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Clinical Article

Risk Factors Associated with Subdural Hygroma after Decompressive Craniectomy in Patients with Traumatic Brain Injury : A Comparative Study

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Objective : Subdural hygroma (SDG) is a complication occurring after head trauma that may occur secondary to decompressive craniectomy (DC). However, the mechanism underlying SDG formation is not fully understood. Also, the relationship between the operative technique of DC or the decompressive effect and the occurrence and pathophysiology of SDG has not been clarified. Purpose of this study was to investigate the risk factors of SDG after DC in our series.

Methods : From January 2004 to December 2008, DC was performed in 85 patients who suffered from traumatic brain injury. We retrospectively reviewed the clinical and radiological features. For comparative analysis, we divided the patients into 2 groups : one group with SDG after craniectomy (19 patients; 28.4% of the total sample), the other group without SDG (48 patients; 71.6%). The risk factors for developing SDG were then analyzed.

Results : The mean Glasgow Outcome Scale (GOS) scores at discharge of the groups with and without SDG were 2.8 and 3.1, respectively ($p<$ 0.0001). Analysis of radiological factors showed that a midline shift in excess of 5 mm on CT scans was present in 19 patients (100%) in the group with SDG and in 32 patients (66.7%) in the group without SDG ($p<0.05$). An accompanying subarachnoid hemorrhage (SAH) was seen in 17 patients (89.5%) in the group with SDG and in 29 patients (60.4%) in the group without SDG ($p<0.05$). Delayed hydrocephalus accompanied these findings in 10 patients (52.6%) in the group with SDG, versus 5 patients (10.4%) in the group without SDG ($p<0.05$). On CT, compression of basal cisterns was observed in 14 members (73.7%) in the group with SDG and in 18 members of the group without SDG (37.5%) ($p<0.007$). Furthermore, tearing of the arachnoid membrane, as observed on CT, was more common in all patients in the group with SDG (100%) than in the group without SDG (31 patients; 64.6%) ($p<0.05$).

Conclusion : GOS showed statistically significant difference in the clinical risk factors for SDG between the group with SDG and the group without SDG. Analysis of radiological factors indicated that a midline shifting exceeding 5 mm, SAH, delayed hydrocephalus, compression of basal cisterns, and tearing of the arachnoid membrane were significantly more common in patients with SDG.

Key Words : Subdural hygroma · Decompressive craniectomy · Traumatic brain injury · Risk factor · Cerebrospinal fluid.

INTRODUCTION

Decompressive craniectomy (DC) is an important method for managing refractory intracranial hypertension in patients with severe head injury[3,6,9]. In recent years, DC has been regarded as a last resort for reducing intracranial hypertension that is unresponsive to other medical treatments. Subdural hygroma (SDG) is a complication that can occur after head trauma and secondary to DC[14,15,20,22]. Factors contributing to the development of SDG are unknown, but this seems to be an underlying disturbance of normal CSF absorption or an alteration of the dynamics of CSF circulation. SDG can be seen in the first week after surgery, and this fluid collection may increase for up to 4 weeks[1]; however, most gradually resolve without necessitating surgical management. In some cases, SDG requires surgical intervention because it can result in neurological deterioration. SDG may be a complication of removal of the bone flap, which then distributes space in which fluid can accumulate[2,7,20,22]. SDGs can be located ipsi- or contralateral to the side of the DC, although in some cases they are bilateral or located in the interhemispheric space. The relationship between the operative technique of DC or the decompressive effect and the occurrence and pathophysiology of SDG has not been clarified. Purpose of this study was to investigate the risk factors of SDG after DC in our series.

