Ventricular enlargement due to acute hypernatremia in a patient with a ventriculoperitoneal shunt

Case report

ROBERT H. ANDRES, M.D.,1,2 ARJUN V. PENDHARKAR, B.S.,1 DOMINIQUE KUHLEN, M.D.,2 AND LUIGI MARIANI, M.D.2,3

1Department of Neurosurgery, Stanford University Medical Center, Stanford, California; 2Department of Neurosurgery, University of Berne; and 3Department of Neurosurgery, University of Basel, Switzerland

Patients requiring CSF shunts frequently have comorbidities that can influence water and electrolyte balances. The authors report on a case involving a ventriculoperitoneal shunt in a patient who underwent intravenous hyperhydration and withdrawal of vasopressin substitution prior to scheduled high-dose chemotherapy regimen for a metastatic suprasellar germinoma. After acute neurological deterioration, the patient underwent CT scanning that demonstrated ventriculomegaly. A shunt tap revealed no flow and negative opening pressure. Due to suspicion of proximal shunt malfunction, the comatose patient underwent immediate surgical exploration of the ventricle catheter, which was found to be patent. However, acute severe hypernatremia was diagnosed during the procedure. After correction of the electrolyte disturbances, the patient regained consciousness and made a good recovery. Although rare, the effects of acute severe hypernatremia on brain volume and ventricular size should be considered in the differential diagnosis of ventriculoperitoneal shunt failure. (DOI: 10.3171/2009.10.JNS09845)

KEY WORDS • hypernatremia • electrolyte shifting • brain volume • hydrocephalus • ventriculoperitoneal shunt

Ventriculoperitoneal shunt failure in patients with obstructive hydrocephalus represents a potentially life-threatening condition that must be investigated immediately. Clinical symptoms suggestive of increased ICP following acute neurological deterioration are often associated with VP shunt malfunction. These symptoms may include headache, nausea, vomiting, impaired level of consciousness, and incontinence. Typically, CT or MR imaging will demonstrate ventriculomegaly, and percutaneous access to the VP shunt system will usually reveal increased ICP and/or impaired CSF flow from the ventricular catheter.

Comorbidities that may influence water and electrolyte balances are common in patients with VP shunts. Acute hypernatremia can give rise to rapid shifting of transcellular fluid, leading to a decrease in brain water content and neurological deterioration. We present the case of a patient in whom classic signs of VP shunt failure developed, including ventriculomegaly and failure of CSF to aspirate on shunt tap, due to acute severe hypernatremia.

Case Report

History and Examination. We report on a 28-year-old man with a history of pilocytic astrocytoma of the quadrigeminal plate and complete remission after 56-Gy proton beam radiotherapy. Prior to radiotherapy, he had undergone VP shunt placement to treat obstructive hydrocephalus. Ten years later, the patient presented with vision loss and panhypopituitarism due to a rapidly progressing suprasellar mass lesion causing optic chiasm compression. After establishing the diagnosis of a germinoma by open biopsy, the mass was treated using radiotherapy, with a total 45 Gy to the sellar region. Full hormone replacement was established, including desmopressin substitution for central diabetes insipidus. Two years later, multiple supra- and infratentorial leptomeningeal metastases of the germinoma were found on a routine follow-up MR imaging study. The patient was subsequently admitted to a medical ward for chemotherapy. Serum sodium and chloride levels on admission as well as serum osmolality were within normal limits. The ventricle widths were normal on MR imaging (Fig. 1A).

Treatment. To minimize the nephrotoxicity associated with the scheduled cisplatin-based chemotherapy,
the patient underwent hyperhydration, with 1000 ml of intravenous 0.9% saline administered over 24 hours. Following hydration, serum sodium and osmolality were 128 mEq/L and 259 mOsm/L, respectively. To correct the hyponatremia, desmopressin was not administered the next morning. Serum sodium and osmolality were found to be 139 mEq/L and 284 mOsm/L, respectively. The patient subsequently developed a rapidly progressive disturbance of consciousness and cognitive dysfunction. Within 4 hours, the patient was found to be deeply comatose (Glasgow Coma Scale score of 7). Cranial CT scanning was immediately performed and demonstrated prominent ventriculomegaly (Fig. 1B). With suspicion of VP shunt malfunction, probably arising from obstructive debris originating from leptomeningeal tumor dissemination, we cannulated the shunt tap reservoir with a 25-gauge needle connected to a water column manometer. A negative ICP was measured, and no CSF could be aspirated using a 2-ml syringe. Because of the patient’s poor neurological status, prominent ventriculomegaly, and absence of severe electrolyte disturbances when assessed 4 hours prior, serum electrolytes were not analyzed at this time, and the patient was taken to the operating room immediately for VP shunt exploration.

Operation. Intraoperatively, the suspected VP shunt system malfunction could not be confirmed. The ventricular catheter was found to be patent and the distal shunt system was functioning normally. The ventricular catheter was replaced, but still no CSF could be aspirated. The cortical surface was not bulging through the bur hole but was instead detached by some millimeters from the inner dural surface, suggesting brain “shrinking” and intracranial hypotension. We noted an exceptionally hard consistency of the brain parenchyma. Intraoperative analysis of serum electrolytes and osmolality was performed, and sodium and osmolality were found to be 169 mEq/L and 348 mOsm/L, respectively.

