Am J Transl Res 2025;17(4):3162-3170
www.ajtr.org /ISSN:1943-8141/AJTR0163334

Original Article

Effect of early stepwise controlled decompression combined with mild hypothermia therapy on efficacy, cerebral edema volume, and serum biochemical indices in patients with severe hypertensive intracerebral hemorrhage

Dawei Liu[1], Binbin Fu[2], Yixin Wang[1]

[1]Department of Cardiology, Tangshan Gongren Hospital, Tangshan 063000, Hebei, China; [2]Department of Cardiology, The People's Hospital of Qianxi County, Qianxi 064300, Hebei, China

Received January 15, 2025; Accepted March 11, 2025; Epub April 15, 2025; Published April 30, 2025

Abstract: Objective: To investigate the effects of early stepwise controlled decompression combined with mild hypothermia therapy (MHT) on clinical outcomes in patients with severe hypertensive intracerebral hemorrhage (sHICH). Methods: A total of 80 sHICH patients were included, with 32 cases in the control group receiving conventional craniotomy for hematoma evacuation and MHT, and 48 cases in the observation group undergoing early stepwise controlled decompression plus MHT. The two groups were compared in terms of treatment efficacy, hematoma clearance rate, prognosis, National Institutes of Health Stroke Scale (NIHSS) and Functional Ambulation Category (FAC) scores, complications, cerebral edema volume, and serum biochemical indices. Results: The observation group demonstrated significantly better efficacy, higher hematoma clearance rates, and improved clinical outcomes compared to the control group (all $P<0.05$). Postoperatively, the observation group had significantly lower NIHSS scores, reduced levels of neuron-specific enolase, central nervous system-specific protein, and malondialdehyde, as well as lower incidences of encephalocele, cerebral infarction, and delayed intracranial hematoma (all $P<0.05$). Additionally, cerebral edema volume was significantly reduced, while FAC scores were notably higher in the observation group (both $P<0.05$). Conclusion: Early stepwise controlled decompression combined with MHT is highly effective in the treatment of sHICH, leading to better neurological recovery, reduced cerebral edema, and improved biochemical profiles.

Keywords: Early stepwise controlled decompression, mild hypothermia therapy, severe hypertensive intracerebral hemorrhage, efficacy

Introduction

Intracerebral hemorrhage (ICH) is a severe neurological condition primarily caused by the non-traumatic rupture of blood vessels within the brain parenchyma, posing a significant threat to human health [1]. Hypertension is a major risk factor, contributing to cerebral microvascular hyaline degeneration, fibrosis, and abnormal hemodynamic changes, which can lead to decreased vascular elasticity and the development of hypertensive intracerebral hemorrhage (HICH) [2-4]. Patients with HICH often present with headache, agitation, lethargy, coma, nausea, and vomiting. The resulting increase in intracranial pressure can exacerbate cerebral edema, cause neurological impairment, and, in severe cases, lead to disability or death [5, 6].

Severe HICH (sHICH) is typically defined by a Glasgow Coma Scale score of <8, a hematoma volume >50 mL, or a midline shift >1 cm, indicating a more critical condition [7]. Given the current lack of an optimal treatment strategy for sHICH [8], further refinement and optimization of therapeutic approaches are urgently needed.

Surgical intervention remains the primary treatment for sHICH, aiming to rapidly evacuate the

https://doi.org/10.62347/TRKY5505

hematoma and restore neurological function [8]. Conventional craniotomy effectively removes the hematoma, alleviates mass effect, and achieves hemostasis [9]. However, this approach has notable limitations, including prolonged operative time, significant surgical trauma, high costs, and an elevated risk of postoperative complications [10, 11]. In contrast, early stepwise controlled decompression is an optimized surgical technique that considers intracranial pressure dynamics and performs gradual decompression, thereby offering enhanced vascular protection [12]. This method not only reduces cerebral perfusion but also rapidly lowers intracranial pressure to facilitate hematoma evacuation [13].

Additionally, mild hypothermia therapy (MHT) is a minimally invasive neuroprotective strategy commonly employed in the management of acute brain injury-associated cerebral edema [14, 15]. Studies suggest that the neuroprotective effects of MHT are mediated through its regulation of cerebral metabolism, promotion of lactate clearance, reduction of cerebral oxygen consumption, and maintenance of blood-brain barrier integrity [16].