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MATERIALS AND METHODS

From January 2004 to December 2008, DC was performed in 85 patients who suffered from traumatic brain injury. We excluded patients who had been hospitalized for less than one week. We retrospectively reviewed the clinical and radiological features and analyzed risk factors for developing SDG. We divided the patient sample into 2 groups. The first group consisted of 19 patients (28.4% of the total sample) with SDG after craniectomy (Group A), and the second group consisted of 48 patients (71.6%) without SDG (Group B). Clinical factors included gender, age, admission day, mechanism of injury, and score on the Glasgow Coma Scale (GCS). Clinical outcome was evaluated at discharge and follow-up according to the Glasgow Outcome Scale (GOS). Manner of injury and combined injuries were evaluated by examining medical records. We examined radiological features evaluated by CT scan, including midline shift, hematoma thickness, accompanying subarachnoid hemorrhage (SAH), cortical hemorrhage, hydrocephalus, compression of basal cisterns (Fig. 1), intraventricular hemorrhage (IVH), and tearing of arachnoid membrane (we determined the arachnoid-pial layer disruption that revealed traumatic cortical and arachnoid hemorrhage in same area in CT) (Fig. 2).

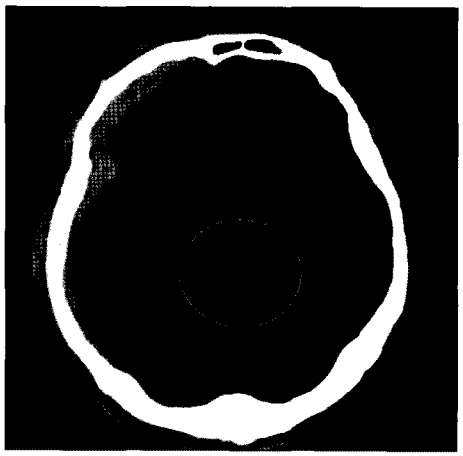

Fig. 1. Basal cistern compression on CT. No visible prepontine, interpeduncular, ambient, and suprasellar cisterns.

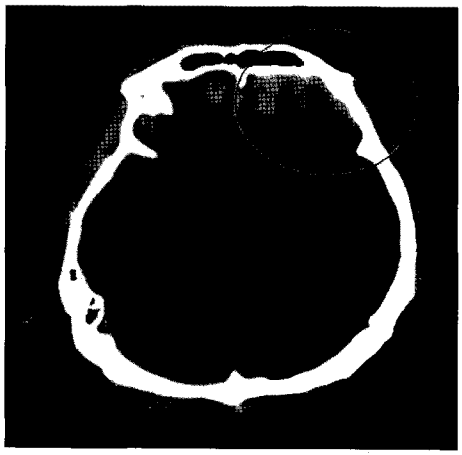

Fig. 2. Arachnoid tear, we determined the arachnoid-pial layer disruption that revealed traumatic cortical and arachnoid hemorrhage in same area in CT.

We analyzed the data using the statistical package SPSS for Windows, release 12.0. The Chi-square test, Fisher's exact test, and Student's t-test were used. p values lower than 0.05 were considered statistically significant.

RESULTS

The characteristics of the 67 patients included in the study are shown in Table 1. Group A consisted of 19 (28.4%) patients, and group B consisted of 48 (71.6%) patients. The mean age of patients in group A was 44.8 years and that of patients in group B was 50.1 years (p=0.3246). They ranged in age from 5 to 83 years. The ratio of male to female patients was 2.8 : 1 in group A and 3 : 1 in group B (p>0.05). The most frequent cause of severe head injury in all cases was motor vehicle accident (34.3%) and this was followed by slipping (25.4%) and falling (20.9%) (p=0.1780). The mean GCS score of group A was 8 and that of group B was 9.6 (p=0.1683). The GCS score at admission in group A was 3-5 in

Table 1. Demographic features of traumatic brain injury patients undergoing decompressive craniectomy

	SDG	Non SDG
Number of patients (%)	19 (28.4%)	48 (71.6%)
Gender		
Male	14	36
Female	5	12
Age		
Average (years old)	44.8	50.1
<20	1	2
20-40	6	8
41-60	7	26
>60	5	12
Mechanism of injury		
Motor vehicle accident	8	15
Fall	2	12
Slip down	5	12
Violence	0	4
Rolling	2	4
ETC	2	1
GCS		
3-5	9	12
6-8	2	10
>8	8	26
GOS		
Dead	4	11
Vegetative	4	11
Severe disability	3	3
Moderate disability	8	9
Good recovery	0	14

9 patients, 6-8 in 2 patients, and 9-15 in 8 patients. The GCS score at admission in group B was 3-5 in 12 patients, 6-8 in 10 patients, and 9-15 in 26 patients. The duration of hospital stay for group A was longer (74.1 days) than for group B (58 days).

