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감압성 두개골 절제수술이 뇌수두증의 발생에 미치는 영향

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The Role of Decompressive Craniectomy in The Development of Shunt-Dependent Hydrocephalus. - Clinical Article -

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Abstract

The Role of Decompressive Craniectomy in The Development of Shunt-Dependent Hydrocephalus

Objective : Decompressive craniectomy is well known method of surgical treatment for excessive increment of intracranial pressure by intracerebral lesion. It has been reported that craniectomy after head trauma contributes to the development of hydrocephalus. But, there are no reports about it's role in intracranial aneurysm and brain tumor. This study is designed to evaluate the role of decompressive craniectomy in the development of hydrocephalus in trauma, aneurysm, and tumor surgery.
Methods : Study of the 1264 patients with head trauma, aneurysm, and tumor between Jan. 1997 and Dec. 2001 was made retrospectively by reviewing their medical record. The incidence of shunt dependent hydrocephalus was evaluated in trauma, aneurysm, and tumor patients with or without decompressive craniectomy, and analysed it with statistically..
Results : Shunt-dependent hydrocephalus was developed in 10 (2.7%) of 359 patients with trauma, 24 (6.9%) of 347 with subarachnoid hemorrhage by aneurysmal rupture, and 9 (1.6%) of 558 with brain tumor. After decompressive craniectomy, it was developed in 6 (18%) of 33 patients ($p<0.01$), 7 (63.6%) of 11 ($p<0.01$), and 3 (42.8%) of 7 ($p<0.01$), respectively.
Conclusion : These results suggest that decompressive craniectomy act as a facilitating factor, not a causing factor in the development of shunt-dependent hydrocephalus. Scalp sagging and hydrodynamic change of cerebrospinal fluid circulation may play a primary role.

Key words : Decompressive craniectomy · Hydrocephalus · Trauma · Tumor · Aneurysm.

Introduction

A decompressive craniectomy was usually applied when human life was threatened with excessively increased intracranial pressure (ICP). Up to now, decompressive craniectomy was perceived in many neurosurgical centers as a last resort for treating either uncontrollable ICP or sudden brain swelling during an operation when routine measures failed to alleviate elevated ICP or cerebral swelling associated with head trauma[20]. A hydrocephalus, developed after head trauma, was generally known to be related with a decompressive craniectomy but the relationship between the hydrocephalus after an operation for tumor and vascular disease and the decompressive craniectomy was not clear. This research was going to ascertain that the

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decompressive craniectomy for brain tumor and vascular disease including head trauma seemed to be contributed to the development of hydrocephalus postoperatively.

Materials and Methods

Subjects of this study were 1264 patients who had been treated at the neurosurgical department of KMC during the 5 year period from Jan. 1997 to Dec. 2001. They were comprised of 359, 558, and 347 patients with head trauma, brain tumor, and SAH due to aneurysmal rupture, respectively. Frequency of development of hydrocephalus after craniotomy was compared with it after decompressive craniectomy at each disease. Expired patients after an operation in each disease were excluded.

When two groups which had a decompressive craniectomy and didn't have it were compared, a variable which was able to compare with each group's characteristics of head trauma or brain tumor didn't be objectified and this was impossible to compare with it accurately. So, we made a simple comparison.

A variable about characteristics of patients of aneurysmal rupture was relatively objectified. Especially, factors that were contributed to hydrocephalus were well known. After subarachnoid hemorrhage due to aneurysmal rupture, those factors like intraventricular hemorrhage, age, sex, volume of subarachnoid hemorrhage on computed tomography (Fischer grade), the location of aneurysm, consciousness of patients on admission (Hunt-Hess grade), hyponatremia, and hypertension were contributed to develop a hydrocephalus. For subarachnoid hemorrhage, frequency of development of hydrocephalus after decompressive craniectomy was compared and analyzed allowing for influence of these factors. Relation of craniectomy and hydrocephalus using Chi-square test and multiple logistic regression analysis of statistical program Social Science Statview Package (SPSS version 11.0) was evaluated, and it was considered statistically significant with $p<0.01$ (Table 1.).

Table 1. Comparison of the Ratios of Shunt Procedure Necessitated by Patients' Condition According to the Diseases and Respective Craniectomy.