This study aims to compare the clinical outcomes of early stepwise controlled decompression plus MHT versus conventional craniotomy plus MHT in patients with sHICH.

Materials and methods

Patient information

This retrospective study included 80 sHICH patients admitted to Tangshan Gongren Hospital between July 2020 and January 2024. Among them, 32 patients (control group) underwent conventional craniotomy for hematoma evacuation plus MHT, while 48 patients (observation group) received early stepwise controlled decompression plus MHT. The study protocol was approved by the Ethics Committee of Tangshan Gongren Hospital.

Inclusion and exclusion criteria

Inclusion criteria: Patients meeting the diagnostic criteria for HICH were included if they had supratentorial hemorrhage confirmed by cranial computed tomography (CT), a Glasgow Coma Scale score of ≤8, a history of hypertension, a hematoma volume of 30-50 mL, and complete clinical data.

Exclusion criteria: Patients were excluded if they had unstable vital signs, suspected cerebral aneurysm rupture or vascular malformations, traumatic hematoma, brainstem or cerebellar hemorrhage, coagulation disorders, significant dysfunction of the heart, lungs, or kidneys, or were on long-term anticoagulant therapy.

Methods

The control group underwent conventional craniotomy for hematoma evacuation combined with MHT. Preoperatively, the scalp was marked, and a skin-muscle flap was dissected layer by layer. Following skull drilling, the dura mater was suspended and incised, allowing access to the hematoma cavity by gently separating brain tissue along the lateral fissure. A conventional microscopic cortical fistulation was performed for hematoma evacuation, and meticulous hemostasis was ensured. The decision to retain or remove the bone flap depended on intracranial pressure status.

In the observation group, early stepwise controlled decompression combined with MHT was performed. The hematoma location and extent were determined via cranial CT, and the lateral fissure projection and hematoma center were marked. A frontotemporal bone flap craniotomy was performed, followed by rapid drilling. The dura mater was incised in a cross-like manner while avoiding superficial cerebral vessels. A drainage tube or puncture needle was inserted into the hematoma center to aspirate 5-10 mL of blood for controlled decompression, with adjustments made as necessary. After partial decompression, the bone window was enlarged, and the bone flap was milled. If intracranial pressure remained high, stepwise hematoma evacuation continued until decompression was adequate and encephalocele was absent. If intracranial pressure persisted at a high level after hematoma evacuation, a tension-reducing suture of the dura mater was performed, and the bone flap was removed for decompression. In cases with stable intracranial pressure, the dura mater was routinely sutured, and the bone flap was preserved.

Postoperatively, both groups received standard care, including infection control, dehydration therapy, acid-base balance maintenance, and correction of water-electrolyte disturbances. MHT was initiated using a water-circulating cooling blanket, reducing rectal temperature to 33-35°C within 4-6 hours. Patients were administered a continuous infusion of a muscle-lytic cocktail (200 mg of Tracrium [Shanghai Hengrui Pharmaceutical Co., Ltd., H20061298], 100 mg of Chlorpromazine [Shanxi Fenhe Pharmaceutical Co., Ltd., H14021851], and 20 mL of normal saline [Huaren Pharmaceutical Co., Ltd., H20034093]) at 2-4 mL/h for three days. Controlled rewarming followed, with the temperature gradually increased by 1°C every 4-6 hours over 12-24 hours until stabilizing at 36.5-37°C.

Outcome measures

Efficacy [17]. Treatment efficacy was evaluated based on the following criteria: Marked effectiveness: Significant alleviation of all clinical symptoms with >90% reduction in neurological deficits; Effectiveness: Improvement in clinical symptoms with a 75-90% reduction in neurological deficits; Ineffectiveness: Failure to meet the above criteria. The total effective rate was calculated as the sum of the marked effectiveness and effectiveness rates.

Hematoma clearance rate [18]. Hematoma clearance was assessed via CT reexamination two days postoperatively. The clearance rate/% = volume of cleared hematoma/volume of preoperative hematoma × 100%.

Prognosis [19]. Patients were followed up for six months postoperatively, and outcomes were assessed using the Glasgow Outcome Scale, which ranges from grade 1 to 5: Grade 1: Death; Grade 2: Vegetative state; Grade 3: Severe disability (conscious but dependent on daily care); Grade 4: Mild disability (capable of independent living and work with certain limitations); Grade 5: Good recovery (minor deficits but able to live and work normally).

