Asymptomatic Hyponatremia in a Patient with Mild Head Injury Due to Syndrome of Inappropriate Diuretic Hormone — A Case Report —

AKIRA ISHIBASHI AND YOSHITAKE YOKOKURA*

Departments of Neurosurgery and Surgery*, Yokokura Hospital, Miike-gun 839-0295, Japan

Summary: Hyponatremia is commonly seen in patients with severe and moderate head injury, but it is rarely reported in those with mild head injury. The authors report a patient with mild head injury who presented with data typical of inappropriate secretion of antidiuretic hormone (SIADH), but showed no clinical deterioration. Though the clinical significance of this condition is unclear, the true incidence of this pathology might well be found to be higher than expected, should it receive more clinical and/or serological attention. Continuing clinical assessment will be needed to determine the significance of this condition in relation to that in patients with SIADH following the various causes reported previously.

Key words mild head injury, hyponatremia, inappropriate secretion of antidiuretic hormone, hypophysis, MRI

INTRODUCTION

Sodium and fluid imbalance are commonly reported in association with malignant tumors, infectious disease and some drugs [1]. In the central nervous system, hyponatremia related to subarachnoid hemorrhage or severe and moderate head injury has frequently been identified. However, it rarely occurs in patients with mild head injury. Most patients hospitalized with mild head injury are discharged from hospital with little or no medical attention.

In the present report, the authors describe an unusual case with mild head injury who presented with laboratory data typical of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), but had no clinical deterioration.

CASE REPORT

A 68-year-old woman sustained a closed head injury in a motor vehicle accident. Initially she had loss of consciousness for 20 min. One hour later at our hospital the Glasgow Coma Scale was 14. Plain skull X-ray showed a linear fracture of the right occipital bone. CT-scan demonstrated a thin subdural hematoma in the left longitudinal fissure and subarachnoid hemorrhage in the left site of the sylvian fissure. Localized left fronto-temporal cerebral contusions were also identified (Fig. 1a, b). No increase

Fig. 1. a, b: Initial CT taken on the day of admission demonstrated a cerebral contusion in the left fronto-temporal region, thin subdural hematoma in the left longitudinal fissure and subarachnoid hemorrhage in the left site of the sylvian fissure.
in the size nor the number of the abnormal density
areas were observed on CT-scan taken three hours
later. Her initial blood pressure was 150 and 92
mmHg and her plasma electrolytes on admission
were as follows: Sodium concentration 144 mEq/L,
potassium concentration 3.5 mEq/L, and chloride
concentration 111 mEq/L. Three days later, she was
noted to have a decreased serum sodium concentra-
tion of 131 mEq/L, and a chloride concentration of
3.4 mEq/L. In the following three weeks, serum
sodium concentration decreased to 119 mEq/L and
chloride concentration to 89 mEq/L. Urinary sodium
excretion was 78 mEq/L. Serum osmolarity was 252
mOsm/kgH2O, and urine osmolarity was 639
mOsm/kgH2O. The urine and serum osmolarity find-
ings showed that urine was hypertonic to the serum.
Body weight throughout the period of study
remained almost unchanged. Her thyroid and renal
function was normal serologically and she had no
liver nor heart failure clinically.

Low serum sodium concentration and chloride
concentration lasted for a further two weeks, and did
not return to normal values, despite the fact that she
was receiving sufficient food and fluids to maintain
her general condition. She remained conscious and
alert without any neurological deficits. Magnetic
resonance imaging (MRI) taken on the 26th day of
admission did not demonstrate any morphological
changes of the hypophysis and hypothalamic region,
but only the localized cerebral contusion (Fig. 2).
She was diagnosed as having data typical of SIADH,
though she did not have the clinical symptoms of
SIADH. Demeclocycline was administered, and
seven days later serum sodium concentration had
risen to 135 mEq/L. She was discharged from hos-
pital and returned to her normal daily life.

DISCUSSION

Schwartz [3] described hyponatremia and hypo-
osmolarity in association with bronchial carcinoma
in 1957, and these conditions in association with
neurosurgical disease have been well discussed for
many years. Recently there have been several reports
of head injury causing the syndrome of inappropriate
secretion of antidiuretic hormone (SIADH). In their
retrospective study, Dočzi et al. [1] divided patients
with head injury into three groups according to the
degree of injury; Mild injury characterized by cer-
bral concussion and lineal skull fracture, moderate
injury by longer periods of unconsciousness with
some neurological signs, and severe injury by
prolonged unconsciousness and severe neurological
deficits. In their 1,808 consecutive patients, SIADH
occurred in 0.6% of the patients with mild head
injury, 10.6% of those with moderate injury, and
4.7% of the patients with severe head injury. They
remarked that because of low fluid intake, SIADH
was less frequently seen in the patients with severe
head injury. In a prospective study, Lee [4] assessed
a series of 1,812 consecutive patients with mild head
injury and found only three (0.2%) patients that had
deteriorated due to SIADH. It may be stated that the
risk of SIADH resulting from mild head injury may
range approximately from 0.2% to 0.6%.

As to the pathophysiological mechanism for
hyponatremia following severe head injury, it is
traditionally stated that hypothalamic-pituitary hypo-
function may be the factor responsible for the
development of SIADH [2,5], as it causes a resetting
of the osmoreceptor, and consequently results in a
release of ADH at lower serum osmolarity leading to
clinical deterioration [1,6]. In our case, laboratory
inclusion criteria (plasma hypo-osmolarity, hypona-
tremia, and urinary hyperosmolarity, normonaturia)
indicated the existence of SIADH, though MRI did
not show any abnormal intensity area in the
hypothalamic or pituitary region. Furthermore, the
patient’s clinical course was uneventful and she did
not have any clinical deterioration during hospital-

Fig. 2. Parasagittal gadolinium-enhanced T1-
weighted image showed a small hyperintense lesion
suggestive of cerebral contusion, but demonstrated
no abnormal intensity signal in the pituitary or
hypothalamic region.
ization.

Most patients with mild head injury who visit hospital, are usually sent home after a short period of hospitalization or observation without further clinical and/or laboratory assessment. Therefore, the true incidence of asymptomatic hyponatremia due to SIADH resulting from minor head injury is uncertain, but might be higher than expected if more attention were paid to the possible occurrence of this condition.

The clinical significance of this condition remains to be debated, and further prospective studies are needed to determine whether or not this condition has any relationship with that in patients with symptomatic SIADH originating from various causes reported previously [1].

REFERENCES

1. Zafonte RD, and Mann DNR. Cerebral salt wasting syn-
2. Papastolou C, Mantzoros C, Evagelopoulou C, Moses AC, and Kleefield J. Imaging of the sella in the syn-
3. Schwartz WB, Benet W, Curelop S, and Barter FC. A
syndrome of renal sodium loss and hyponatremia prob-
ably resulting from inappropriate secretion of antidiuretic
4. Lee ST, Wong CW, Yeu YS, and Tzaan WC. Relative
risk of deterioration after mild closed head injury. Acta
5. Doczi T, Tarjanyi J, Huszka E, and Kiss J. Syndrome of
inappropriate secretion of antidiuretic hormone (SIADH)
6. Robertson GI. The physiopathology of ADH secretion.
In: Clinical Neuroendocrinology: a pathological approach,