Four patients underwent drainage of the SDG (2 burr hole, 1 subdural-peritoneal shunt, and 1 subdural-peritoneal shunt and ventricle-peritoneal shunt) and the mean GOS scores at discharge for the groups with and without SDG were 2.8 and 3.1, respectively (p<0.0001). Patients who had a good outcome (GOS of 4 or 5) constituted 42.1% of the group with SDG and 47.9% of the group without SDG.

A total of 19 patients (100%) in the group with SDG showed a midline shift exceeding 5 mm on CT, whereas a similar midline shift was observed in 32 patients (66.7%) in the group without SDG (p=0.0011). The mean hematoma thickness was 25.4 mm in the group with SDG and 17.2 mm in the group without SDG (p=0.1836). An accompanying SAH was seen in 17 patients (89.5%) in the group with SDG and in 29 patients (60.4%) in the group without SDG (p<0.0208). Hydrocephalus was observed in 10 patients (52.6%) in the group with SDG, versus 5 patients (10.4%) in the group without SDG (p<0.0001). Compression of basal cisterns on CT scan was observed in 14 patients (73.7%) in the group with SDG and 18 patients (37.5%) in the group without SDG (p<0.0075). IVH was only seen in 1 patient (5.3%) in the group with SDG but was seen in 5 patients (10.4%) in the group without SDG (p=0.6666). Cortical hemorrhage was present in 14 patients (73.7%) in the SDG group and in 37 patients (77.1%) in the group without SDG. Finally, tearing of the arachnoid membrane, as observed on CT, was more frequent in all patients in the SDG group (100%), versus the non-SDG group (31 patients; 64.6%) (p=0.7597).

DISCUSSION

SDG, which was first reported by Mayo in 1894[17], is defined as the accumulation of cerebrospinal fluid (CSF) in the subdural space due to rupture of the arachnoid membrane after head injury. SDG is one of several complications that can occur after head trauma and secondary to DC. In a study by Aarabi et al.[2], SDG developed in 25 (50%) of 50 patients after a mean of 8 days following DC. Hygromas are generally ipsilateral to the skull defect with volumes ranging from 10 to 120 mL, with a mean volume of approximately 50 mL[2]. Most gradually resolve without necessitating surgical management. However, in some cases, SDG dose require surgical intervention, due to the potential for neurological deterioration if left untreated. Yang et al.[21] reported that from a group of 169 patients who had undergone DC, 11 patients developed SDGs. In this report, the SDG spontaneously resolved in 7 patients and had to be drained in 4. Aarabi et al.[1,2] reported that 4 patients underwent SDG drainage from a group of 39 patients with SDG. In our study, 4 patients underwent drainage of the SDG (2 burr hole trephination, 1 subdural-peritoneal shunt, and 1 subdural-peritoneal shunt and ventricle-peritoneal shunt).

SDGs gradually declined and disappeared in almost cases. However, in our study, the presence of an SDG was shown to influence outcome. The mean GOS at discharge of the groups with and without SDG were 2.8 and 3.1, respectively (p<0.0001). Patients who had a good outcome (GOS of 4 or 5) constituted 42.1% of the group with SDG and 47.9% of the group without SDG.