Neurological function [20]. The National Institutes of Health Stroke Scale (NIHSS) was used to assess neurological function, with scores ranging from 0 to 42. Higher scores indicate greater neurological impairment.

Lower limb mobility [21]. The Functional Ambulation Category (FAC) was employed to evaluate lower limb function, where higher scores indicate better mobility.

Complication incidence [22]. The occurrence of adverse events, including encephalocele, cerebral infarction, delayed intracranial hematoma, arrhythmia, and chills, was recorded for both groups, and the incidence rate was calculated.

Cerebral edema volume [23]. Cranial CT scans were performed on postoperative days 7 and 14. The cerebral edema area in each layer was measured using an image analysis system, and the total cerebral edema volume was calculated by multiplying the edema area by the slice thickness.

Serum biochemical indices [24]. Fasting venous blood samples (5 mL) were collected from each patient before surgery and on postoperative day 7. After centrifugation, serum levels of neuron-specific enolase (NSE), central nervous system-specific protein S100-β, and malondialdehyde (MDA) were quantified using enzyme-linked immunosorbent assay.

Statistical analysis

Continuous variables were expressed as mean ± standard error of the mean (Mean ± SEM). Between-group comparisons were conducted using independent sample t-tests, while within-group comparisons utilized paired t-tests. Categorical data were reported as percentages and analyzed using the χ^2 test. All statistical analyses were performed using SPSS 20.0, with a significance level of $P<0.05$.

The sample size was determined using a validated sample-size calculation formula, accounting for a 10% dropout rate. The calculation indicated that a minimum of 37 participants per group (total n = 74) was required for adequate statistical power. The sample size included in this study fully met this requirement. The sample-size calculation formula is as follows:

$$n = \frac{2 \times (Z_{\alpha/2} + Z_{\beta})^2 \times [p(1 - p)]}{\Delta^2}$$

Table 1. Comparison of demographic characteristics

Indicators	Control group (n = 32)	Observation group (n = 48)	χ^2/t	P
Gender			0.034	0.854
Male	18 (56.25)	28 (58.33)		
Female	14 (43.75)	20 (41.67)		
Age (years)	60.06±9.33	57.56±9.87	1.134	0.260
Body mass index (kg/cm^2)	23.31±5.28	22.75±5.46	0.455	0.650
Duration of time from onset (h)	13.00±3.75	12.73±4.26	0.291	0.772
Bleeding type			0.543	0.762
Basal ganglia hemorrhage	14 (43.75)	25 (52.08)		
Frontal lobe hemorrhage	12 (37.50)	15 (31.25)		
Temporal lobe hemorrhage	6 (18.75)	8 (16.67)		

Table 2. Comparison of efficacy

Indicators	Control group (n = 32)	Observation group (n = 48)	χ^2	P
Marked effectiveness	9 (28.13)	18 (37.50)		
Effectiveness	14 (43.75)	28 (58.33)		
Ineffectiveness	9 (28.13)	2 (4.17)		
Total effectiveness	23 (71.88)	46 (95.83)	9.293	0.002

Table 3. Comparison of hematoma clearance rates

Indicators	Control group (n = 32)	Observation group (n = 48)	χ^2	P
90-100%	11 (34.38)	34 (70.83)	10.370	0.001
80-89%	13 (40.63)	11 (22.92)	2.867	0.090
<80%	8 (25.00)	3 (6.25)	5.692	0.017

Table 4. Comparison of prognosis

Indicators	Control group (n = 32)	Observation group (n = 48)	χ^2	P
Grade 1	5 (15.63)	1 (2.08)	5.075	0.024
Grade 2	6 (18.75)	2 (4.17)	4.537	0.033
Grade 3	14 (43.75)	6 (12.50)	10.000	0.002
Grade 4	6 (18.75)	29 (60.42)	13.545	<0.001
Grade 5	1 (3.13)	10 (20.83)	5.077	0.024

Results

Comparison of demographic characteristics

The comparison of demographic characteristics between the two groups showed no significant differences in sex, age, body mass index (BMI), time from onset to treatment or bleeding type (all P>0.05), indicating clinical comparability (**Table 1**).

Comparison of efficacy

The total effective rate in the observation group was 95.83%, significantly higher than the 71.88% observed in the control group (P<0.05) (**Table 2**).

