symptoms had occurred later, the next day...

According to this patient's medical history, he had undergone several P.T.C.A. procedures in the past years due to heart problems, and he had no history of vascular abnormalities such as cerebral aneurysms.

Aneurysmal subarachnoid hemorrhage, despite comprising only 5% of all types of strokes, imposes a heavy economic and social burden due to its occurrence at a younger age and high rate of disability. Unlike focal ischemic strokes, in SAH we are faced with a widespread injury (Diffuse Brain Injury) that targets the superior functions of the brain due to the diffusion of blood into the middle cerebral space and the sudden increase in intracranial pressure.

Pathophysiology

(Review of EBI and the role of neuroinflammation).

Subarachnoid hemorrhage (SAH) is not just a simple vascular event, but the beginning of a series of complex pathophysiological reactions whose long-term cognitive and behavioral consequences are sometimes more severe than the initial physical injury.

SAH is mainly caused by the rupture of saccular aneurysms (in 85% of cases) in the circle of Willis. However, from a scientific point of view, we are faced with two phases of injury:

A. Primary brain injury (EBI):

In the first minutes, a sudden increase in intracranial pressure (ICP) leads to a decrease in cerebral perfusion pressure (CPP) and transient global ischemia. This phenomenon can cause disruption of the blood-brain barrier and apoptotic cell death.

B. Secondary brain damage:

which includes vascular vasospasm (usually days 4 to 14) and impaired microcirculatory

Clinical manifestations

(from Hunt & Hess grading to cognitive manifestations).

Complications of SAH can be divided into three categories, each with a different neuropsychological outcome:

- **Hydrocephalus:**

impaired CSF absorption leading to enlargement of the ventricles and pressure on frontal projection fibers (the main cause of impaired processing speed).



- Electrolyte disturbances:

Especially hyponatremia due to cerebral salt wasting syndrome (CSW), which impairs consciousness and acute cognitive functions. This is a CT scan of the patient before the shunt operation

- Subclinical seizures:

Abnormal electrical activity that may not be visible but causes cognitive deterioration of the patient.

Pathophysiological phases (The Two-Hit Model)

A. Early brain injury (EBI)

This injury occurs in the first 72 hours.

- Transient Global Ischemia:

At the moment of rupture, intracranial pressure (ICP) suddenly rises to the level of arterial blood pressure, leading to a temporary cessation of cerebral blood flow (CBF).

- Blood-brain barrier (BBB) damage:

Infiltration of inflammatory cells and formation of vasogenic edema.

- Cell death:

Induction of apoptosis in neurons and endothelial cells due to glutamate toxicity and oxidative stress.

B. Delayed Cerebral Ischemia (DCI)

This phase, which peaks between days 4 and 14, is the most challenging for neuropsychologists.

- Microthrombosis:

Formation of microscopic clots in small vessels that are not visible on angiography but cause small, scattered infarcts.

- Cortical Spreading Depolarization (CSD):

Waves of depolarization that travel across the cortex, depleting energy and worsening ischemia. This phenomenon is directly related to long-term cognitive deficits.

Complications:

A. Acute Complications:

- Hydrocephaly:

Due to impaired reabsorption of cerebrospinal fluid (CSF) by the arachnoid granulations (Acute vs. Chronic).

- Seizures:

Which increase the risk of secondary injuries.

- Dysautonomia:

Excessive activity of the Pathology leading to cardiomyopathy and pulmonary damage.

B. Neurocognitive sequelae:

- Global Cognitive Impairment:

Even in patients with good scores on the Glasgow Coma Scale (GCS), impairment in memory, attention, and executive functions is seen in 60% of cases.

- The "Invisible" Disability:

The patient may walk and talk, but due to damage to the fronto-striatal circuits, may not be able to return to work or manage interpersonal relationships.

Other Clinical Panels

A. Neuropsychological Profile and Behavioral Changes:

This section is the core of neurology. Survivors of SAH usually have chronic deficits in the following areas:

B. Executive Dysfunction:

Due to the high sensitivity of the frontal lobe to changes in pressure and inflammation, patients have serious problems on tests of response inhibition (Stroop) and mental flexibility.

C. Memory and Learning:

Anterior communicating artery (ACoA) damage often results in a specific amnesic syndrome. These patients may suffer from Confabulation, i.e. filling in memory gaps with false but unconscious information.

D. Mood and Personality Disorders:

- Apathy:

Decreased motivation due to damage to the mesolimbic dopaminergic circuits.

- Depression & Anxiety:

Not only a psychological reaction to the disease, but also due to structural changes in the amygdala and cingulate cortex.

- Neuropsychological Profiling:

(Details of cognitive deficits differentiated by aneurysm area).

- Rehabilitation & Future Perspectives:

(Role of cognitive rehabilitation).

Long-term outcomes and quality of life (Outcomes)

- Fatigue:

One of the most common complications that stems from neuroendocrine changes (hypothalamic-pituitary axis).

- Mental disorders:

High prevalence of depression (up to 35%) and anxiety.