The mechanisms underlying SDG cannot be explained completely by a single theory. The different theories that have been proposed to elucidate the pathogenesis of SDG, include arachnoid rupture, arachnoid flap, blood-brain barrier (BBB) failure, and brain atrophy[16,19]. The commonly accepted theory for SDG formation is that it is due to arachnoid membrane rupture or arachnoid flap caused by trauma, provoking CSF influx into the subdural space; this CSF is not absorbed and remains trapped because of a flap valve mechanism (the formation of a one-way arachnoid flap)[10,12,13]. The interface between the dura mater and subarachnoid space consists of an interface layer composed of an arachnoid barrier layer and a dural border cell layer[2]. Whereas a significant number of collagen fibers bond the cellular structure of the periosteal and meningeal dura, the dural border cell layer contains very little collagen, and cellular elements are loosely held together by an amorphous material prone to disruption by physical stress[8,18]. A pathologic or traumatic event can produce a cleavage in the dura-arachnoid interface in its most fragile layer, forming a new space[24]. In clinical cases, motor vehicle accidents produce shearing stress generated by kinetic energy; this can tear arachnoid barrier cells and damage the integrity of the dura-arachnoid interface layer[8]. Aarabi et al.[1] have reported that motor vehicle accidents were significantly associated with SDG following DC; however, in the present study, the results were not significant (p=0.178). By contrast tears in the arachnoid membrane on CT findings were associated with the presence of SDG (p=0.0031).

The BBB failure theory is as follows : BBB failure caused by cerebral injury increases the permeability of blood capillaries. The plasma component oozes and accumulates in the subdural

Table 2. Radiological factors of traumatic brain injury patients undergoing decompressive craniectomy

Radiologic factors	Group A (19)	Group B (48)	p
Midline shifting (>5 mm)	19 (100%)	32 (66.7%)	0.0011
Mean hematoma thickness (mm)	25.4	17.2	0.1836
Hydrocephalus	10 (52.6%)	5 (10.4%)	<0.0001
Basal cistern compression	14 (73.70%)	18 (37.50%)	<0.0075
IVH	1 (5.30%)	5 (10.40%)	0.6666
Cortical hemorrhage	14 (73.70%)	37 (77.10%)	0.7597
Tear of Arachnoid	19 (100%)	31 (64.60%)	0.0031
SAH	17 (89.50%)	29 (60.40%)	0.0208

space. The osmotic pressure increases because of the high protein content in the subdural fluid. This causes water influx into the brain tissue and arachnoid space, leading to formation of SDG, which gradually increases in volume[16]. SAH and tearing of the arachnoid membrane on CT findings may reflect damage due to BBB failure. In this study the presence of SDG was significantly associated with SAH (p=0.0208).

Finally, there was CSF circulation change theory. The CSF flows caudally into the spinal subarachnoid spaces, rostral into the basal cisternal spaces (prepontine, interpeduncular, ambient, and suprasellar cisterns), and dorsally into the subarachnoid spaces over the cortical convexities and cerebellum. Movement of the CSF via these pathways is thought to occur primarily through bulk flow with multiple contributory factors generating a current, including respiratory variation, pulsatility imparted by the cardiac pressures transduced through the circle of Willis within the basal cisterns, ciliary movement at the surface of the choroid, and production of new CSF[1,5,11]. SDGs have been shown to develop early after decompressive surgery[20]. DC alters the dynamics of CSF circulation[4,23]. This may increase the occurrence of SDGs and hydrocephalus. It was assumed that hydrocephalus, basal cistern compression, and IVH were related to alteration of the dynamics of CSF circulation in this study. There was a significant difference between the SDG and non-SDG groups with regard to hydrocephalus (p<0.0001) and basal cistern compression (p=0.007). However, there was no statistical difference between the groups with regard to IVH (p=0.6666).

CONCLUSION

GOS showed statistically significant difference in the clinical risk factors for SDG between the group with SDG and the group without SDG. Analysis of radiological factors showed that a midline shift exceeding 5 mm, SAH, delayed hydrocephalus, compression of basal cisterns, and tearing of the arachnoid membrane were statistically significant between groups. SDG is predicted to influence outcome in patients with traumatic brain injury.

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