CLINICAL ARTICLE

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Polyuria after Surgery of Ruptured Cerebral Aneurysm : with Special Reference to the Administration of Osmotic Diuretics

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Objective : Subarachnoid hemorrhage(SAH) is commonly associated with polyuria (solute diuresis or water diuresis). The authors investigate the incidence and clinical characteristics of polyuria with special reference to the administration of osmotic diuretics.

Methods : One hundred and forty eight patients with high urine output (>200ml/hr) after ruptured cerebral aneurysm operated early from Jan 1998 to Jun 2003 were selected. Water diuresis (diabetes insipidus, DI) was differentiated from solute diuresis by lower urine specific gravity (<1.005) and higher plasma osmolality. The incidence and mode of onset of polyuria were compared between two types of diuresis. Additionally, the relationships between development of polyuria and clinical features including aneurysm location, clinical grade, Fisher grade, and outcome were analyzed. Osmotic diuretics were not routinely used in patients with Hunt-Hess grade I-III since July 2001.

Results : Annual incidence of polyuria decreased markedly since July 2001 : 45.2% in 1998, 34.5% in 2001, 11.9% in 2003. Postoperative DI occurred in 2.4~11.1%. DI developed mainly from ruptured anterior communicating artery aneurysm. The mean interval between the last SAH and the onset of DI was 7.1 days (range 1~27 days) and lasted mean 4.6 days. When compared with solute diuresis, the development of DI was significantly delayed. Other clinical features were not closely related to polyuria.

Conclusion : Uncontrolled polyuria may lead to cerebral ischemia and electrolyte imbalance because SAH patients are already predisposed to hypovolemia, and will risk precipitating the opposite situation with overhydration. We can decrease the development of polyuria without routine use of osmotic diuretics, by avoiding the increased intracranial pressure such as the intraoperative ventriculostomy and gentle brain retraction in good grade patients.

KEY WORDS : Polyuria · Subarachnoid hemorrhage · Aneurysm · Osmotic diuretics.

Introduction

Polyuria can arise from inadequate secretion of vasopressin (diabetes insipidus, DI, water diuresis) and osmotic (solute) diuresis in the neurosurgical fields. DI has a 2% incidence in patients presenting subarachnoid hemorrhage(SAH)[7] but may also occur as a result of the altered mechanism operating in this patients as a result of SAH[14].

Polyuria is relatively more common with administration of osmotic diuretics following surgery of ruptured cerebral aneurysms. Careful monitoring of intake and output, and electrolyte of serum and urine in SAH patients is essential. Significant dehydration can produce the risk of cerebral ischemia caused by vasospasm. In contrast, overtreatment with administration of antidiuretic hormone can lead to water intoxication and the danger of brain swelling or pulmonary edema. Recently the decrease of administration of osmotic diuretics coupled with advancement of microsurgical techniques and neuroanesthesia has contributed to the reduction in the development of polyuria.

The authors investigated the incidence and clinical characteristics of polyuria that may influence the postoperative critical care in patients with ruptured cerebral aneurysms for recent 5 years, with special reference to the administration of osmotic diuretics.

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Materials and Methods

One hundred and forty-eight patients who showed polyuria (>200ml/hour) after surgery of the ruptured intracranial aneurysm within 3 days after the attack from January 1998 to June 2003 were selected. Patients with inadequate salt intake, evidence of renal disease, cardiac failure were excluded. The diagnosis of DI was defined by a plasma osmolality greater than 300mOsm/kg and dilute urine of low osmolality (specific gravity<1.005). Solute diuresis had a higher urinary specific gravity (>1.009) than DI and the urine less dilute[1,6,18].

Although mannitol was used in almost all SAH patients pre-and postoperatively from January 1998 to June 2001, but we did not administrate any osmotic diuretics since July 2001 in almost all good grade patients. The cases of the mannitol

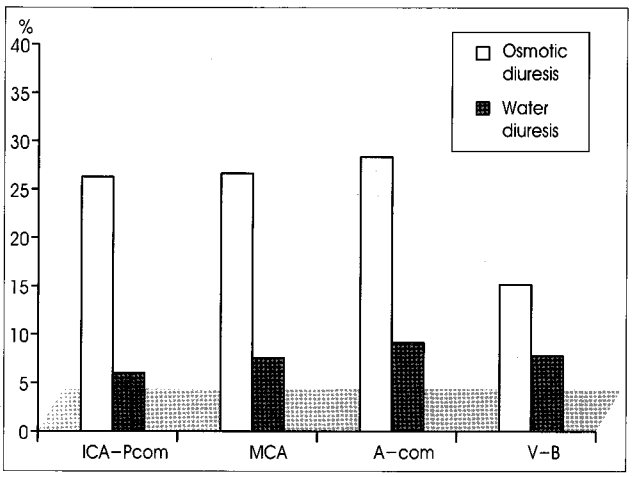

Fig. 3. Polyuria and aneurysm location. Water diuresis(DI) tend to be developed mainly from ruptured anterior communicating artery aneurysm.