- Personality changes:

Caused by damage to the Ventromedial Prefrontal Cortex (VMPFC), especially in anterior communicating artery aneurysms (ACoA).

- Memory disorders

Due to the different actions and reactions that the patient showed in dealing with different people and family members, in order to be able to assess his condition and reality, we put two memory assessment tests on the agenda:

- A. MoCA (Montreal Cognitive Assessment) was performed and on day 7, the score was estimated to be around 22. Especially since the patient did not have the patience to answer the MoCA questions, but receiving this score was a source of joy for us, considering his age and the problems he had encountered. Interestingly, when we re-tested him 25 days after the original incident and the shunt

surgery, the score had improved to 26.

B. MMSE test:

We also used the MMSE test for the same purpose in addition to MoCA, and the scores obtained were not qualitatively different from each other. The result was that these events that occurred in the brain field did not affect the memory function of our patient before and after the shunt surgery.

Treatment

Given that the patient did not have any serious complaints in the first days, especially in the initial movements, we decided to drain his cerebrospinal fluid a little with Tab Acetazolamide, but I must say that we were not successful at all, and incidentally, in the following days, symptoms of hydrocephalus It became very obvious that the triad of hydrocephalus, i.e. slow and point steps, along with urinary incontinence without diabetes or other causes, confirmed that the patient's problem was progressing towards aggravating clinical symptoms. I realized that the patient had deteriorated psychologically and was experiencing hallucinations, so I decided to raise the issue with the neurosurgeons, and when we did, my colleagues recommended brain shunt surgery as soon as possible.

Rapid surgical intervention

“Rapid surgical intervention (Surgical Shunting) is not only a life-saving measure to reduce intracranial pressure, but also a neuro-protective measure to prevent permanent dementia in SAH patients. There is a direct correlation between the time of shunt placement and the potential for return of executive functions and walking.”

We were faced with a key question: whether the patient's problems will improve after shunt surgery? Has the patient been able to say his first meaningful words without repetition (Perseveration) after surgery? This is the first sign of success. The function of the shunt is at the superior level of the brain.

A. Surgical management and the role of timing in shunt placement

In this study, due to the occurrence of secondary hydrocephalus following subarachnoid hemorrhage (SAH), a decision was made to surgically insert a ventriculoperitoneal shunt. The key to the success of this case was the speed of diagnosis and transfer to the operating room.

B. Details of the surgical procedure:

- Preoperative:

Immediately after confirming CSF flow obstruction on repeat imaging and failure to respond to temporary drains, the patient was a candidate for permanent shunt placement.

- Surgical technique:

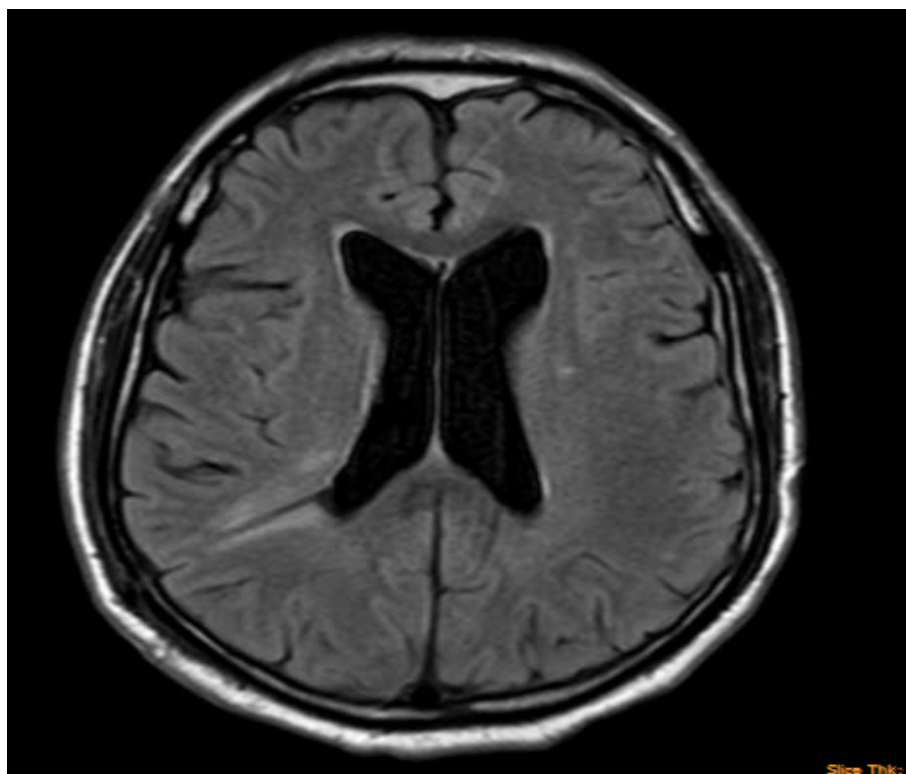
The surgery was performed under general anesthesia and in full compliance with sterile protocols. The ventricular catheter was inserted through a burr hole in the anterior horn of the lateral ventricle. The shunt valve pressure was adjusted based on preoperative radiological indicators and intracranial pressure to prevent over-drainage.