Comparison of hematoma clearance rates

Hematoma clearance rates were significantly better in the observation group (P< 0.05). A higher number of patients achieved a clearance rate of 90-100% compared to the control group (P<0.05). In contrast, significantly fewer patients in the observation group had a clearance rate below 80% compared to the control group (P<0.05) (**Table 3**).

Comparison of prognosis

Based on the Glasgow Outcome Scale at the six-month follow-up, the observation group had significantly fewer patients with a GOS range of 1-3 and more patients with a range of 4-5 than the control group (P<0.05) (**Table 4**).

Comparison of NIHSS and FAC scores

Preoperative NIHSS and FAC scores were comparable between the two groups (both P>0.05). Postoperatively, both groups exhibited decreased NIHSS scores and increased FAC scores, with the observation group showing a significantly lower NIHSS score and a higher FAC score than the control group (both P<0.05) (**Figure 1**).

Comparison of complication incidence

The incidence of complications, including encephalocele, cerebral infarction, delayed intracranial hematoma, arrhythmia, and chills, was

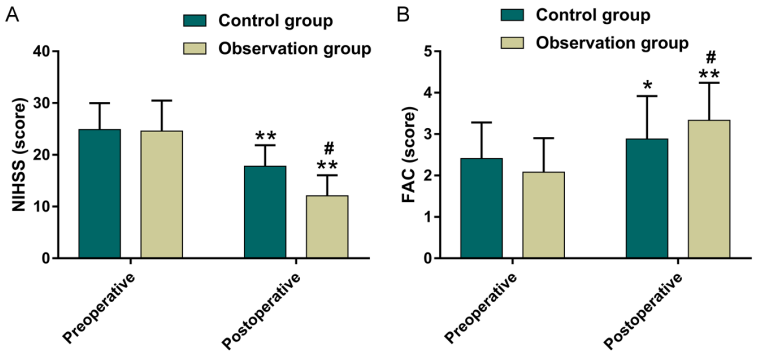

Figure 1. Comparison of NIHSS and FAC scores. A. Comparison of NIHSS before and after surgery. B. Comparison of FAC before and after surgery. Note: *P<0.05, **P<0.01 vs. preoperative levels; #P<0.05 vs. control group. NIHSS, the National Institutes of Health Stroke Scale; FAC, Functional Ambulation Category.

Table 5. Comparison of complications

Indicators	Control group (n = 32)	Observation group (n = 48)	χ^2	P
Encephalocele	5 (15.63)	1 (2.08)	5.075	0.024
Cerebral infarction	5 (15.63)	1 (2.08)	5.075	0.024
Delayed intracranial hematoma	4 (12.50)	0 (0.00)	6.316	0.012
Arrhythmia	1 (3.13)	0 (0.00)	1.519	0.218
Chills	1 (3.13)	1 (2.08)	0.085	0.770

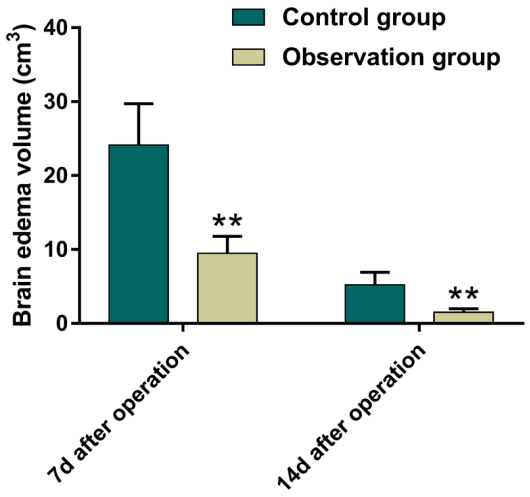

Figure 2. Comparison of cerebral edema volume. Inter-group comparison of brain edema volume 7 days after surgery. Inter-group comparison of cerebral edema volume 14 days after surgery. Note: **P<0.01 vs. control group.

analyzed. The observation group had significantly lower incidence rates of encephalocele, cerebral infarction, and delayed intracranial hematoma compared to the control group (all P<0.05). However, the incidence rates of arrhythmia and chills were similar between the two groups (both P>0.05) (**Table 5**).

Comparison of cerebral edema volume

Cerebral edema volume was measured on postoperative days 7 and 14. The observation group had significantly lower cerebral edema volumes at both time points compared to the control group (both P<0.05) (**Figure 2**).

