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CLINICAL STUDY

Neuroendoscopy-assisted evacuation for supratentorial intracerebral hemorrhage versus conventional craniotomy: A comparative analysis of efficacy and outcome

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ABSTRACT

BACKGROUND: The endoscopic-assisted approach for intracerebral hemorrhage minimizes traumatization of the brain and allows direct access to hematoma. The study aimed to compare the results of the endoscopic-assisted evacuation for supratentorial hemorrhage versus conventional craniotomy.
METHODS: A retrospective study analyzed medical records of patients with supratentorial intracerebral hemorrhage treated at our Neurosurgical Department between January 2015 and December 2023. The study included 91 patients who underwent either neuronavigational-assisted neuroendoscopy (n=25) or conventional craniotomy treatment (n=66).
RESULTS: For conventional craniectomy procedures, the average residual volume was 10.1 ml, representing an average 82.1% hematoma evacuation efficacy. After endoscopically assisted procedures, the average residual volume was 3.9 ml, achieving a 92.5% hematoma evacuation efficacy. After the endoscopically assisted procedure, fewer than 10% of patients were revised for early or late complications (2 out of 25 patients), whereas after the craniectomy procedure, revision or the introduction of lumbar drainage for cerebrospinal fluid leakage was performed on 35% of patients (20 patients).
CONCLUSIONS: The endoscopic-assisted evacuation of supratentorial intracerebral hemorrhage is becoming preferred because of its efficacy, a small number of complications, and minor trauma for the patient *(Tab. 1, Fig. 3, Ref. 16)*. Text in PDF www.elis.sk
KEY WORDS: intracerebral hemorrhage, treatment, endoscopy.

Introduction

Intracerebral hemorrhage is bleeding within the brain parenchyma, commonly referred to as hypertensive bleeding. Bleeding typically occurs during daytime activities, rarely during sleep, and is associated with increased blood pressure or increased blood flow in the brain. Risk factors include age, with a significant increase in incidence after the age of 55, doubling every decade until the age of 80, where the risk is 25 times higher than in the previous decade. It is more common in men, and chronic alcohol abuse increases the risk up to 7-fold. Smoking increases the risk of ischemic cerebrovascular disease but likely does not increase the risk of bleeding. Other significant factors include liver diseases involving thrombocytopenia, coagulation factor disorders, and hyperfibrinolysis, considered a consequence of excessive alcohol consumption. Anticoagulant therapy with warfarin increases the risk of intracerebral hemorrhage by approximately 0.3–1.8% annually, with mortality rates reaching up to 65% after the event. Generally, the rupture of lenticulostriate arteries is considered the source of intracerebral hematomas, described by Charcot and Bouchard in 1868 (1). Based on the location of bleeding, hematomas are defined as thalamic, putaminal, lobar – also referred to as subcortical (Fig. 1).

Unlike ischemic strokes where the neurological deficit is maximal at their onset, the neurological deficit in intracerebral hemorrhages is generally characterized by gradual worsening over minutes to hours. Severe headache, nausea, vomiting, and progressive impairment of consciousness are the most common and earliest clinical signs. The most frequent prodromes are TIA-like symptoms, present in up to 50% of patients. The neurological state deteriorates concurrently with the localization of bleeding. Re-bleeding can contribute to the worsening neurological state, more common in the basal ganglia than in lobar hematomas. Within the first hour, a CT scan has documented and evaluated as "ultra-early" re-bleeding, accompanied by a worsening neurological state. Between 1–3 hours, there's an average volume increase of 30–40%, 16% between 3–6 hours, and 14% within 24 hours after the hemorrhage. Patients with confirmed re-bleeding and coagulopathy have a worse prognosis (2).