Postoperative Course. The patient had suffered severe acute hypernatremia resulting from hyperhydration and inappropriate correction of electrolyte imbalance by desmopressin withdrawal. The hypernatremia was slowly corrected over the next 3 days, and the patient’s neurological status improved rapidly. Three days after surgery CT scanning demonstrated nearly normalized ventricular widths and restoration of brain volume (Fig. 1C), and serum sodium and osmolality were 139 mEq/L and 293 mOsm/L, respectively. The patient recovered with no lasting neurological deficits, and chemotherapy was performed as planned, with strict volume and electrolyte control.

Discussion

In the healthy individual, serum sodium concentration is the major determinant of plasma osmolality. This balance is closely regulated by water homeostasis, which is controlled by thirst and antidiuretic hormone (arginine vasopressin) release. A disruption in the water balance thus results in disturbances of serum sodium concentration. Hypernatremia, usually defined as a serum sodium level exceeding 145 mEq/L, frequently develops in hospitalized patients as an iatrogenic condition. Sodium is an extracellular cation that is functionally unable to cross the cell membrane. As a result, this molecule contributes significantly to tonicity and can induce shifting of water across cellular membranes. Hypernatremia, then, is a state of hypertonic hyperosmolality and can present as marked cellular dehydration.

The brain is particularly vulnerable to changes in osmotic homeostasis. The most serious complications arise from inappropriate changes in brain volume, with hyperosmotic states resulting in volume depletion and hypovolemic states resulting in edema and volume expansion. Acute hypernatremia is known to result in a decrease of brain volume, as demonstrated by hypertonic saline loading in experimental animals. Secondary tearing of bridging veins leading to hemorrhage has also been reported. Reports of this phenomenon in patients are very rare. A case of ventricular enlargement and intracranial hypoten-
sion has been described in a 3-year-old child with severe hypernatremia, while severe hypernatremia resulting in brain shrinkage with predominant widening of the subdural space has been reported in the case of a 73-year-old patient.

In our case, although the serum sodium level after desmopressin withdrawal for correction of the hyponatremia was within normal limits (139 mEq/L), we hypothesize that the rapid correction of the hypoosmotic state (259–284 mOsm/L within 12 hours) was responsible for the initial neurological deterioration. Furthermore, hypothalamic regulation of osmotic homeostasis might be severely disturbed in this patient after high-dose radiotherapy to the quadrigeminal and sellar regions. However, because only one electrolyte analysis was performed between desmopressin withdrawal and emergency surgery, information on the temporal dynamics of electrolyte and osmolarity changes is not available. The decision to intervene operatively in patients with symptoms suggestive of VP shunt malfunction is ultimately based on the severity and trajectory of the patient’s condition. Poor flow of CSF on shunt tap has been shown to be highly predictive of obstruction of the proximal catheter. However, negative ICP and difficulty in aspiration of CSF may also be present in cases of acute severe hypernatremia with intracranial hypotension. As in our patient, these findings can therefore lead to unnecessary surgical interventions. In our opinion, the failure to aspirate CSF in our case could mainly be due to several different reasons. First, the aspiration pressure at the tip of a 25-gauge needle connected to a 2-ml syringe is low and might not be sufficient to allow aspiration against the pressure gradient if the ICP is negative. Second, the shunt reservoir was tapped with a needle connected to a water column manometer to allow measurement of the opening pressure. It is possible that the negative ICP initially resulted in aspiration of air into the ventricular system through the open water column manometer, later resulting in failure to aspirate CSF. Third, the strikingly increased consistency of the brain parenchyma due to dehydration may have led to stran-gulation of the ventricular catheter. Although we cannot completely exclude the possibility of shunt malfunction contributing to neurological deterioration, normal findings at revision and the presence of severe acute hypernatremia explaining the comatose state make it unlikely that shunt revision was significantly involved in reversing the patient’s neurological decline. If possible, given the clinical condition of the patient, serum electrolytes and osmolality should be determined prior to surgery, particularly in patients predisposed to electrolyte disturbances. However, surgical intervention in cases of comatose patients with acute neurological deterioration and findings consistent with VP shunt failure should not be delayed due to pending laboratory results.

Conclusions

Our case demonstrates that reversible brain shrinkage and compensatory widening of the ventricular system may occur in a dramatically rapid fashion in response to severe acute hypernatremia and may lead to unnecessary revision surgery in patients with VP shunts. Acute electrolyte disturbances should therefore be considered in the differential diagnosis in these cases, particularly in predisposed patients.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: RH Andres. Acquisition of data: RH Andres, D Kuhlen. Analysis and interpretation of data: RH Andres, A Pendharkar, L Mariani. Drafting the article: RH Andres. Critically revising the article: RH Andres, A Pendharkar, D Kuhlen, L Mariani. Final approval of the article: RH Andres.

References


Please include this information when citing this paper: published online November 13, 2009; DOI: 10.3171/2009.10.JNS09845.
Address correspondence to: Robert H. Andres, M.D., Department of Neurosurgery, Stanford University Medical Center, 1201 Welch Road, MSLS P304, Stanford, California 94305. email: randres@stanford.edu.