Comparison of serum biochemical indices

Preoperative levels of NSE, S100-β, and MDA were similar between the two groups (all P>0.05). Postoperatively, all three indices showed a greater reduction in the observation group compared to the control group (all P<0.05) (**Figure 3**).

Discussion

HICH is a common type of intracerebral hemorrhage associated with emotional agitation, excessive physical exertion, and cognitive overload [25]. Its occurrence leads to brain neuronal damage, while the hematoma compresses surrounding brain tissue, increasing the risk of secondary cerebral edema, elevated intracranial pressure, and potentially life-threatening brain herniation [26]. Consequently, HICH patients face a high risk of early mortality and, if they survive, may experience long-term sequelae such as dysphagia, cognitive impairment, and motor dysfunction [27]. Reducing postoperative complications, improving treatment efficacy, minimizing cerebral edema, and optimizing serum biochemical indices are key challenges in the management of sHICH.

Early stepwise controlled decompression enables the gradual regulation of intracranial

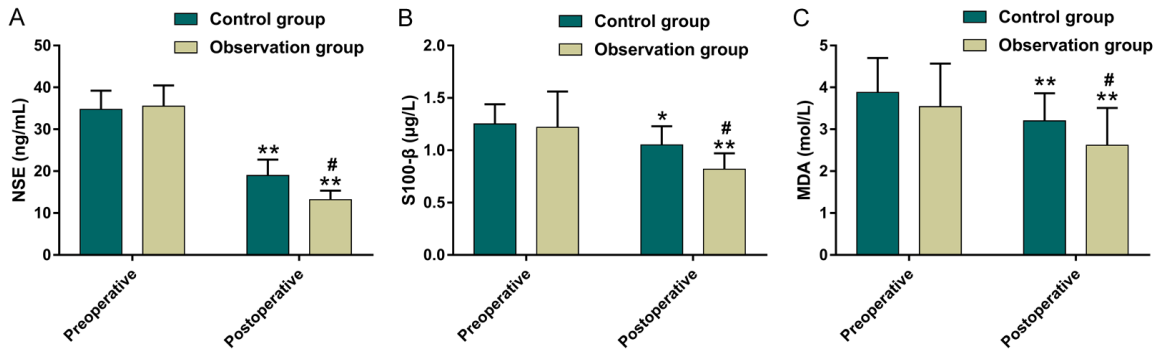

Figure 3. Comparison of serum biochemical indices. A. Comparison of NSE levels before and after surgery. B. Comparison of S100-β levels before and after surgery. C. Comparison of MDA levels before and after surgery. Note: *P<0.05, **P<0.01 vs. preoperative levels; #P<0.05 vs. control group. NSE, neuron-specific enolase; S100-β, central nervous system-specific protein; MDA, malondialdehyde.

pressure during surgery. When applied to sHICH patients, it allows for early rapid drilling, partial hematoma drainage, and progressive intracranial pressure control, thereby enhancing therapeutic outcomes while ensuring procedural safety [28]. In this study, the total effective rate in the observation group (95.83%) was significantly higher than that in the control group (71.88%), indicating that early stepwise controlled decompression combined with MHT offers greater clinical benefits compared to conventional craniotomy plus MHT.

Moreover, patients in the observation group exhibited higher hematoma clearance rates and better overall prognoses. Postoperatively, they demonstrated significantly lower NIHSS scores and higher FAC scores compared to the control group, suggesting superior neurological recovery and functional mobility. The improved outcomes may be attributed to the procedural strategy of early stepwise controlled decompression, which removes only 5-10 mL of hematoma per step. This approach not only minimizes disruption to normal brain tissue but also reduces the risk of brain tissue displacement caused by reperfusion injury [29]. Ling et al. [30] reported that early stepwise controlled decompression significantly improved neurological recovery and daily living abilities in patients with cerebral hemorrhage, aligning with our findings. In this study, complication analysis revealed that the observation group had a lower incidence of encephalocele, cerebral infarction, and delayed intracranial hematoma compared to the control group, suggesting that early stepwise controlled decompression combined with MHT can effectively reduce postoperative complications in patients with sHICH.

The analysis of cerebral edema showed that the observation group had significantly lower cerebral edema volumes on postoperative days 7 and 14 compared to the control group, indicating the efficacy of early stepwise controlled decompression plus MHT in reducing postoperative brain edema. This may be attributed to the gradual repositioning of brain tissue toward its original anatomical location during stepwise decompression, which helps prevent acute cerebral vascular congestion, perfusion pressure breakthrough, and mechanical injury. These mechanisms contribute to the protection of the blood-brain barrier, a reduction in acute brain swelling, and the mitigation of secondary neurological damage [31, 32].