Computed tomography (CT) is readily available for diagnosing intracerebral hemorrhage and is a rapid examination

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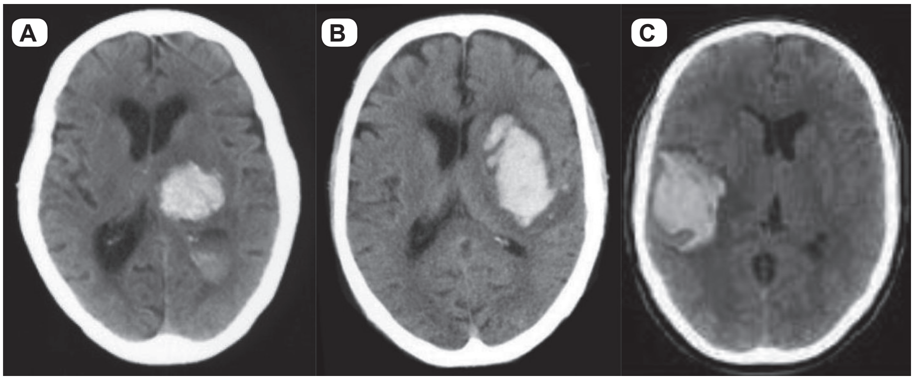

Fig. 1. Axial CT scans shows three types of localization of intracerebral hemorrhage. (A) thalamic, (B) putaminal and (C) lobar or subcortical hemorrhage.

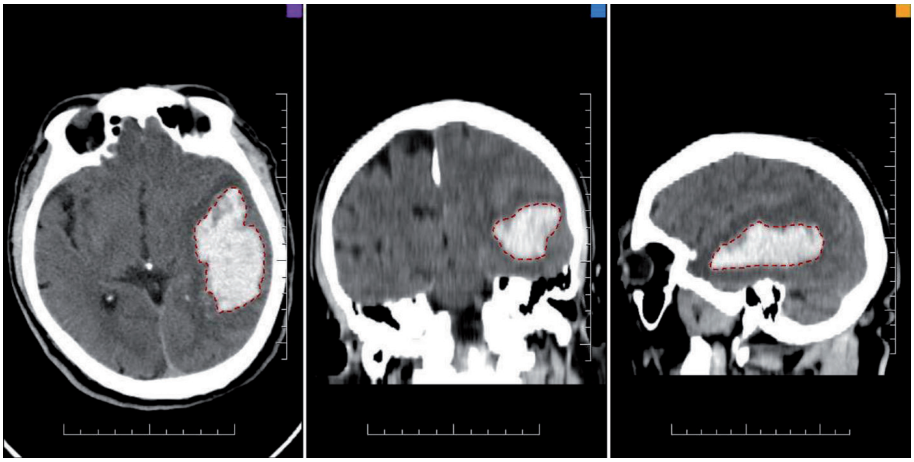

Fig. 2. Volume calculation of subcortical intracerebral hemorrhage using software. Volume of haematoma 52.4ml.

that visualizes the hematoma as hyperdense compared to the brain parenchyma. A common finding recognized is 'hot spots,' significantly correlating with hematoma expansion. These spots are detectable on CT contrast scans within 3 hours of hematoma formation, with a minimum size of 1.5mm, twice the density of the hematoma, and located at its periphery without association with brain vessels. Although mass effect is common, the hematoma usually spreads through the parenchyma and causes a lesser mass effect than the hematoma's size might suggest. Apart from localization, the size of the hematoma is crucial for indicating neurosurgical intervention or determining prognosis. Some radio diagnostic facilities calculate volume using software (Fig. 2), which is most accurate, but in cases where computation is necessary, a simple modified formula for ellipsoid calculation is employed.

$$V = \frac{AP\ x\ LATx\ HT}{2},$$

AP, LAT a HT are average diameters of hematoma in three dimensions of CT projection.

There isn't a clear-cut approach to managing intracerebral hemorrhage. Following diagnosis through CT and assessment of the clinical condition, treatment mostly occurs in intensive care units. Correcting hypertension can reduce the risk of re-bleeding but increases the risk of central nervous system (CNS) damage due to reduced perfusion. Achieving adequate perfusion is ideal, aiming for a mean arterial pressure (MAP) as pre-bleed levels. Recommended blood pressure is around 140/90 mmHg, while hypotension should be avoided (3, 4). In cases where there's neurological deterioration leading to stupor or coma, intubation becomes necessary, usually when the GCS drops below 8. It's crucial to maintain normal blood sugar levels and body temperature. Antiepileptic drugs are indicated for seizures, mainly occurring with lobular hematomas, but their prophylactic use isn't recommended (5). Many patients are on anticoagulants, associated with increased bleeding risk (6). The administration of corticosteroids is controversial due to potential side effects, including infection, gastrointestinal bleeding, and diabetes induction (7). Their use is only beneficial when outweighing risks, particularly in significant peri-hemorrhagic edema. Mannitol offers a possibility to lower intracranial pressure while correcting hypertension and can be combined with furosemide. Mannitol administration is safe but doesn't improve the outcome of conservative treatment. Its use should be considered for hematomas larger than 15ml, with no recorded negative impact (8).