		No. of Case	No. of Shunt Patients	%	p value
Tumor	Total	551	9	1.7	
	Craniectomy	7	3	42.8	<0.01
Aneurysm	Total	336	24	7.1	
	Craniectomy	11	7	63.6	<0.01
Trauma	Total	326	10	3.0	
	Craniectomy	33	6	18	<0.01

Results

Relation with decompressive craniectomy and hydrocephalus in brain tumor

Cases that underwent shunt operation for hydrocephalus after brain tumor surgery were 9 (1.6%) out of 558 cases and hydrocephalus developed in 3 (42.8%) out of 7 cases that underwent decompressive craniectomy for the brain tumor ($p<0.01$).

Relation with decompressive craniectomy and hydrocephalus in head trauma

Cases that underwent shunt operation for hydrocephalus after surgery for head trauma were 10 (2.7%) out of 359 cases and hydrocephalus developed in 6 (18%) out of 33 cases that underwent decompressive craniectomy for head trauma ($p<0.01$).

Relation with decompressive craniectomy and hydrocephalus in aneurysmal subarachnoid hemorrahge

Cases that developed of hydrocephalus by aneurysmal subarachnoid hemorrhage were known to have high incidence of hydrocephalus when patients were advanced in age, level of consciousness of patients being in admission was lower, intraventricular hemorrhage was presented, and an aneurysm was located at the area of anterior communicating artery. According to the result of our study, cases that intraventricular hemorrhage was combined and aneurysm was located at the area of anterior

communicating artery were more frequently developed hydrocephalus but cases that patients were advanced in age and consciousness of patients being in admission was bad were the less development of hydrocephalus.

Cases that underwent shunt operation for hydrocephalus by aneurysmal subarachnoid hemorrhage were 24 (6.9%) out of 347 cases and 7 (63.6%) out of 11 cases that had decompressive craniectomy for aneurysmal subarachnoid hemorrhage had shunt operation for postoperatively developed hydrocephalus ($p<0.01$).

With comparative study through multiple logistic regression analysis to remove various factors that could influence in development of hydrocephalus, possibility of development of hydrocephalus was high more than 19 times in a group that had decompressive craniectomy ($p<0.01$).

In 7 cases that did shunt operation for hydrocephalus after decompressive craniectomy for subarachnoid hemorrhage, the proportion of males and females was almost equal (3:4), age incidence was peak in fifth decades and they were 5 cases (71.4%). As for the Hunt-Hess grade, cases were not different at each grade as 2 cases in the grade 4, 2 cases in the grade 3 and 2 cases in the grade 2. As for the Fischer grade, higher volume of hemorrhage, higher the frequency of development of hydrocephalus with 3 cases (42.8%) in the grade 4, 2 cases (28.5%) in the grade 3. But intraventricular hemorrhage was 2 (28.5%) out of 7 cases.

Discussion

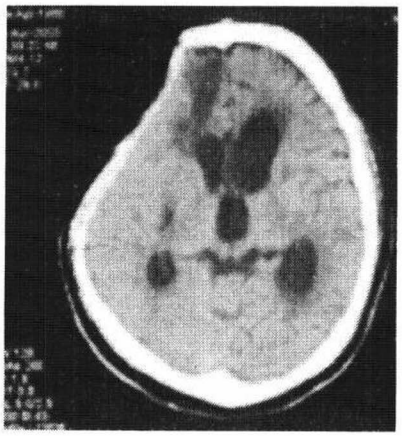
Hydrocephalus may occur via obstructive mechanisms when blood products or adhesions block cerebrospinal fluid (CSF) circulation within the ventricular system or may result from absorptive problems attributable to impaired CSF absorption at the arachnoid granulations[22].