Furthermore, serum biochemical analysis demonstrated a significant postoperative reduction in NSE, S100-β, and MDA levels in the observation group, which were lower than those in the control group. This suggests that early stepwise controlled decompression plus MHT effectively reduces abnormally elevated levels of NSE, S100-β, and MDA - key biomarkers of brain injury. The downregulation of these markers reflects improved hematoma clearance, reduced intracranial pressure, decreased brain tissue damage, and alleviation of oxidative stress [33-35]. The neuroprotective effects observed in this study may be attributed to the controlled and orderly removal of hematoma at the lesion site, which optimizes intracranial

pressure regulation and minimizes oxidative stress-induced injury [36, 37].

This study highlights the comprehensive clinical benefits of early stepwise controlled decompression plus MHT in sHICH patients by evaluating multiple aspects, including efficacy, hematoma clearance, prognosis, neurological function, lower limb mobility, complication rates, cerebral edema volume, and serum biochemical indices. The findings confirm the superiority of this approach over conventional craniotomy plus MHT, providing valuable insights into optimal treatment strategies in sHICH patients. Implementing this approach in clinical practice may contribute to improved patient outcomes and a higher quality of care.

In conclusion, early stepwise controlled decompression combined with MHT significantly enhances treatment efficacy, improves functional mobility and prognosis, increases hematoma clearance, and alleviates neurological deficits. Additionally, it reduces brain edema volume and downregulates NSE, S100-β, and MDA levels, while also lowering the incidence of complications such as encephalocele, cerebral infarction, and delayed intracranial hematoma. These findings underscore its potential as an effective and safe therapeutic strategy for sHICH management.

This study also has several limitations, including its single-center, retrospective design, which may limit generalizability, and a relatively small sample size that could impact statistical robustness. The follow-up period was short, assessing prognosis only up to six months, and lacked advanced neuroimaging assessments such as MRI, which could provide deeper insights into brain recovery. Additionally, potential selection bias, limited biochemical markers, and variability in surgical execution may have influenced outcomes. Future research should focus on multi-center randomized controlled trials, incorporating long-term follow-up, advanced neuroimaging, and additional neuroprotective strategies. Comparative studies with other minimally invasive techniques, predictive modeling using AI, and biomarker discovery could further refine treatment approaches.

Acknowledgements

This study was supported by the Medical Science Research Project of Hebei, Fund No. 20190168.

Disclosure of conflict of interest

None.

Address correspondence to: Dawei Liu, Department of Cardiology, Tangshan Gongren Hospital, Tangshan 063000, Hebei, China. Tel: +86-0315-5723168; E-mail: liudawei199@hotmail.com

References

[1] Chen Zhou ZH, Salvador Alvarez E, Hilario Barrio A, Cardenas Del Carre AM, Romero Coronado J and Ramos Gonzalez A. Primary and secondary non-traumatic intra-cerebral haemorrhage: MRI findings. Radiologia (Engl Ed) 2023; 65: 149-164.

[2] Puy L, Parry-Jones AR, Sandset EC, Dowlatshahi D, Ziai W and Cordonnier C. Intracerebral haemorrhage. Nat Rev Dis Primers 2023; 9: 14.

[3] Fang C, Magaki SD, Kim RC, Kalaria RN, Vinters HV and Fisher M. Arteriolar neuropathology in cerebral microvascular disease. Neuropathol Appl Neurobiol 2023; 49: e12875.

[4] Wei W, Ma D, Li L and Zhang L. Cognitive impairment in cerebral small vessel disease induced by hypertension. Neural Regen Res 2024; 19: 1454-1462.

[5] Querfurth H, Marczak I, Rahimian N, Jijakli A and Green-LaRoche D. Spontaneous intracranial hemorrhages in a community of Asian Americans: case series and literature review. Cureus 2024; 16: e52128.

[6] Wan Y, Holste KG, Hua Y, Keep RF and Xi G. Brain edema formation and therapy after intracerebral hemorrhage. Neurobiol Dis 2023; 176: 105948.

