Non-endoscopic minimally invasive evacuation of intracerebral hematoma (ICH): A case report

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ABSTRACT

Spontaneous intracerebral hemorrhage (ICH) is one of the most serious causes of stroke, leading to high rates of disability and mortality. In addition to intensive medical treatment, surgery may help to improve the prognosis in patients with ICH. A rapid reversal of coagulopathy is essential in these patients, although it may be difficult to achieve in various bleeding disorders. In such cases, when surgery is needed, a minimally invasive approach is recommended. In this case report, we described and shortly discussed the evacuation of ICH with a minimally invasive non-endoscopic surgical technique.

KEY WORDS: Intracerebral hemorrhage; ICH; bleeding disorders; hematoma evacuation; minimally invasive surgery

INTRODUCTION

Spontaneous intracerebral hemorrhage (ICH) is frequently encountered in clinical practice, accounting for 15-20% of all strokes [1,2]. Despite the advances in intensive care and the treatment of hematoma, the mortality and morbidity of ICH still remain very high, with the documented mortality rate reaching >40%. About 10-15% of the survivors remain fully dependent [2-4].

The role of surgery in the treatment of ICH is still controversial. The advances in neurosurgical techniques, including endoscopy and neuronavigation, may help to improve the prognosis of ICH patients. Minimally invasive techniques have been used in hematoma evacuation, to reduce the surgical trauma injury as well as to maximize the removal of hematoma [5]. Several studies reported that some patients with ICH may also benefit from the evacuation of hematoma with those minimally invasive procedures [4,6,7]. The possible surgical options include endoscopy and stereotaxy, sometimes with a catheter inserted into the hematoma through which fibrinolytic drugs are injected, to accelerate the resolution of hematoma [3]. Another approach is the use of a minimally invasive non-endoscopic technique, which may be utilized when an endoscope is not available or in those patients at high risk due to the concomitant diseases, especially coagulopathies. The evacuation of a primary ICH with such technique is shortly discussed [3,5].

CASE REPORT

A 59-year-old man was admitted to the emergency department due to progressive left arm and leg weakness. The symptoms started with acute nausea and slight headache a few hours before the admission, and gradually deteriorated. The headache was getting more intense and dysarthria developed. Neither loss of consciousness nor seizures were reported by the relatives. The patient had no signs of possible head trauma nor the history of arterial hypertension, diabetes, hypercholesterolemia, or other risk factors related to cerebral atherosclerosis. In the past medical history, severe thrombocytopenia with hypersplenism and advanced alcoholic liver cirrhosis were documented.

On the neurological examination, initially, the consciousness was intact. The patient was stable, hypertensive (blood pressure [RR] 150 mmHg), and the cardiovascular function was regular with a normal heart rate. The speech was slow and dysarthric. The Glasgow Coma Scale (GCS) was scored as 15. The modified Rankin score at admission was 4 (before the admission it was 0). A deviation of the head to the right was noted. Otherwise, the testing of cranial nerves was normal. No meningeal signs were observed. There was left-sided hemiplegia with the extensor plantar response on the same side. The muscle tone was reduced and the reflexes could

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not be elicited on the left side. The right extremities were normal. No tremor was observed. Walking was not possible. The neurological status started to deteriorate quickly. The headache intensified and the level of consciousness declined rapidly, below 8 according to the GCS. The blood pressure rose to 190 mmHg/96 mmHg, and the effect of antihypertensive drugs was small. No changes in the cardiovascular function were documented. Sedation and intubation with mechanical ventilation were required, and thereafter, the blood pressure dropped to 160 mmHg/80 mmHg. The pupils were symmetrical and responsive.

A computed tomography (CT) scan of the head revealed an extensive ICH of 7 cm in diameter and of mixed density. Haematocephalus of the right lateral ventricle was also present (Figure 1). The shift of the brain due to cerebral edema was evident as was the compression of the subarachnoid space of the right cerebral hemisphere. There were no signs of the compression of the brainstem or posterior fossa. The CT angiography was negative, thus classifying the hematoma as a primary one. The laboratory findings showed completely deranged coagulation and aggregation values as a result of liver failure. The international normalized ratio (INR) and prothrombin time (PT) were lowered to 1.56 and 0.47, respectively. The platelet count was reduced to 33, and the platelet function tests were completely impaired.