- Shunt route:

Catheter The distal end was directed subcutaneously into the peritoneal cavity to allow for sustained drainage of cerebrospinal fluid.

- Timing Impact Analysis:

The success of the treatment in this case was directly related to the short time window between the diagnosis of hydrocephalus and the surgical procedure.



- Surgical Conclusion:

This case showed that shunting, if performed as a “preemptive” treatment and immediately after the initial SAH condition is stabilized, can change the patient’s prognosis from a critical condition to a complete and functional recovery.

Here is the CT scan image of the patient after one week cerebral shunt surgery

If precise information on the type of valve (e.g., programmable or fixed) or specific postoperative radiological parameters (e.g., reduced ventricular diameter on CT scan). This emphasizes that the rapid intervention not only saved the patient's life, but also prevented the occurrence of permanent neurological deficits.

Clinical Recovery and Follow-up:

The most striking feature of this case was the speed of the patient's recovery. Despite the complications common to subarachnoid hemorrhage and the need for cerebral shunting, the patient returned to a completely normal functional status (Baseline status) only one week after surgery.

This rapid recovery can be attributed to two main factors:

- A. Positive physiologic response to cerebrospinal fluid drainage and intracranial pressure readjustment.
- B. Lack of permanent structural damage due to timely surgical intervention, which did not allow the hydrocephalic process to destroy the brain parenchymal tissue.

On the seventh postoperative day, no motor, speech, or cognitive impairment was observed, and the patient was ready for discharge with a full consciousness level (GCS 15/15). This experience suggests that the recovery period in SAH patients can be significantly shorter than the average if hydrocephalus is managed promptly.”

The critical impact of timely management in the treatment of hydrocephalus due to subarachnoid hemorrhage (SAH) was investigated. Based on the findings of this study, the following conclusions were reached:

- Importance of early intervention:

The remarkable success in this treatment case showed that performing shunt placement surgery within the shortest time after the onset of hydrocephalus symptoms is a determining factor in the patient's prognosis. Rapid intervention prevented the intracranial pressure from becoming a chronic and irreversible injury.

- Rapid functional recovery:

The patient's complete return to normal within just one week was beyond the usual expectation in similar cases. This proves that the potential for regeneration and return to baseline function is very high if the brain parenchyma is not subjected to prolonged stress.

- Clinical Recommendation:

This clinical experience suggests that in patients with SAH, careful and continuous radiological and clinical monitoring for early detection of hydrocephalus should be a priority. Shunt surgery should not be considered as a last resort, but rather as a “timely and targeted” treatment to prevent permanent neurological deficits.

Discussion

The findings of this report indicate that rapid return to normal is directly related to time management. In hydrocephalus caused by SAH, delayed CSF drainage can lead to irreversible ischemic damage to the brain parenchyma. In this case, early intervention prevented the occurrence of chronic pathological changes. Complete recovery within a week suggests that neurons have a high potential for functional recovery if hydrostatic pressure is rapidly relieved. This reinforces the “Time is Brain” paradigm in shunt surgery.

A. New paradigms in vasospasm and DCI

Today, the focus in the literature has shifted from “macroscopic vasospasm” to delayed cerebral ischemia (DCI).

- Change of approach:

It has become clear that even without visible vasospasm on angiography, a patient may develop DCI.

- Cause:

Microthrombosis, cortical inflammation, and spreading cortical depolarization, which, like silent electrical earthquakes, deplete neuronal energy stores and lead to secondary cognitive deficits.

B. Neuropsychological profile in SAH survivors

Even in patients who have recovered physically (Grade I/II on the Hunt and Hess scale), persistent cognitive deficits are observed, which will form the core of your paper:

- Executive Functions:

Due to the sensitivity of the frontal lobe to changes in pressure and inflammation, response inhibition, mental flexibility, and planning are usually impaired.

- Working and verbal memory:

Damage to the limbic system and basal forebrain circuits is particularly common in ACoA (anterior communicating artery) aneurysms.

- Information processing speed:

Psychomotor slowness is one of the most persistent complaints of survivors.

C. Psychological aspects and quality of life (QoL)

A very valuable point is the examination of personality changes and mood disorders:

- Apathy syndrome:

Often confused with depression but is caused by damage to the reward circuits and striatum.

- PTSD after SAH:

The sudden and fatal nature of this condition traumatizes many patients, which in turn negatively impacts

cognitive function.

Conclusion

This report confirms that in the management of hydrocephalus due to SAH, shunt surgery should not be a secondary option, but can be used as a “preemptive” treatment. Prevent chronic defects. Intelligent diagnosis and prompt surgical intervention are the keys to the patient's complete return to normal life in the shortest possible time.

I consider it my duty to sincerely thank and appreciate all the efforts and efforts that my colleagues made in the examination, decision-making, and ultimately the successful brain shunt surgery for this patient:

Professor Dr. Ali Mahmmmed Ridwan, neurosurgeon

Dr. Elvis Darwish, and his team neurosurgeon who performed the surgery

Mrs. Dr. Mimosah Spahiu, neurologist

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