Diabetes Insipidus after Traumata of Two Extremes in Severity

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Two patients with post-traumatic diabetes insipidus (DI) are reported. One had suffered a fatal injury and the other a mild contusion without amnesia before DI developed. These two instances exemplify the wide spectrum of post-traumatic DI and, hence, the importance of ruling out DI even after a mild closed-head injury.

Key Words: Diabetes insipidus, trauma

Diabetes insipidus (DI) after trauma with or without physical damage to the cranium occurs infrequently (Blotner 1985; Machiedo et al. 1975). Ordinarily, however, a major trauma to the central nervous system results in post-traumatic DI (Porter and Miller, 1977). Two patients are presented to give various aspects of traumatata which resulted in DI; one trauma episode was fatal and the other so mild that no amnesia accompanied it.

PATIENTS’ REPORT

Both of the two patients were Japanese who had otherwise been healthy prior to the causative traumata and had no family history of DI. In the treatment of Patient 1, domestic ethical codes were duly observed because in Japan, no legislations were yet available concerning an individual’s death. An informed consent was obtained from Patient 2 to release the patient’s medical information.

Patient 1

A 25-year-old male presented to the Emergency Room of Kyushu University Hospital on ventilation support after a high-speed automobile accident. Because of an immediate respiratory arrest at the scene, the patient had been resuscitated for 20 minutes until arrival. When first seen, the patient showed cardiopulmonary arrest and dilated pupils not reactive to light. An intratracheal injection of epinephrine regained the patient’s heart beat. Subsequent resuscitation with vasopressors stabilized the patient’s vital signs but failed to recover his consciousness. Superficially, the patient had a laceration on the forehead. A roentgen study of his spine, on the other hand, revealed a dislocation of the first cervical vertebra.

Nine hours after the injury, an electroencephalogram showed no brain activity; the bilateral fundus oculi demonstrated papilledema. The patient’s urinary volume, meanwhile, increased to 1,300 ml/hour with a specific gravity of 1.004. Laboratory examinations revealed a serum sodium of 145 mEq/L, a serum glucose of 152 mg/dL, blood urea nitrogen of 9.0 mg/dL, and a serum creatinine of 1.0 mg/dL. The formula to estimate plasma osmolarity using the concentrations of sodium, glucose, and blood urea nitrogen (Ganong 1977) gave the osmolarity of 300.6 milliosmols/liter. Because an intramuscular injection of vasopressin caused a temporary decrease in the urinary volume (300 ml/hr) and increase in urinary specific gravity (1.007), the diagnosis of DI was entertained.

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Disclaimer: Because Patient 1 in this report was treated in 1983, the management of brain death in that year should not be misconstrued as what it is now in Japan.

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On Day 1, total urinary volume amounted to 13,060ml with a specific gravity of 1.007 (sodium 93.8 and potassium 29.0 mEq/L) despite frequent injections of vasopressin. The rate of infusion volume and electrolyte components were determined based on the urine output of a preceding interval. On Day 2, an intracranial pressure monitor showed a reading of 130 torr, an equivalent level of the systolic arterial pressure, indicating negligible blood delivery into the brain. The urinary volume was 9,904 ml/day.

On Day 3, the injections of vasopressin were changed to 1-desamino-8-D-arginine vasopressin (DDAVP; Desmopressin, Kyowa Hakko Kogyo Co., Tokyo, Japan; 10 μg/day) administered intranasally. The urine decreased to 5,865 ml/day as did subsequent daily urinary volumes. These volumes, with specific gravity in parentheses, marked 6,683ml (1.006) on Day 4, 4,258ml (1.007) on Day 5, and 3,238ml (1.009) on Day 6. The patient's life support was, however, discontinued with a demise on Day 7 because of prevailing sepsis.

After the administration of the DDAVP, urinary volumes decreased sequentially into 250, 250, 100, 50, and 50 ml/hr during an infusion of 5.0 percent glucose (60 ml/hr). The specific gravity of the last hourly urine was 1.020. Because the DDAVP improved polyuria with low specific gravity, post-traumatic DI was diagnosed.

