The Management of Postoperative Diabetes Insipidus

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The usual response to a major surgical procedure is temporary oliguria which persists for several days.\(^1\)\(^3\)\(^12\)\(^17\) The secretion of antidiuretic hormone (ADH) that produces this oliguria is, in a sense, an “inappropriate” secretion, in that it is not controlled by osmotic stimuli.

After surgical manipulation in the sellar and suprasellar areas, however, the suprapontico-hypophyseal system may be temporarily or permanently damaged, so that a transient or permanent diabetes insipidus results. The coexistence of uncontrolled polyuria with postoperative sodium retention, or with possible excessive sodium loss in patients with prolonged hypopituitarism, may make the management of these patients very difficult. Two cases will be described to illustrate some of these problems.

In the charts illustrating these cases, “crude” water balance indicates only fluid intake and urine output. Insensible fluid loss, water of oxidation, and water in the food are not indicated. However, when this balance is considered along with changes in body weight, a good first approximation of the actual water balance can be made.

Corrected serum sodium concentration, or osmolality, indicates the ratio between body water and body electrolytes and tends to vary directly with changes of exchangeable electrolytes and inversely with changes of total body water.\(^5\)\(^9\)

Case Reports

Case 1. In November, 1955, a chromophobe adenoma of the pituitary was partially removed from a 30-year-old man with hypopituitarism. This procedure was followed by radiation therapy. The patient was placed on a maintenance dose of thyroid hormone and cortisone. In June, 1965, at the age of 40, he reported progressive loss of vision.

On September 30, 1965, under general anesthesia, a recurrent chromophobe adenoma of the pituitary was partially removed and the optic nerves and chiasm decompressed. The immediate postoperative course was uncomplicated except for marked polyuria and polydipsia. The maintenance dose of thyroid hormone and cortisone was continued. Initially, no specific treatment was given for the diabetes insipidus, and water balance was maintained by increasing the intake. A slight deficiency soon developed, however, as can be seen in Fig. 1. Treatment with pituitary “snuff” was started on October 6 and urinary volume decreased slightly. Intermittent injections of Pitressin\textsuperscript{®} tannate in oil were begun on October 9 and resulted in definite decrease in urinary volume and slight weight gain.

The patient had been alert and ambulatory until the evening of October 11 when he suddenly became drowsy and incoherent. Serum sodium concentration that morning had been 122 mEq./L; at the time of this sudden drowsiness it was 114 mEq./L. Therapy during the night consisted of 2 liters of normal saline. On the following morning, 500 cc. of 3% sodium chloride were given with definite improvement of the patient’s condition and an increase in his serum sodium concentration to 128 mEq./L. by noon of October 12. Lumbar puncture at that time revealed a slightly xanthochromic fluid under a pressure of 175 mm. of water. Also on October 12, there was definite negative water balance and subsequent weight loss. Serum sodium concentration rose to 145 mEq./L. on the morning of October 13. Despite these changes, the patient’s alertness and orientation did not return to the prehypotonic state for several days. Beginning October 12, 9-alpha-fluoro-hydrocortisone was administered daily in addition to hydrocortisone. Intermittent injections of Pitressin\textsuperscript{®} tannate in oil were given (Fig. 1). Radiation therapy was started on October 22.

Comment. In this case, symptomatic hypotonicity was precipitated by the postoperative treatment. The patient stayed in reasonably good water balance with a maximum daily fluid flux of 9.5 liters. Treatment was started with pituitary “snuff” followed by Pitressin\textsuperscript{®} tannate in oil, mainly because of the inconvenience of the large fluid flux. Water retention and weight gain occurred, culminating in stupor and a serum sodium concentration of 114 mEq./L.

Two factors appear to have caused the
hyponatremia in this case, namely excessive postoperative sodium loss and positive water balance induced by administration of ADH. The 24-hour urinary sodium excretion at the time of the symptomatic hyponatremia was approximately 300 mEq. This patient had longstanding hypopituitarism. While the control of aldosterone secretion and sodium metabolism are partially independent of control by the pituitary, and while aldosterone output and sodium conservation are generally intact in association with pituitary lesions, aldosterone may be decreased and the ability to conserve sodium may be impaired in patients with prolonged hypopituitarism.

Thus, a combination of negative sodium balance over several days followed by a shorter period of water retention (dilution) precipitated acute symptomatic hypotonicity (hyponatremia). Treatment with 0.9% saline was relatively ineffective, but administration of 3% saline promptly restored body fluid tonicity to normal levels and improved the patient’s condition. The synthetic salt-retaining hormone, 9-alpha-fluoro-hydrocortisone, was then added to the clinical regimen.

Case 2. A 25-year-old man who had had a craniotomy in 1954 for a craniopharyngioma, reported an increase in the number and severity of his headaches, progressive visual loss, and convulsive seizures. Since his craniotomy he had had panhypopituitarism and had been receiving testosterone, cortisone and thyroid extract. Pneumoencephalography revealed a prepontine mass as well as a suprasellar mass that indented the 3rd ventricle.

On November 17, 1965, a right frontal craniotomy was performed and a craniopharyngioma was partially removed. Replacement therapy included hydrocortisone, 100 mg, prior to the operation and 50 mg. every 6 hours postoperatively. The patient did not regain full consciousness after operation.