The correction of coagulopathy was initiated immediately. Fresh frozen plasma, recombinant coagulation factor VIIa, prothrombin complex concentrate, vitamin K, and platelet plasma were given. However, the control hemostasis tests improved only slightly. As a result of extensive intracerebral bleeding and decline in consciousness, a surgery was recommended despite unfavorable laboratory results. A minimally invasive approach was chosen for the removal of ICH. The hematoma was located according to the standard craniometric points on the skull and based on the neuroimaging. Since the bleeding was extensive, superficial, and relatively easily accessible, there was no need for neuronavigation. Over the hematoma location in the right temporal area, a linear skin incision of 2 cm in length was made and the cranial bone was exposed. After local deperistiation, a burr hole of 1 cm in diameter was made with a compressed air-driven drill into the exposed bone in the middle of the skin incision. The dura was then exposed, coagulated, and cut. The approach to the hematoma was transcortical. Under the microscope, the brain cortex was first coagulated at the entry point. The entry point was kept as small as possible, to preserve the brain tissue, and the vessels running through the exposed part were kept intact. At approximately 5-mm depth, the hematoma was reached and then the liquefied blood, which was under pressure, was evacuated through this hole with an aspirator and bipolar coagulation forceps. The majority of the intracerebral hematoma consisted of liquefied blood with some blood clots that were all aspirated. The hematoma cavity was rinsed with saline, and the walls were inspected for bleeding. Minute hemorrhages and those in the cortical entry point were stopped with small patches of surgicel (hemostatic agent) and electrocoagulation. No bleeding was observed from the brain substance at the entry point nor in the canal or hematoma cavity. Despite the impaired hemostasis results, no excessive bleeding was encountered during the surgery. The intracranial pressure (ICP) probe was inserted thereafter, and the values remained normal during the course of treatment.

During the treatment, the correction of coagulation and hemostasis was essential, and with the great effort of the neuroanesthesiologists and hematologists, the values improved after a few days and remained stable (the INR and PT were 1.02 and 0.7, respectively; the platelet count was 110). The control CT scan showed a successful evacuation of the hematoma and the normal width of the ventricles (Figure 2). The sedation was gradually discontinued after a week. The patient was awake with persistent left-sided hemiplegia. The communication was possible; the speech and understanding were not affected. Although the neurological disease was stable, the complications of hepatic cirrhosis and pneumonia developed. In addition, liver and cardiac failure ensued. Despite all the intensive treatment and resuscitation, the patient unfortunately died after 1 month of the hospitalization.

DISCUSSION

The ICH is a disease with a grave prognosis. According to the statistics, only 48–65% of patients survive more than 1 month, and only 10% of these patients can further live independently [1,2,4]. The natural course of an acute ICH

FIGURE 1. A computed tomography of the head showing an extensive intracerebral hemorrhage of 7 cm in diameter and of mixed density (the hypodense areas indicate active bleeding), with haematocephalus of the right lateral ventricle. The brain shift is evident.
is progressive. After the initial irreversible tissue injury in or near the hemorrhage location, a combination of increased intracerebral pressure, edema, and excitotoxicity cause a secondary injury to the surrounding brain tissue [8]. The perihematomal inflammation that contributes to the mass effect, leads to acute neurological deterioration, and has even been associated with poor long-term functional outcomes. Much of this secondary process is thought to develop as a result of the mass effect of the new hemorrhage, as well as due to the toxicity associated with hematoma decomposition and release of inflammatory and free radical mediators [2,9].

Etiologically, ICHs may be classified as primary or secondary [2]. The majority of hematomas, about 80%, are primary. The main risk factor, here, is uncontrolled and long-term arterial hypertension, causing spontaneous rupture of small vessels. These ICHs are generally located deep in the brain substance, usually in the internal capsule and the basal ganglia. On the other hand, about 20% of hematomas are secondary. The typical locations are cerebral hemispheres, cerebellum, and pons. These hematomas are more frequently associated with anticoagulant therapy or coagulation disorders, tumors, and vascular abnormalities [8,10-12].

There are many issues related to when to operate ICH and about the outcomes of the operation. The treatment strategies include medical, surgical, and combined management, with the reduction of blood pressure and reversal of coagulopathy as well as treatment of cerebral edema [6,13,14]. The main causes of ICH are arterial hypertension and amyloid angiopathy. The hemorrhage is worse in the presence of bleeding diathesis of various etiologies, such as the anticoagulant therapy, trauma, and alcoholism [6,15]. Our patient had a clear history of decompensated liver cirrhosis with damaged hemostatic mechanisms. The bleeding was accelerated by impaired coagulation and aggregation. The blood accumulated rapidly, causing brain edema and shifting with the increase in intracerebral pressure and other clinical consequences.