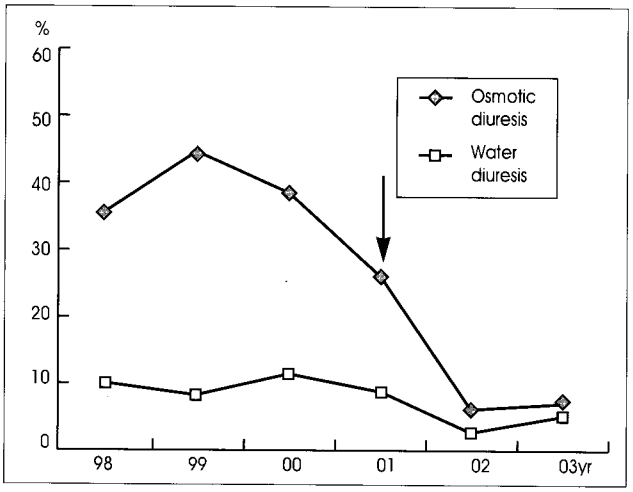

Fig. 1. The incidence of polyuria. Annual incidence of osmotic diuresis decreased markedly since 2001(arrow), but there is no significant change over the years in water diuresis.

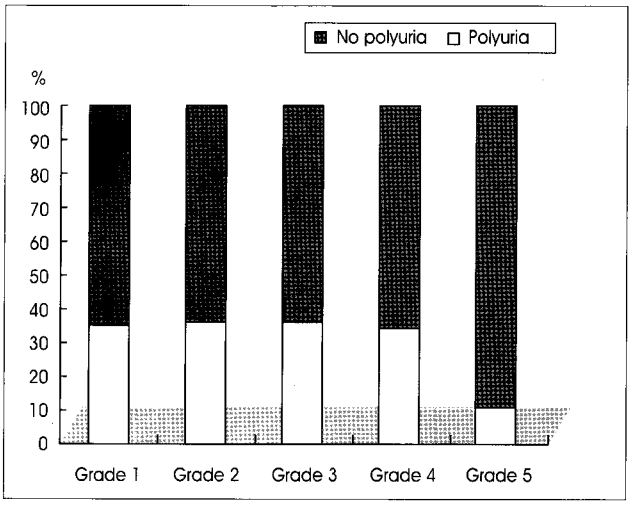

Fig. 4. Polyuria with clinical grades. The difference between clinical grades was not significant (p=0.162).

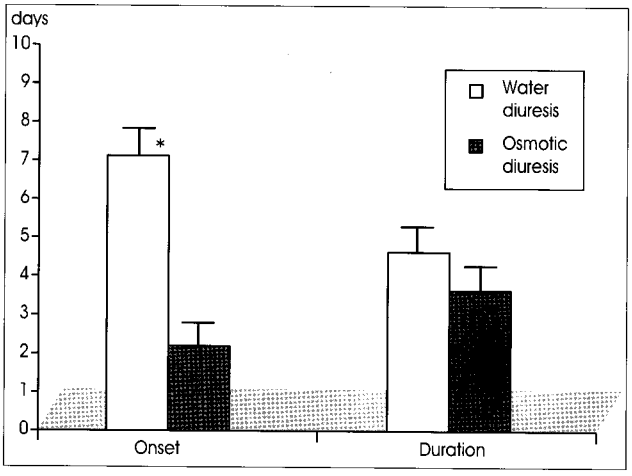

Fig. 2. The onset and duration of polyuria. When compared with osmotic diuresis, the development of water diuresis(DI) is significantly delayed(*Student's T test, p<0.05). All values are expressed as the mean plus or minus the standard error.