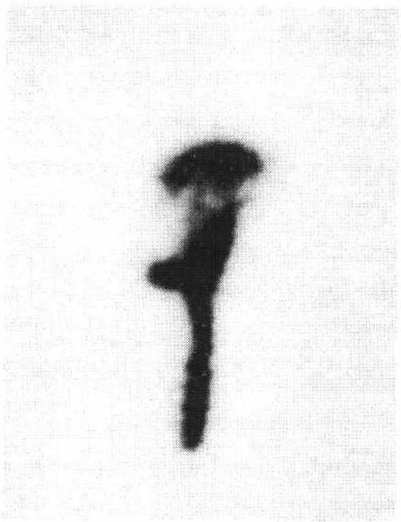
Post-traumatic ventricular dilatation might have a wide range of etiological factors : starting from neuronal loss due to head trauma and possible secondary ischemic insults, to obstruction of CSF circulation from ventricle to subarachnoid space resulting in hydrocephalus[1]. In the case of tumor, type, location and hemorrhage after an operation could cause hydrocephalus. Hydrocephalus after aneurysmal subarachnoid hemorrhage was well known and the incidence of it has been reported to range from 6 to 67%[6]. Pathogenesis of hydrocephalus after aneurysmal rupture and factors correlated with this were well known. There were no difference between man and woman on the incidence but, in cases of advanced age, worse consciousness of patients on admission[7, 21], and more volume of subarachnoid hemorrhage, hydrocephalus was more frequently developed. Especially, in cases that combined intraventricular hemorrhage developed more incidence of hydrocephalus[5, 11, 17, 19]. When aneurysm of anterior communicating artery was ruptured, frequency of intraventricular hemorrhage was high and this was known to be related with hydrocephalus[7, 11].

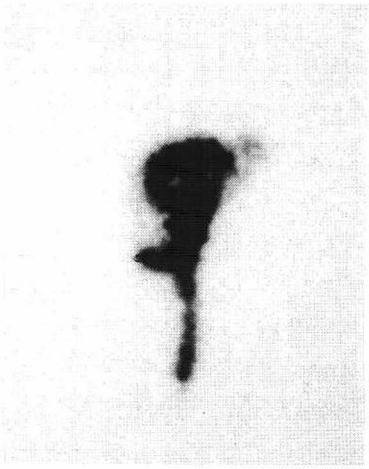
When human life was threatened due to excessively increase ICP by trauma, tumor and aneurysmal subarachnoid hemorrhage, decompressive craniectomy was usually undergone. Several weeks and months later, the hydrocephalus developed. A variety of theories have been developed regarding the role of the skull bone defect in the development of the neurological symptoms. The size and location of the cranial defect may be important[18]. Derangement of CSF hydrodynamics have been described[3], as well as regionally impaired cerebral blood flow[12]. It has also been suggested that the atmospheric pressure acts directly on the cerebral cortex in cases with a concave deformity[18]. Pheunpathom, et al[10]. was reported that decompressive craniectomy was contributed to development of hydrocephalus after head trauma and occlusion of CSF space at the part that remove skull was one of the causes. Yang, et al[20]. was also reported to relate high frequency of shunt-dependent hydrocephalus mainly to the extradural bleeding into subarachnoid space in case of the incomplete dural closure and partly to traumatic bleeding of brain contusion and subdural hemorrhage.

Pathogenesis of hydrocephalus after decompressive craniectomy could be explained by sagged scalp and dynamic change of CSF circulation[2].

Scalp sagging of the craniectomy site was found and causes of this are estimated that scalp without support sinks due to weight by gravity or elasticity of scalp itself makes scalp sunk. Some authors indicated atmospheric pressure as the cause of scalp sagging. The unsupported scalp sagged against the brain and transmitted atmosphere pressure directly to the brain to obliterate subarachnoid space underlying the cranial defect[20]. Therefore, there were seemed to be contributed to the development of hydrocephalus. When radioisotope cisternography was undergone to patients who had decompressive craniectomy, isotope activity was hardly seen at outside of ventricle that remove a skull (Fig. 1.)







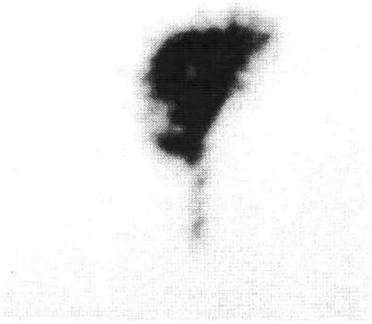


Fig. 1. A : Brain computed tomography of a patient with decompressive craniectomy in right frontotemporoparietal bone and hydrocephalus. B(after 3 hours), C(after 6 hours), D(after 24 hours) : Radioisotope cisternography of same patient showing rarely activity of isotope lateral to the ventricle in posteroanterior view.