Surgical treatment can reduce morbidity and mortality, decrease the development of edema, or necrosis caused by the mass effect of the hematoma. In ICH treatment, there's no unified global classification, clear-cut recommendations for surgical treatment, or consensus on the timing of the procedure (9).

Material and methods

A retrospective study analyzed medical records of patients with supratentorial intracerebral hemorrhage treated at our hospital between January 2015 and December 2023. The study included 80 patients who underwent either neuronavigation-assisted neuroendoscopy (n=25) or conventional craniotomy treatment (n=66).

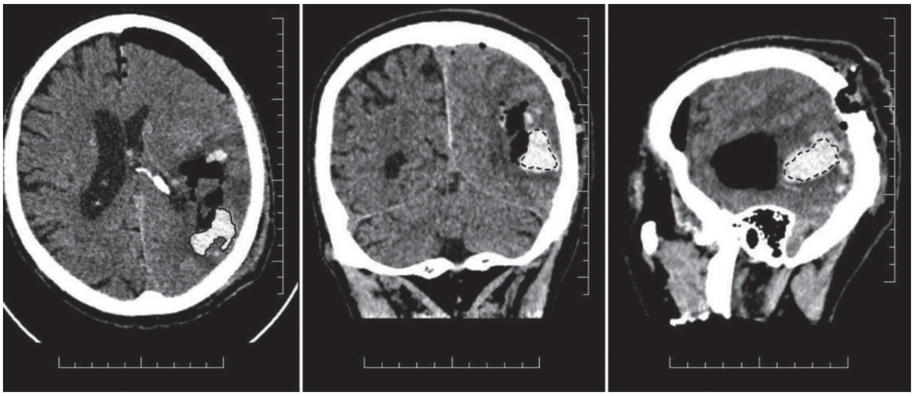

Fig. 3. Post operative endoscopic assisted evacuation CT scans with residual volume calculation – 7ml.

Until 2020, all intracerebral hematomas were indicated for surgical treatment. The patients underwent decompressive craniectomy/craniotomy, either with or without the use of a microscope. Since 2020, endoscopically assisted procedures have also been performed, with 25 out of the total 91 procedures being operated using an endoscopic approach. All patients underwent standard neurological and clinical examinations and routine laboratory tests upon admission. The diagnosis of supratentorial intracerebral bleeding was based on computed tomography (CT) findings. The two groups evaluated and compared basic parameters, clinical status on admission, imaging results, management, and outcome measures.

Using endoscopic techniques for evacuating intracerebral hematomas has become a standard procedure over the past decade, and some facilities have gradually replaced microscopic techniques. The fundamental equipment includes a cranial endoscope with 0 or 30-degree optics with rinsing and suction, and depending on the hematoma's location, neuronavigation capabilities are employed. In indicated cases and after stabilizing the patient's condition, the procedure is performed under general or local anesthesia, in most cases, without using the Mayfield three-pin head fixation. We determine the entry point and plan the corridor for accessing the hematoma using electromagnetic neuronavigation after patient registration. In cases of lobular/subcortical hematomas, the most suitable entry point is the location closest to the skull surface to access the hematoma, with the entry corridor parallel to the long axis of the hematoma. If the cortical area lies between the hematoma and the entry point, the approach to the hematoma is planned away from eloquent cortical regions (e.g., motor, speech, and visual). We recommend a parafascicular approach for putaminal hematomas, which is also used in endoscopy of the lateral ventricles and the third ventricle. In this case, the entry point is the frontal Kocher's point or its modification according to the hematoma's shape. The procedure starts with a 2cm linear skin incision, skull drilling, coagulation, and dural incision into X. We used a transparent sheath to reach the hematoma boundary, allowing regular monitoring of the surgical corridor in case of iatrogenic bleeding. After entering the hematoma, aspiration occurs using suction, with the endoscope enabling precise hematoma aspiration and preventing damage to peri-hemorrhagic structures. An advantage of the endoscope is the localization of the damaged vessel, which causes bleeding and coagulation. The hematoma evacuation and postoperative cavity inspection are performed within the cavity, which is either spontaneously filled with air after aspiration or utilizes a fluid environment. The outcomes of both procedures are comparable. Endoscopic hematoma evacuation aims to leave as little residue as possible, ideally up to 5ml (gross total resection). A satisfactory resection, not requiring revision or conversion of the surgical approach, involves leaving a residue between 5 and 20 ml. Postoperatively, a follow-up brain CT is indicated 6-12 hours after the procedure or in case of worsening neurological status.