[7] Qiu W, Liu C, Ye J, Wang G, Yang F, Pan Z, Hu W and Gao H. Age-to-Glasgow coma scale score ratio predicts gastrointestinal bleeding in patients with primary intracerebral hemorrhage. Front Neurol 2023; 14: 1034865.

[8] Li W, Li SG, Li L, Yang LJ, Li ZS, Li X, Ye AY, Xiong Y, Zhang Y and Xiong YY. Soyasaponin I alleviates hypertensive intracerebral hemorrhage by inhibiting the renin-angiotensin-aldosterone system. Clin Exp Hypertens 2023; 45: 2177667.

[9] Tang C, Zhang M and Li W. Meta-analysis of stereotactic hematoma removal and craniotomy hematoma removal in the treatment of hypertensive intracerebral hemorrhage in the elderly. Medicine (Baltimore) 2023; 102: e36533.

[10] Li Y, Yang S, Zhou X, Lai R and Tan D. A retrospective cohort study of neuroendoscopic surgery versus traditional craniotomy on surgical success rate, postoperative complications,

and prognosis in patients with acute intracerebral hemorrhage. Comput Intell Neurosci 2022; 2022: 2650795.
[11] Xu Z, Sun Z, Xu M, Zhao B, Wei J, Guo H, Li Y and Mao J. The effect of small bone window craniotomy removal on lactic acid and CRP in patients with hypertensive intracerebral hemorrhage in the basal Ganglia. Neurol India 2022; 70: 2047-2052.
[12] Zheng J, Zhang C, Wu Y, Zhang C, Che Y, Zhang W, Yang Y, Zhu J, Yang L and Wang Y. Controlled decompression alleviates motor dysfunction by regulating microglial polarization via the HIF-1alpha signaling pathway in intracranial hypertension. Mol Neurobiol 2023; 60: 5607-5623.
[13] Yang X. Effects of decompressive craniectomy combined with edaravone on postoperative neurological functions and hemodynamics of patients with severe traumatic brain injury. Neurosciences (Riyadh) 2024; 29: 25-31.
[14] Al-Nashash H and All AH. Neuroprotective role of hypothermia in acute spinal cord injury. Biomedicines 2022; 10: 104.
[15] Zhou C, Wang J, Shao G, Xia X, Wu L, Yu A and Yang Z. Mild hypothermia protects brain injury after intracerebral hemorrhage in mice Via enhancing the Nrdp1/MyD88 signaling pathway. Neurotox Res 2022; 40: 1664-1672.
[16] Yang G and Shao G. Clinical effect of minimally invasive intracranial hematoma in treating hypertensive cerebral hemorrhage. Pak J Med Sci 2016; 32: 677-681.
[17] Xie Q, Zhang X, Ding Y and Zhang L. Assessing the clinical efficacy of stereotactic puncture in combination with postoperative rehabilitation training for hypertensive cerebral hemorrhage. Altern Ther Health Med 2024; 30: 122-127.
[18] Gui C, Gao Y, Hu D and Yang X. Neuroendoscopic minimally invasive surgery and small bone window craniotomy hematoma clearance in the treatment of hypertensive cerebral hemorrhage. Pak J Med Sci 2019; 35: 377-382.
[19] Zhang J, Lu S, Wang S, Zhou N and Li G. Comparison and analysis of the efficacy and safety of minimally invasive surgery and craniotomy in the treatment of hypertensive intracerebral hemorrhage. Pak J Med Sci 2018; 34: 578-582.
[20] Hou Y, Sang Y, Ma M, Yang K, Yang F and Wei G. Relationship between changes in neurological deficit severity and adverse cardiac events in elderly patients with hypertensive intracerebral hemorrhage: a retrospective cohort study. Int J Neurosci 2024; 1-6.
[21] Ikeda N, Sakurai M, Yamada E, Gotoh S, Tanabe N, Hayashi Y and Matsushita I. Factors associated with independent ambulation at 3 months after putaminal hemorrhage: an observational study. J Phys Ther Sci 2024; 36: 167-174.
[22] Lv K, Wang Y, Chao H, Cao S and Cao W. Comparison of the efficacy of subosseous window neuro-endoscopy and minimally invasive craniotomy in the treatment of basal ganglia hypertensive intracerebral hemorrhage. J Craniofac Surg 2023; 34: e724-e728.
[23] Huang L, Wu Q, Ye F, Che W, Zhao X, Yang C, Ren S, Wu G and Wang L. Apolipoprotein E-epsilon4 allele is associated with perihematomal brain edema and poor outcomes in patients with intracerebral hemorrhage. Sci Rep 2025; 15: 5682.
[24] Chen C, Ma S, Liao L, Xiao Y and Dai H. Alda-1 alleviates brain injury after cardiopulmonary resuscitation by regulating acyl-CoA synthetase long-chain family member 4/glutathione peroxidase 4 pathway-mediated ferroptosis in swine. Zhonghua Wei Zhong Bing Ji Jiu Yi Xue 2023; 35: 376-380.
[25] Zhang Y, Song L and Zhao J. Role of scalp hypothermia in patients undergoing minimally invasive evacuation of hypertensive cerebral hemorrhage. Pak J Med Sci 2019; 35: 1451-1455.
[26] Xu S, Du B, Shan A, Shi F, Wang J and Xie M. The risk factors for the postoperative pulmonary infection in patients with hypertensive cerebral hemorrhage: a retrospective analysis. Medicine (Baltimore) 2020; 99: e23544.
[27] Qureshi AI, Foster LD, Lobanova I, Huang W and Suarez JI. Intensive blood pressure lowering in patients with moderate to severe grade acute cerebral hemorrhage: post Hoc analysis of antihypertensive treatment of acute cerebral hemorrhage (ATACH)-2 trial. Cerebrovasc Dis 2020; 49: 244-252.
[28] Lo YT, See AAQ and King NKK. Decompressive craniectomy in spontaneous intracerebral hemorrhage: a case-control study. World Neurosurg 2017; 103: 815-820, e812.
[29] Qureshi AI, Palesch YY, Martin R, Novitzke J, Cruz-Flores S, Ehtisham A, Ezzeddine MA, Goldstein JN, Hussein HM, Suri MF and Tariq N; Antihypertensive Treatment of Acute Cerebral Hemorrhage Study Investigators. Effect of systolic blood pressure reduction on hematoma expansion, perihematomal edema, and 3-month outcome among patients with intracerebral hemorrhage: results from the antihypertensive treatment of acute cerebral hemorrhage study. Arch Neurol 2010; 67: 570-576.
[30] Ling M, Zhang Q, Zang L, Li X and Liu Q. Decompressive craniectomy can improve the recovery of neurological function, daily living ability and life quality of patients with intracerebral hemorrhage after surgery. Am J Transl Res 2021; 13: 11364-11374.