Several hours after operation, diabetes insipidus was recognized and careful monitoring of
body weight and fluid intake and output was carried out, with fluid intake and urine output being determined every 30 minutes. Under this regimen, good fluid balance was maintained (Fig. 2) in spite of urinary outputs often greater than 1 liter per hour. Some sodium was administered in the intravenous fluids on the operative day; very little sodium was administered on the following day. Serum sodium concentration was 110 mEq./L on the morning of the 1st postoperative day and again at 5:00 pm. Analysis of 12-hour urinary specimens revealed that, while the sodium concentration in the urine was relatively low, the large volume of urine resulted in an actual loss of sodium amounting to approximately 450 mEq./24 hours. In addition, some unmeasured previous sodium loss had occurred during and immediately after the operation. When this situation was recognized, replacement was carried out intravenously with 3% sodium chloride. The serum sodium concentration was raised to 141 mEq./L by 9:00 pm and to 142 mEq./L by the following morning.

During that night, however, and early the next morning, the patient had several cardiac arrests. He died at approximately 9:00 am. on the 2nd postoperative day without ever having regained full consciousness.

Autopsy demonstrated a large subdural hematoma in the operative site.

Comment. In this case, severe diabetes insipidus was evident within several hours of completion of the operative procedure and urine output had reached a rate of greater than 1 liter per hour. There was excellent monitoring of fluid balance with rapid accurate replacement of losses as charted in Fig. 2; the adequacy of replacement was confirmed by the relatively minimal weight loss of 1 Kg. on the morning of the first postoperative day. A significant amount of sodium was administered during the operative procedure, but very little was given afterwards. This patient, who had prolonged hypopituitarism, apparently also had impaired ability to conserve sodium, since he excreted a total of 460 mEq. of sodium during the first 24 hours after operation. Hypotonicity (hyponatremia) appeared to be due mainly to this sodium loss and was rapidly corrected with 3 per cent sodium chloride.

Management

Postoperative diabetes insipidus must be recognized before it can be treated. It may be overlooked in the immediate postoperative
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period if the patient is incontinent and in an impaired state of consciousness due to anesthesia or the brain lesion. If it is missed, severe hypertonicity may develop rapidly due to water deficit. Administration of hypertonic osmotic agents during the operative procedure to decrease brain mass may result in marked diuresis for several hours postoperatively which may be confused with the early onset of diabetes insipidus.

Once postoperative polyuria is recognized, both water and salt balance must be considered.

Water Balance. During the early postoperative period, we generally prefer to withhold Pitressin® and replace fluid volume for volume.* The volume and specific gravity of urine should be determined hourly (or even every half-hour as was done in Case 2) and fluids should be replaced as needed. The patient should be weighed carefully every 12, or even every 8, hours. The diabetes insipidus may be transient, or there may be an interphase of decreased urinary volume followed by the return of permanent diabetes insipidus.

If urinary volumes are very large and difficult to replace because of nausea, difficulty with intravenous administration, etc., Pitressin® should be used to decrease the urinary volume. We usually prefer to begin treatment with a single subcutaneous or intramuscular injection of 5 units of aqueous Pitressin®. This amount generally acts for several hours during which urinary volume is determined hourly. Additional Pitressin® is then given as needed; 5 units whenever the hourly volume is greater than 200 ml. This method permits prompt recognition of termination of the diabetes insipidus. When Pitressin® is given, intake of water (if it has been high) must be restricted, especially if it is being administered intravenously. If this is not done, rapid water intoxication (dilutional hypotonicity) may be induced. When prolonged or permanent diabetes insipidus is evident, Pitressin® tannate in oil may be more convenient. Injection of 2.5 to 5 units intramuscularly is usually effective for 1 to 3 days.

Pituitary “snuff” may be applied to the nasal mucosa. If nasal irritation or systemic reactions occur, a synthetic vasopressin administered by nasal spray may be substituted. Electrolyte Balance. The second problem in the management of postoperative diabetes insipidus is the electrolyte balance. The usual response to craniotomy is sodium conservation and a positive balance if large amounts of sodium are administered.

Thus, replacement of large water losses in diabetes insipidus with salt-containing fluids may cause retention of large quantities of sodium and resultant severe hypertonicity. When this usual postoperative sodium conservation is in effect, most of the fluid replacement should be dextrose in water. We administer 1000 to 1500 cc. of 0.45 per cent sodium chloride every 24 hours to replace the usual electrolyte losses.

The ability to conserve sodium may be impaired in some patients with prolonged hypopituitarism. In these patients, large losses of electrolytes may cause severe hypotonicity quite rapidly as in Case 2, or more slowly as in Case 1. This possibility must be considered during the postoperative period in any patient with prolonged hypopituitarism. Urinary sodium concentration and serum sodium concentration should be determined frequently with prompt replacement of the lost sodium. Severe hypotonicity is best treated with hypertonic sodium chloride, either 3 per cent or 5 per cent.

Summary

We have reported two cases of postoperative diabetes insipidus to illustrate problems in the management of fluid and electrolyte balance. To avoid excessive sodium retention and hypertonicity, most of the fluid replacement in the usual situation should not contain electrolytes. In patients with prolonged hypopituitarism, postoperative sodium conservation may be defective, leading to hypotonicity if the sodium losses are not recognized and replaced.

References