Results

The average hematoma volume was 52.1ml with the endoscopic technique (n=25) and 56.3ml with the craniectomy procedure (n=66) (Tab. 1). The average intervention time from the onset of clinical symptoms was 10.5 hours, with ultra-rapid evacuation within 6 hours performed on 10 patients (13%), rapid evacuation within 24 hours on 68 patients (73%), and delayed evacuation, after more than 24 hours, on 13 patients (14%).

The most crucial parameter for evaluating treatment results and comparing them was the residual hematoma volume in milliliters and the calculation of hematoma evacuation efficacy in % (HEE) (Fig. 3). For conventional craniectomy procedures, the average residual volume was 10.1 ml, representing an average 82.1% hematoma evacuation efficacy. After endoscopically assisted procedures, the average residual volume was 3.9 ml, achieving a 92.5% hematoma evacuation efficacy. In both groups, the average evacuation efficacy was satisfactory, meaning minimal residuals above 75% or less than 20ml residual volume in most cases. Both

Tab. 1. A comparative analysis of efficacy and outcome.

Variable	Endoscopicaly assisted	Craniotomy/-ectomy	p
Patients	n=25	n=66	
Volume in ml	52.1 (37.120) [1]	56.3 (28.138) [1]	
Residual hematoma volume in ml	3.9 (25.<1) [1]	10.1 (46.<1) [1]	0.2180
Hematoma evacuation efficiency in %	92.5 (76.99) [1]	82.1 (55.99) [1]	0.2180
Length of surgery (min)	63 (44.89) [1]	127 (75.155) [1]	0.0001
Complications	2	32	0.0005
Length of ICU hospitalisation in hours	49	204	0.0468

[1] Range (minimum. Maximum)

these surgical procedures are suitable and nearly equivalent for evacuating spontaneous or traumatic supratentorial hematomas. A total of 17 neurosurgeons performed the conventional operation (decompressive craniectomy/craniotomy with or without the use of a microscope), while only 5 neurosurgeons, who had the most experience with neuroendoscopy and neuronavigation, performed the endoscopically assisted evacuation. It is presumed that the postoperative outcome favoring endoscopically assisted evacuation is influenced by the neurosurgeons' proficiency in endoscopy and the utilization of neuronavigation (Fig. 3).

Another parameter studied was the duration of the surgical procedure, measured according to the skin-to-skin standard, from skin incision to skin suture. Even considering the size and area of the surgical approach, the endoscopic procedure took 64 minutes less (63 minutes in the endoscopically assisted group versus 127 minutes in the conventional approach).

During the postoperative period, a follow-up brain CT was performed between 6 and 12 hours after the procedure. Overall, 12 patients underwent revision due to early postoperative complications (re-bleeding, subdural or epidural hematoma, obstructive hydrocephalus). For late postoperative complications (hypo resorptive hydrocephalus, postoperative cerebrospinal fluid leakage, surgical wound infection), 28 patients underwent revision or received temporary/permanent drainage of the ventricular system. After the endoscopically assisted procedure, fewer than 10% of patients were revised for early or late complications (2 out of 25 patients), whereas after the craniectomy procedure, revision or the introduction of lumbar drainage for cerebrospinal fluid leakage was performed on 35% of patients (32 patients).

In favor of endoscopically assisted hematoma evacuation was the length of hospitalization at the ICU. The average hospitalization was 49 hours post-procedure for the endoscopically evacuated hematoma group and, due to associated complications requiring observation or intervention, 8.5 days for the craniotomy group (Tab. 1).