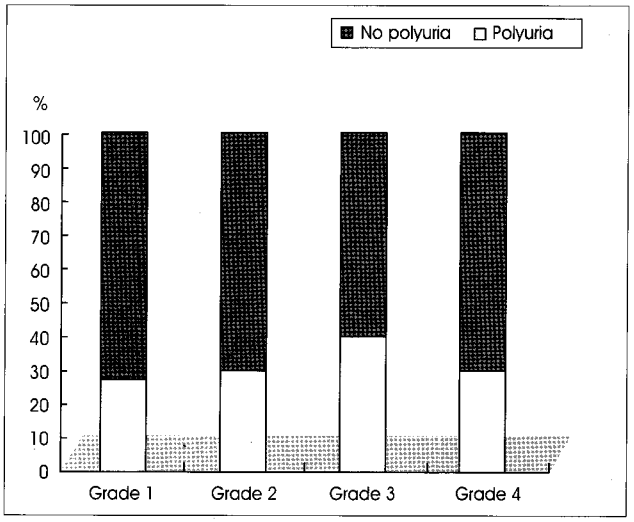

Fig. 5. Polyuria with Fisher grades. The difference between Fisher grades was not significant (p=0.355).

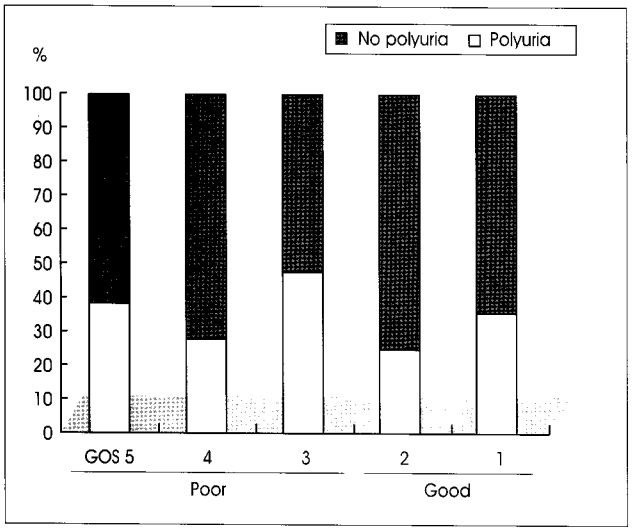

Fig. 6. Polyuria with outcome. The difference between Glasgow outcome scales was not significant (p=0.260).

administration in those patients who showed intracerebral hematoma, small ventricle, and intraventricular hemorrhage on initial computerized tomogram or swollen brain intraoperatively were excluded in this study. Initially, 15% mannitol 200ml was given over a period of 10 minutes at Emergency Room, and after the bolus injection a dose of 0.25g/kg was administered 4 times a day for 3 days to 1 week postoperatively. The annual incidence of polyuria were examined. The incidence and mode of onset of polyuria were compared between DI and solute diuresis. Additionally, the relationship between development of polyuria and clinical features including aneurysm location, clinical grade, Fisher grade, and outcome were analyzed. Patients with vasospasm were not compared because of small number of patients. Outcome was assessed at last follow-up intervals according to the Glasgow Outcome Scale with "good" or "moderate disability" classified as a good outcome and "severe disability", "vegetative" or "death" classified as a poor outcome.

The statistical significance of observed differences between the variables were assessed by Student's T test and X^2 test. A p value 0.05 or less was considered significant.

Results

Annual incidence of polyuria decreased markedly since July 2001 that started to decrease administration of osmotic diuretics : 45.2% in 1998, 34.5% in 2001, 11.9% in 2003. Postoperative DI occurred in 2.4~11.1% and there was no significant change over the years (Fig. 1). The mean interval between the last SAH and the onset of DI was 7.1±7.0 days (range 1~27 days), and the development of DI was significantly delayed (Student's T test, $p<0.05$) when compared with osmotic diuresis (2.2±2.5 days). DI lasted mean 4.6±4.2 days, and it was similar to osmotic diuresis (3.6±2.7 days) (Fig. 2). Polyuria developed mainly from ruptured anterior communicating artery aneurysm in 66 out of 177 patients (DI, 16), and it was relatively higher than middle cerebral artery aneurysm in 41 out of 120 patients (DI, 9), and internal carotid-posterior communicating artery in 38 out of 118 patients (DI, 7) (Fig. 3). Other clinical features including clinical grade (Fig. 4), Fisher grade (Fig. 5), and outcome (Fig. 6) were not closely related to polyuria.