The discouraging results of conventional hematoma evacuation may be attributed to the type of surgical approach. While standard craniotomy is effective in hematoma evacuation and maintenance of hemostasis, this approach frequently causes damage to uninjured brain over the hematoma. Minimally invasive surgical strategies have been designed to reduce this damage [9,15,18,19]. They include image-guided and frameless stereotactic procedures. These approaches are commonly combined with the use of thrombolytic agents and require more time for the preoperative preparation and hematoma evacuation. In addition, the hemostasis is difficult. The endoscopic-assisted evacuation of ICHs is gaining a growing attention as a suitable minimally invasive alternative due to its effectiveness and possibility to evacuate the hematoma immediately and completely, with good hemostasis [5,20,15]. Here, we presented such surgical approach. There is no difference in the placement or size of a burr hole, and the principle of hematoma evacuation is similar as in craniotomy. For thorough visualization, an endoscope is used, and in our case, it was supplemented with a microscope. The use of easily accessible microscope is especially important in urgent cases, where emergency surgery is needed [5,20]. When an endoscope is not available, minimal craniotomy or burr hole is an appropriate selection. The aspiration of blood and hemostasis is possible with the use of a microscope, providing a good control over the hematoma through the burr hole and corticotomy.

In our opinion, surgery has an advantage over conservative treatment. However, it must be performed safely [8,19]. If it is not possible to reverse the bleeding disturbances completely and quickly enough, the minimally invasive approach is recommended. Due to the severe liver failure and consequent impairment in coagulation and aggregation, a classical surgical evacuation of the hematoma with craniotomy was not possible in our patient. The delay to improve hemostasis was also not possible, because of potential brain damage [5,16,20,21]. The best possible solution was a burr hole and decompression of the brain with the removal of ICH. With this technique,
the wound and bleeding were minimal as well as the cut to the bone, with the smallest injury to the overlying brain tissue and reduced operation time. Overall, it is a safe and efficient option when endoscope is not available.

An operating microscope is practically mandatory in modern neurosurgery, which greatly improved surgical possibilities and safety [22,23]. In addition to providing improved working conditions with excellent lighting, the operating microscope offers perfect visualization during the entire procedure and a three-dimensional view. Coaxial illumination and variable magnifications allow better identification of anatomical structures and easier recognition of tissues. Moreover, a microscope enables a comfortable working position for the surgeon and there is no need for other surgical instruments, such as retractors, which may cause additional, iatrogenic tissue damage [22-24]. During our operation, the microscope offered an excellent view of the hematoma cavity and control of the working canal, uncovering all hematoma remnants and bleedings from the brain substance, which were then stopped with electrocoagulation. Such extensive hematoma removal and accurate hemostasis would be impossible with a naked eye.

In our patient, this technique proved to be successful. It enabled to a complete evacuation of ICH and reduced the brain edema, as confirmed on the control CT and thorough continuous ICP monitoring. The prompt action and minimally invasive surgery were important factors that lead to the satisfactory neurological outcome. Nevertheless, there are several limitations to our case report. We reported a new approach for minimally invasive hematoma evacuation only in one patient, and larger series are required to confirm our results. Unfortunately, the patient did not survive the treatment, although this cannot be attributed to the surgery or neurological complications but instead to the multiple organ failure, as a result of pneumonia, liver and heart failure. Despite described limitations, in our opinion, this surgical technique is efficient, especially in patients with coagulopathy and comorbidities, and is a suitable alternative when an endoscope is not available.

CONCLUSION

Although spontaneous ICH is a well-known pathology, the treatment is not always clear and the outcomes are usually poor. Many factors may influence the prognosis, such as the condition of the patient, neurological status, location and the extent of the hematoma, presence of edema, concomitant diseases, and associated anticoagulation. In the case of a patient with numerous risk factors and emergency surgery, a minimally invasive surgery for intracerebral hematoma is warranted.

DECLARATION OF INTERESTS

The author declares no conflict of interests.

REFERENCES

Tomaz Velnar: Minimally invasive evacuation of ICH

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https://doi.org/10.1227/00006123-198110000-00007.


https://doi.org/10.1016/j.jcma.2014.08.013.


https://doi.org/10.1161/01.STR.31.10.2511.