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Study on the Correlation between Bone Window Design in Decompressive Craniectomy for Spontaneous Intracerebral Hemorrhage and the Formation of Postoperative Subdural Effusion

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Abstract: *Objective:* To study the correlation between bone window design in decompressive craniectomy for spontaneous intracerebral hemorrhage and the formation of postoperative subdural effusion. *Methods:* 36 patients with spontaneous intracerebral hemorrhage who underwent decompressive craniectomy in our hospital from September 2020 to September 2022 were selected as the research subjects. All patients were divided into a case group (with subdural effusion, $n = 16$) and a control group (without subdural effusion, $n = 20$) based on whether they developed subdural effusion within 2 weeks after surgery. The situation of hemorrhage breaking into the ventricles was compared between the two groups, and skull CT thin-layer scan was performed after decompressive craniectomy to reconstruct the skull in three dimensions. The distance from the edge of the decompression window to the midline, the height of the decompression window, the maximum anteroposterior diameter of the decompression window, and the maximum area of the decompression window were measured. *Results:* There were no significant differences in gender, age, hemorrhage volume, and hemorrhage location between the two groups ($P > 0.05$), indicating comparability. There were significant differences in the height of the decompression window, the maximum anteroposterior diameter of the decompression window, and the area of the decompression window between the case group and the control group ($P < 0.05$). There were no significant differences in whether the hemorrhage broke into the ventricles and the distance from the edge of the decompression window to the midline ($P > 0.05$). Patients with a decompression window height greater than 8 cm, an anteroposterior diameter greater than 10 cm, and an area greater than 50 cm^2 had a higher probability of developing postoperative subdural effusion ($P = 0.018, 0.0008, 0.013 < 0.05$). *Conclusion:* The height, maximum anteroposterior diameter, and area of the decompression window are important factors affecting the formation of postoperative subdural effusion after decompressive craniectomy. Optimizing the design of the decompression window and avoiding excessively large bone windows can help reduce the incidence of postoperative subdural effusion and improve patient prognosis.

Keywords: Spontaneous cerebral hemorrhage; Decompressive craniectomy; Bone window design; Subdural effusion

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1. Introduction

Spontaneous intracerebral hemorrhage refers to the spontaneous rupture of large and small arteries, veins, and capillaries in the adult brain caused by non-traumatic injuries, leading to parenchymal hemorrhage. It has a high mortality and disability rate, posing a threat to patients' lives [1]. Spontaneous intracerebral hemorrhage can be caused by hypertension, amyloid vasculopathy, arteriovenous malformations, aneurysms, and other diseases. In recent years, decompressive craniectomy has been widely used in this disease. Decompressive craniectomy can not only remove the hematoma, reduce intracranial pressure, relieve the compression on surrounding brain tissue, and lower intracranial pressure, but also interrupt the pathological process of cerebral hemorrhage, increase cerebral blood flow perfusion, protect brain tissue, and reduce patient mortality [2]. Currently, decompressive craniectomy often refers to the standard large trauma craniotomy commonly used in foreign clinical practice to design decompression windows for patients, which has achieved good clinical results [3].

Although decompressive craniectomy exhibits strong clinical advantages, it is prone to complications after surgery, among which subdural effusion is one of the common complications. If it is not detected and treated in a timely manner, it will affect the patient's prognosis and even endanger the patient's life [4]. There is evidence in the prognosis study of large bone flaps for traumatic brain injury that the size of the bone flap is significantly correlated with postoperative subdural effusion [5]. Therefore, this study explores the influencing factors of postoperative effusion through a correlation study between the bone window design of decompressive craniectomy and postoperative effusion, providing a theoretical basis for reducing the occurrence of postoperative subdural effusion.

2. Materials and methods

2.1. General information

Thirty-six patients with spontaneous intracerebral hemorrhage who underwent decompressive craniotomy in our hospital from September 2020 to September 2022 were selected as the study subjects. Inclusion criteria were: (1) patients with a clear diagnosis of spontaneous intracerebral hemorrhage and first-time onset; (2) patients who underwent decompressive craniotomy after admission.

Exclusion criteria were: (1) patients with previous craniocerebral surgery; (2) hemorrhage caused by vascular malformations, cerebral aneurysms, or coagulation disorders; (3) patients who died or gave up treatment within two weeks after surgery. This study has passed the hospital ethics review and obtained informed consent from patients and their families.