At the 18th post-injury hour on Day 1, the urinary volume increased to 430 ml/hr and, therefore, DDAVP (5.0 μg) was given. Laboratory results showed a serum sodium of 141 mEq/L, a potassium of 3.7 mEq/L, a blood urea nitrogen of 10.9 mg/dl; and a creatinine of 0.4 mg/dl. The hemoglobin level was 12.7 mg/dl with a hematocrit of 35.9 percent. Then oral intake was allowed ad libitum. For the subsequent seven hours, the urinary output was 130 ml/hr. At the 25th hour, the last dose of DDAVP (2.5 μg) was given. As the headache disappeared, the patient’s polyuria and high blood pressure ceased on Day 2. She has since been well without the syndrome of inappropriate anti-diuretic hormone or anterior pituitary dysfunction.

**DISCUSSION**

Post-traumatic DI is a rare entity not only in English (Porter and Miller 1977) but also in Japanese literature (Sonoda et al. 1984). Rare as it may be, early diagnosis and prompt treatment are the mainstay for this post-traumatic complication.

Porter and Miller (1977) described a series of 18 patients with post-traumatic DI. They used the duration of amnesia to categorize trauma severity, listing one patient (5.6%) with two hours of consciousness loss. The majority of the patients, however, had durations of amnesia longer than a week, in agreement with other authors who linked injurious severity with frequent occurrence of post-traumatic DI (Machiedo et al. 1975; Morris 1978). Patient 1 of this report, therefore, exemplified one trauma of such magnitude that a brain death developed. Patient 2, on the other hand, stood out as an individual who sustained the slightest degree of trauma ever described. This patient had no amnesia, neurologic symptoms, or radiologic abnormality. The severe headache was the only symptom she complained of.

Of interest is the fact that this woman showed transient arterial hypertension. Patients with post-traumatic DI, however, frequently demonstrate low blood pressure due to exsanguination or hypovolemia caused by polyuria. The hypertension observed acutely after the injury in Patient 2 might be attributed to a derangement in the hypothalamus. Because this locus is the center of the autonomic nervous system
regulating vascular tonus, in part, and is the neurosecretory origin of the antidiuretic hormone to the posterior pituitary (Mathias 1987), the DI and arterial hypertension in parallel imply an acute, transient dysfunction of the hypothalamus. Another possible cause for the hypertension might be transient brain edema which, however, was not documented in this patient.

The interval from trauma to the onset of DI varies from a few hours to several months, with the usual onset approximately 10 days after insult (Kern and Meislin 1984). However, our two patients showed short intervals of nine hours and one hour, and Patient 2 had an extremely acute onset of DI at the first postinjury hour. To our knowledge, this patient was one of the unique instances of post-traumatic DI in whom an extremely early onset was documented after an injury of the slightest intensity, with fast recovery as well.

Usual criteria for the diagnosis of DI include polyuria, polydipsia, and consistently low urine specific gravity (Becker and Daniel 1973). The two patients presented above, however, were either too deep in a coma or too early to show polydipsia. Of prime importance in the diagnosis of complete DI are the increase in urinary osmolality in response to antidiuretic hormone, and the absence of urinary osmolality change in response to water deprivation (Levitt et al. 1984). Because both Patient 1 and Patient 2 met these requirements, complete central DI was convincing. An early diagnosis, conversely, allows an expedient, effective therapy with an exogenous antidiuretic hormone. This regimen would prevent fluctuating circulatory volume or could control brain edema by titrating vasopressin, for example, to keep the fluid balance on the modest dry side (Levitt et al. 1984).

Thus, our two patients offered contrasting examples of post-traumatic DI. Insults causative of DI are diverse, including a mild head contusion and a fatal injury. Traumatologists should, therefore, keep their minds alert to this uncommon complication when they see patients with polyuria after trauma.

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