[31] La Rosa G, Conti A, Cardali S, Cacciola F and Tomasello F. Does early decompression improve neurological outcome of spinal cord injured patients? Appraisal of the literature using a meta-analytical approach. Spinal Cord 2004; 42: 503-512.

[32] Vahedi K, Hofmeijer J, Juettler E, Vicaut E, George B, Algra A, Amelink GJ, Schmiedeck P, Schwab S, Rothwell PM, Bousser MG, van der Worp HB, Hacke W, Decimal D and investigators H. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. Lancet Neurol 2007; 6: 215-222.

[33] Liang L, Li X, Dong H, Gong X and Wang G. A comparative study on the efficacy of robot of stereotactic assistant and frame-assisted stereotactic drilling, drainage for intracerebral hematoma in patients with hypertensive intracerebral hemorrhage. Pak J Med Sci 2022; 38: 1796-1801.

[34] Gong W, Zhang S, Li X and Shi L. Dexmedetomidine is superior to midazolam for sedation and cerebral protection in postoperative hypertensive intracerebral hemorrhage patients: a retrospective study. J Int Med Res 2020; 48: 300060520957554.

[35] Xia L, Han Q, Ni XY, Chen B, Yang X, Chen Q, Cheng GL and Liu CF. Different techniques of minimally invasive craniopuncture for the treatment of hypertensive intracerebral hemorrhage. World Neurosurg 2019; 126: e888-e894.

[36] Yu SX, Zhang QS, Yin Y, Liu Z, Wu JM and Yang MX. Continuous monitoring of intracranial pressure for prediction of postoperative complications of hypertensive intracerebral hemorrhage. Eur Rev Med Pharmacol Sci 2016; 20: 4750-4755.

[37] Buschmann U, Yonekawa Y, Fortunati M, Cesnulis E and Keller E. Decompressive hemicraniectomy in patients with subarachnoid hemorrhage and intractable intracranial hypertension. Acta Neurochir (Wien) 2007; 149: 59-65.