2.2. Methods

All patients were divided into a case group (occurred, $n = 16$) and a control group (did not occur, $n = 20$) based on whether subdural effusion occurred within 2 weeks after surgery. The general information of the two groups is shown in **Table 1**.

The relationship between the following observation indicators and the formation of subdural effusion was compared between the two groups:

(1) Hemorrhage characteristics (ventricular rupture);

(2) Bone window parameters: Skull CT thin-layer scans were performed on the two groups after decompressive craniotomy for three-dimensional reconstruction of the skull, and the distance from the edge of the decompression window to the midline, the height of the decompression window, and the maximum anteroposterior diameter of the decompression window were measured. The maximum area of the decompression window was calculated (calculation formula: $S = \pi ab$, where a is 1/2 of the maximum anteroposterior diameter and b is 1/2 of the height).

2.3. Statistical methods

The data obtained in this study were statistically analyzed using SPSS 21.0. Measurement data were tested using the *t*-test, and count data were tested using the chi-square test. When $P < 0.05$, the difference in data was considered statistically significant.

3. Results

3.1. Comparison of general information between the two groups

There were no significant differences in gender, age, hemorrhage volume, and hemorrhage location between the two groups ($P > 0.05$), indicating a certain degree of comparability. See **Table 1** for details.

Table 1. Comparison of general information between the two groups (*n*, mean ± SD)

Basic information		Case group ($n = 16$)	Control group ($n = 20$)	χ^2/t value	*P* value
Gender	Male	11	12	0.295	0.587
	Female	5	8		
Age (years)	21–30	1	2	0.669	0.955
	31–40	3	4		
	41–50	4	3		
	51–60	6	8		
	61–70	2	3		
Amount of bleeding (mL)	/	59.77 ± 6.24	62.19 ± 5.94	1.188	0.243
Location of bleeding	Basal ganglia	7	9	0.008	0.996
	Thalamus	5	6		
	Cerebral lobes	4	5		

3.2. Relationship between various observation indicators and the formation of subdural effusion

There were significant differences in data between the case group and the control group in terms of the height of the decompression window, the maximum anteroposterior diameter of the decompression window, and the area of the decompression window ($P < 0.05$). There was no significant difference in data between whether the hemorrhage broke into the ventricles and the distance from the edge of the decompression window to the midline ($P > 0.05$). See **Table 2**.

Table 2. Relationship between various observation indicators and the formation of subdural effusion in the two groups

Observation indicators		Case group (n = 16)	Control group (n = 20)	χ^2 value	P value
Bleeding into the ventricles	Yes	11	9	2.031	0.154
	No	5	11		
Distance from decompression window to midline (cm)	≤ 1	2	4	0.360	0.548
	> 1	14	16		
	≤ 2	1	5	0.250	0.134
	> 2	15	15		
	≤ 3	5	10	1.286	0.257
	> 3	11	10		
Height of decompression window (cm)	≤ 8	2	10	5.625	0.018
	> 8	14	10		
Maximum anteroposterior diameter of decompression window (cm)	≤ 10	2	11	6.959	0.008
	> 10	14	9		
Area of decompression window (cm^2)	≤ 60	3	12	6.223	0.013
	> 60	13	8		

3.3. Analysis of the proportion of decompression window height, maximum anteroposterior diameter of decompression window, and decompression window area in the formation of subdural effusion

Patients with a decompression window height greater than 8cm, an anteroposterior diameter of the decompression window greater than 10cm, and a decompression window area greater than 60cm² have a higher probability of developing postoperative subdural effusion ($P = 0.018, 0.0008, 0.013 < 0.05$). See **Table 3**.

Table 3. Proportion of decompression window height, maximum anteroposterior diameter of decompression window, and decompression window area in the formation of subdural effusion (n, %)

Observation indicators		Case group (n = 16)	Control group (n = 20)
Height of decompression window (cm)	≤ 8	2 (5.56%)	10 (27.78%)
	> 8	14 (38.89%)	10 (27.78%)
Maximum anteroposterior diameter of decompression window (cm)	≤ 10	2 (5.56%)	11 (30.51%)
	> 10	14 (38.89%)	9 (25.00%)
Area of decompression window (cm^2)	≤ 60	3 (8.33%)	12 (33.33%)
	> 60	13 (36.11%)	8 (22.22%)