Discussion

DI is a relatively common problem following brain injury, pituitary region tumor, and also occurs from markedly elevated intracranial pressure and the neurosurgical intensive care patients, including phenytoin use, alcohol intoxication, and bacterial meningitis[3,9,12,17]. Solute diuresis is caused by the use of osmotic diuretics such as mannitol or glycerol, hyperglycemia, mobilization of third space fluids, or fluid overload[13]. DI after a SAH or surgery of intracranial aneurysms may be related to 1) hemorrhagic or ischemic lesion to the supraoptic and paraventricular nuclei in the anterior hypot-halamus receiving their blood supply from small penetrating branches arising from the anterior cerebral and anterior communicating artery[2,5], 2) the dilated third ventricle exerting direct pressure on the hypothalamic region[4,19], 3) surgical trauma with direct tissue injury[14]. In solute diuresis after surgery of intracranial aneurysms, excessive filtration of a poorly reabsorbed solute can depress reabsorption of NaCl and water in the proximal tubule and cause their loss in the urine, producing polyuria. Additionally, an increase or decrease in osmolality is sensed by osmoreceptors, leading to enhancement or suppression of ADH secretion. However, because the interaction of deficient secretion of ADH and osmotic stress may induce high output of dilute urine after surgery of intracranial aneurysms, clinically two types of polyuria can be coexist.

The diagnosis of DI is defined as polyuria(in the adult, > 200ml/hour) in which the urine is diluted to low osmolality (specific gravity<1.005) associated with a plasma osmolality greater than 300mOsm/kg. Also, thirst is usually a prominent features if patients are conscious[18]. In contrast, solute diuresis has a higher urinary specific gravity (>1.009) than DI and the urine less dilute, but patients are not thirsty[1,6,18].

DI has a 2~3.6% incidence in patients presenting subarachnoid hemorrhage(SAH)[7,14] but it was a little higher with a incidence of 2.4~11.1% in the present study probably due to coexistence of clinically two types of polyuria after surgery of intracranial aneurysms. The incidence of polyuria after mannitol administration is not known so far. In this study,

it was relatively higher than expected with a incidence of 40% after administration of osmotic diuretics in SAH patients. Comparatively, the actual incidence of polyuria with mannitol therapy after head injury is not so higher at my experience because of normal osmotic adaptation. We found that the incidence could be reduced with the limitation of use of osmotic diuretics. The onset of DI usually is delayed following an insult or injury, as endogenous ADH will still be circulating following the event, and it usually is temporary and resolves after a few days or weeks[1,11]. This study also showed that the mean interval between the last SAH and the onset of DI was 7.1 ± 7.0 days(range 1~27 days), and the development of DI was significantly delayed (Student's T test, $p<0.05$) when compared with osmotic diuresis (2.2 ± 2.5 days). DI lasted mean 4.6 ± 4.2 days, and it was similar to osmotic diuresis (3.6 ± 2.7 days). DI developed mainly from ruptured anterior communicating artery aneurysm as the same as the other reports[4,8,10]. No previous published study has contained an analysis of the relationship between development of polyuria and other clinical features including clinical grade, Fisher grade, and outcome. We found that those clinical features were not closely related to polyuria.

Uncontrolled polyuria will lead to serious consequences and a worse or even fatal outcome because SAH patients are already predisposed to hypovolemia[16]. Accordingly, the treatment of polyuria after aneurysm surgery involves judicious fluid replacement, usually with 5% dextrose solution or other hypotonic solutions, and attention to the cerebral vasospasm. Saline solution aggravate the renal loss of water by providing a continuing solute load to the kidneys. When it is difficult to keep up with replacement, a synthetic analogue of ADH can be administered[15]. Overtreatment with ADH can rapidly lead to water intoxication with its features of mental deterioration, seizure, and pulmonary edema. In our patients, a few reversible complications developed but it did not influence the outcome with careful postoperative management of polyuria.

Conclusion

Polyuria after aneurysmal SAH is troublesome to the busy neurosurgeon and is one of the important complications which should be careful for the perioperative management. Significant dehydration can produce the risk of cerebral ischemia caused by vasospasm. In contrast, overtreatment with administration of antidiuretic hormone can lead to water intoxication and the danger of brain swelling or pulmonary edema. We can decrease the development of polyuria without routine use of osmotic diuretics, by the method of avoiding the increased intracranial pressure such as the intraoperative ventriculostomy and gentle brain retraction in good grade patients.

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