Shapiro, et al[16]. and Hochwald, et al[8]. conducted an experiment which did decompressive craniectomy on animals for making animal model of infantile hydrocephalus. Characteristics of infantile hydrocephalus was that the extent of the ventricular enlargement was larger than adults[13]. Dynamic change of the tissue due to immaturity of infantile brain and not closed suture were causes so craniectomy on animals was done to revive dynamic change of skull not influencing as fixed container by unclosed suture[13]. Size of cat's ventricle was not ch anged after craniectomy in the experimental animals. Size

of ventricle started to be getting bigger after craniectomy when hydrocephalus were induced by Kaolin and size of ventricle was stabilized. When hydrocephalus was induced with Kaolin after craniectomy, the size of the ventricle was getting much bigger than that injecting only Kaolin. From the result of this experiment, it was found that hydromechanical changes of CSF circulation after craniectomy did not cause ventricular enlargement directly but was contributed to enlargement of ventricles[8, 15].

Hydromechanical changes of CSF circulation observed after removing a skull were an increase of volume buffering capacity (pressure-volume index, PVI), a decrease of resistance on CSF absorption, a decrease of intracranial pressure, a decrease of ICP variation, a decrease of sagittal sinus pressure and so on[4, 16]. It was known fact that these changes were reversed by doing cranioplasty[4]. Parts that were contributed to volume buffering capacity of normal brain were venous blood and CSF of cranium and spine and brain parenchyme. In a brain which removed a skull, the site that removed a skull and changed of dynamic characteristics of some damaged brain were additionally contributed to volume buffering capacity. Therefore, because obstruction of CSF circulation happened in a brain with increased volume buffering capacity rather than a normal brain, when CSF was accumulated, capacity storing accumulation was preferred more than pressure increase. Accumulated CSF volume was commonly in the ventricle so clinical manifestation of hydrocephalus is appeared by ventricular enlargement[16].

Various extent of ventricular enlargement could be seen after head trauma and subarachnoid hemorrhage. But, clinical significant hydrocephalus which need shunt operation for a relief of symptoms is not uncmmon. When factors that caused clinically meaningless ventricular enlargement and decompressive craniectomy were combined clinically meaning but ventricular enlargement requiring ventricular shunt was developed in high frequency.

In the hydrocephalus after decompressive craniectomy, increase of the ventricle size were gradually proceeded for several days and weeks were done at a time. But, Craniectomy was a factor allowing compensation of CSF circulation in the early stages[1]. Shapiro, et al[14]. attempted to offer a conceptually difficult hypothesis that the time constant (resistance to CSF outflow × compliance of cerebrospinal space) of cerebrospinal system hydrodynamics has a tendency to remain constant. Therefore, a mechanistic increase in compliance after craniectomy tended to be following by a decrease in the resistance to CSF outflow[1]. This process may be reversed after cranioplasty - that is, a decrease in PVI may be followed by an increase in the resistance to CSF outflow[1]. The second possible scenario was more important for clinical management. A large craniectomy may facilitate irreversible ventricular enlargement over weeks or months[9]. Thus, after cranioplasty, the expanded ventricles may, via the cerebral mantle, obstruct the lumen of the cortical subarachnoid space and increase the resistance to CSF outflow[1]. Ventricular enlargement was proceeded due to factors that were contributed to ventricular enlargement as time goes in the increased volume buffering capacity state, so if this state was exposed at the minimum time, the extent of ventricular enlargement could be meaningless clinically. The pattern of the CSF circulation may change dramatically after a cranioplasty resulting from a previous decompressive craniectomy for refractory intracranial hypertension after head injury[1]. The resistance to CSF outflow after craniectomy decreased twofold and brain compliance (expressed using the PVI) increased[14]. Therefore, cranioplasty decreasing volume-buffering capacity was done as soon as possible, frequency of development of hydrocephalus requiring clinically significant ventriculoperitoneal shunt could be reduced.

Conclusion

Decompressive craniectomy was not a cause of

hydrocephalus but contributed to the development of it. Therefore, when decompressive craniectomy is considered for life saving, it need to be decided carefully. Further prospective study will be need to define the correlation of shunt-dependent hydrocephalus and decompressive craniectomy

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