4. Discussion

Spontaneous intracerebral hemorrhage occurs suddenly, rapidly forming a space-occupying lesion in a short period. This can lead to changes in cell morphology, separation or even rupture of nerve fibers, and forced interruption of synaptic connections. At the same time, blood vessels around the hematoma become deformed due to compression, ultimately leading to necrosis. The pressure continues to spread around, causing brain tissue displacement and squeezing crucial neurovascular structures. If the condition further deteriorates, it can trigger cerebral hernia [6]. Early on, conservative medical therapy was commonly used to treat spontaneous intracerebral hemorrhage [7]. However, this method proved to be less effective, with a relatively high mortality rate. With advances in medical technology, surgical craniotomy has been introduced, significantly reducing patient mortality. Therefore, clinicians have reached a consensus: the key to improving the success rate of rescue lies in surgically removing the hematoma as soon as possible after the onset of the disease, reducing intracranial pressure, and fully protecting damaged brain tissue [8]. The commonly used decompressive craniotomy has significant advantages. It can comprehensively and thoroughly remove the hematoma and precisely remove inactive brain tissue, effectively preventing increased intracranial pressure and achieving the goal of extracranial decompression [9]. Moreover, this surgery can completely stop bleeding, effectively reducing the risk of postoperative rebleeding [10]. However, it cannot be ignored that this surgical method also has certain drawbacks. During the operation, damage to intracranial blood vessels and tissues is unavoidable, and complications such as subdural effusion occur occasionally.

After eliminating the interference of basic data factors on the research results, this study conducted a comparative analysis of the morphological parameters of the decompression window and the formation of subdural effusion between 16 patients with subdural effusion and 20 patients without effusion. The results showed that the decompression window height (>8cm), maximum anteroposterior diameter (> 10 cm), and area (> 60 cm^2) in the case group were significantly higher than those in the control group ($P < 0.05$). This finding suggests that a larger decompression window may increase the risk of subdural effusion by changing the pressure gradient and cerebrospinal fluid dynamics within the cranial cavity. Possible mechanisms include: (1) Large bone windows cause excessive displacement of brain tissue, expanding the subdural space and providing room for effusion formation; (2) An excessively large decompression window may affect the natural reduction of brain tissue, leading to persistent gaps between the dura and the brain surface; (3) Large bone windows may interfere with the normal circulation of cerebrospinal fluid, promoting fluid accumulation [11].

This study also found that whether hemorrhage breaks into the ventricles and the distance from the edge of the decompression window to the midline are not significantly correlated with the formation of subdural effusion ($P > 0.05$). The reason for this result may be related to the small sample size of the study or limitations of measurement methods [12]. Additionally, although hemorrhage breaking into the ventricles may increase the risk of intracranial pressure and cerebrospinal fluid circulation disorders, its direct impact on subdural effusion may be masked by other factors.

Further analysis of the data showed that the incidence of subdural effusion was significantly higher in patients with a decompression window height greater than 8 cm, a maximum anteroposterior diameter greater than 10cm, and an area greater than 60 cm^2 compared to the control group ($P < 0.05$). This result is consistent with previous studies indicating that larger decompression windows may increase the risk of postoperative complications [13].

Based on the results of this study, the following surgical optimization suggestions are proposed:

(1) For patients with spontaneous intracerebral hemorrhage, it is recommended to control the height of the decompression window within 8cm, the anteroposterior diameter within 10 cm, and the area below 60 cm^2. This can not only meet the needs of decompression but also reduce the risk of effusion.

(2) Combine intraoperative ultrasound or intracranial pressure monitoring to dynamically evaluate brain tissue

herniation and avoid blindly expanding the bone window.

(3) For patients with larger bone windows, early (within 72 hours postoperatively) cranial CT review is recommended, and consider prophylactic use of elastic bandages for compression dressing.

5. Conclusion

In summary, the height, maximum anteroposterior diameter, and area of the decompression window are important factors that affect the formation of subdural effusion after decompressive craniectomy. Optimizing the design of the decompression window and avoiding excessively large bone windows may help reduce the incidence of postoperative subdural effusion and improve patient prognosis. However, this study still has many limitations. Firstly, the sample size of this study is small, which may have a certain impact on statistical power; secondly, postoperative management factors (such as positional intervention and use of dehydrating agents) that affect the formation of effusion were not included. Finally, long-term follow-up data to evaluate the dynamic evolution of effusion is lacking. Future studies can expand the sample size through multi-center cooperation, combine objective indicators such as intracranial pressure monitoring and cerebrospinal fluid dynamics testing, and further explore the pathophysiological mechanism of decompression window parameters and cerebrospinal fluid circulation disorders, providing more precise evidence for optimizing surgical plans.

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Disclosure statement

The author declares no conflict of interest.

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