Discussion

Intracerebral hematomas are characterized by direct damage to brain tissue and, depending on their location, can result in permanent neurological deficits. The goal of both conservative and surgical treatment is to prevent worsening neurological deficits caused by neurotoxicity, as well as to prevent re-bleeding and hematoma enlargement. The criteria for neurosurgical treatment aren't definitive, typically considering factors such as hematoma location and size, the patient's clinical condition, comorbidities, and age. Despite some generally recognized prognostic factors, individual institution experience and surgeon judgment prevail. A summary from the USA by Andaluz et al. noted that the last decade didn't bring improvement in ICH treatment outcomes – within 30 days, 35–52% of patients die, and only 20% remain self-sufficient after six months (10).

Indications for surgical treatment vary based on the hematoma's location (lobular, putaminal, thalamic), initial level of consciousness, and disease dynamics. Roughly speaking, a hematoma larger than 60ml with an initial GCS<8 has a 90% mortality rate with conservative treatment, while a hematoma <30ml has a 20% mortality rate with a GCS score >8 (11) (Waga et al, 1986). Thalamic hemorrhages are not usually indicated for surgical treatment, but studies on the benefits of minimally invasive procedures are ongoing. Surgical outcomes statistically fare better with putaminal and lobular hematomas, indicated for hematomas larger than 30ml and, according to individual study results, <80–100ml, with a GCS 8–13 and worsening of the initially favorable condition. Individual approaches are recommended for hematomas in eloquent areas and with GCS 5–7, 14–15 (12). The formed hematoma not only causes mass effect (pressure on nervous tissue) but is significantly neurotoxic for the CNS. Direct neurotoxins include thrombin, hemoglobin, and free iron (13).

Currently, research primarily focuses on the size of the residual hematoma, which has a direct impact on the patient's resulting clinical condition. From the MISTIE III study, it was found that patients with a residual volume <5ml achieved a good clinical outcome (73.3% mRS 0–3), while only 28.1% of patients reached a satisfactory clinical state with a residual volume >20ml (14). Studies with smaller patient populations demonstrate a significant influence of residual hematoma on the patient's final condition. Gross total evacuation of hematomas (residue <5ml) significantly impacts neurological improvement and markedly reduces hematoma neurotoxicity through secondary effects.

Most research on surgical treatment is based on the results of hematoma evacuation within a time window of 24–72 hours after the onset of bleeding (15). However, due to the neurotoxicity of the hematoma, the outcomes of these studies are insufficient. Secondary brain tissue damage occurring hours to days after bleeding includes peri-hemorrhagic inflammation, neurotoxicity from blood degradation products, and perifocal edema. The inflammatory process around the hemorrhage develops most prominently within the first two days and can progress for more than two weeks. Therefore, early ICH evacuation minimizes the cascade of secondary damage. This concept is supported by animal research, where the best benefit with minimal neurological damage was achieved by evacuating the hematoma within 6 hours, with similar results seen when evacuated within 12 hours of bleeding. Early hematoma evacuation reduces brain swelling, damage to the blood-brain barrier, glutamate accumulation around the hematoma, and decreases the risk of secondary brain damage (16).

Analysis from MISTIE III demonstrated a reduction in peri-hemorrhagic inflammation with hematoma volume reduction (14). The failure of large, randomized studies like STICH I and II is attributed to delayed bleeding evacuation, typically 30 and 26.7 hours from the onset of clinical symptoms. (15). Although the pathophysiology of ischemic and hemorrhagic strokes differs, the time factor remains crucial in both cases: "time is brain" applies to both. Compared to conservative therapy, the benefits of surgical treatment are achievable when the procedure is performed within 6 hours (ultra-early) or 24 hours (early) from the onset of bleeding symptoms. There's a higher risk of re-bleeding following ultra-rapid hematoma evacuation (within 6 hours). To date, there hasn't been a published randomized study evaluating the outcomes of ultra-rapid evacuation of intracerebral hematomas (3–6 hours).

Conclusion

After a skeptical period regarding surgical evacuation of intracerebral hematomas, the current trend involves a reassessment of the indications for neurosurgical intervention. Endoscopic evacuation offers a significant shift in treatment, now becoming a routine procedure that already achieves comparable or, in some cases, better outcomes than the microscopic technique. The benefit includes shorter operating times, with postoperative residue like the microscopic approach while achieving the desired gross total evacuation. Another advantage is the reduced iatrogenic damage to brain tissue and a decrease in postoperative complications. The limitation remains the operator's limited control over intraoperative bleeding and the surgeon's experience with endoscopic techniques.

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