

LAPORAN KASUS

Management Of Diabetes Insipidus After Sublabial Transsphenoidal Hypophysectomy Surgery

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ABSTRACT

A 36-year-old female patient was diagnosed with diabetes insipidus after sublabial transsphenoid hypophysectomy (SLTH) surgery. The patient had pituitary adenoma. The patient undergoes 14 days of care in the ICU with titrated intravenous vasopressin dose(0,01-0,3unit/hour) and later subcutaneous dose(6-13unit/8 hours). Subcutaneous vasopressin started on day 3 while intravenous was tapering down; at the early transition from the intravenous vasopressin route to the subcutaneous vasopressin route on day 7, there is a sharp surge of urine production as well at plasma sodium level. The intravenous vasopressin started again, along with the elevated dose of subcutaneous vasopressin. The patient shows a response to therapy after a watchfully titrated dose.

KEYWORDS: -

Background

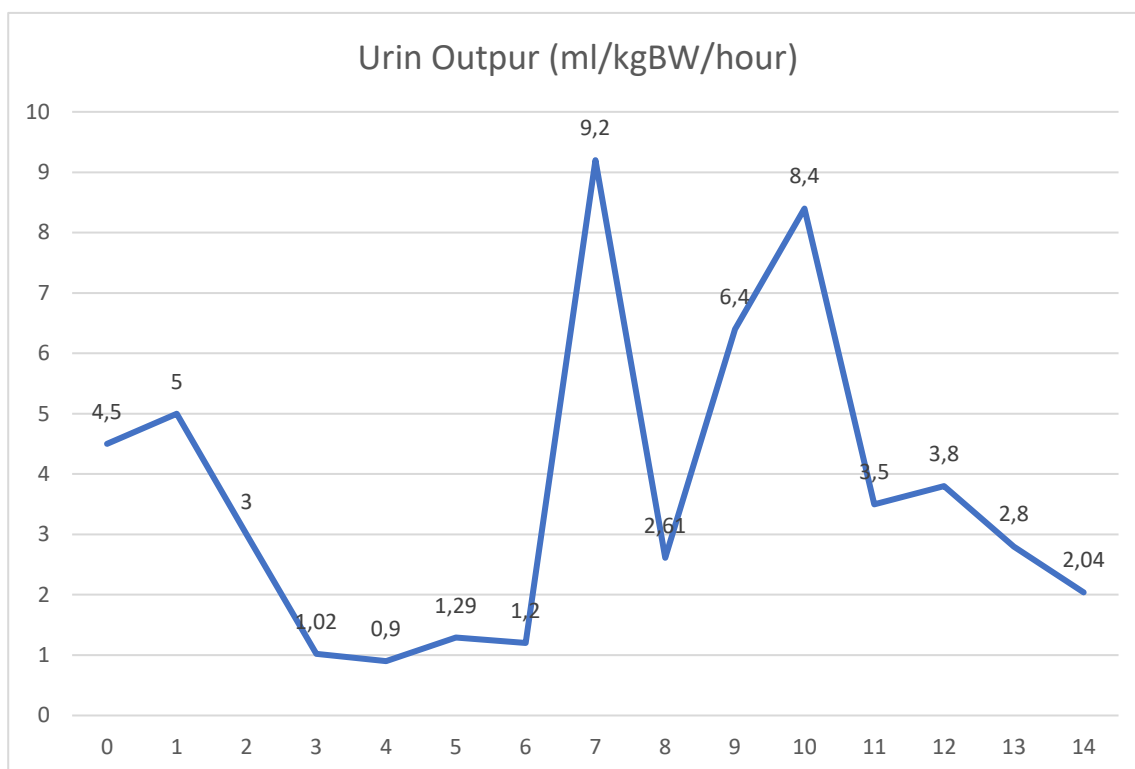
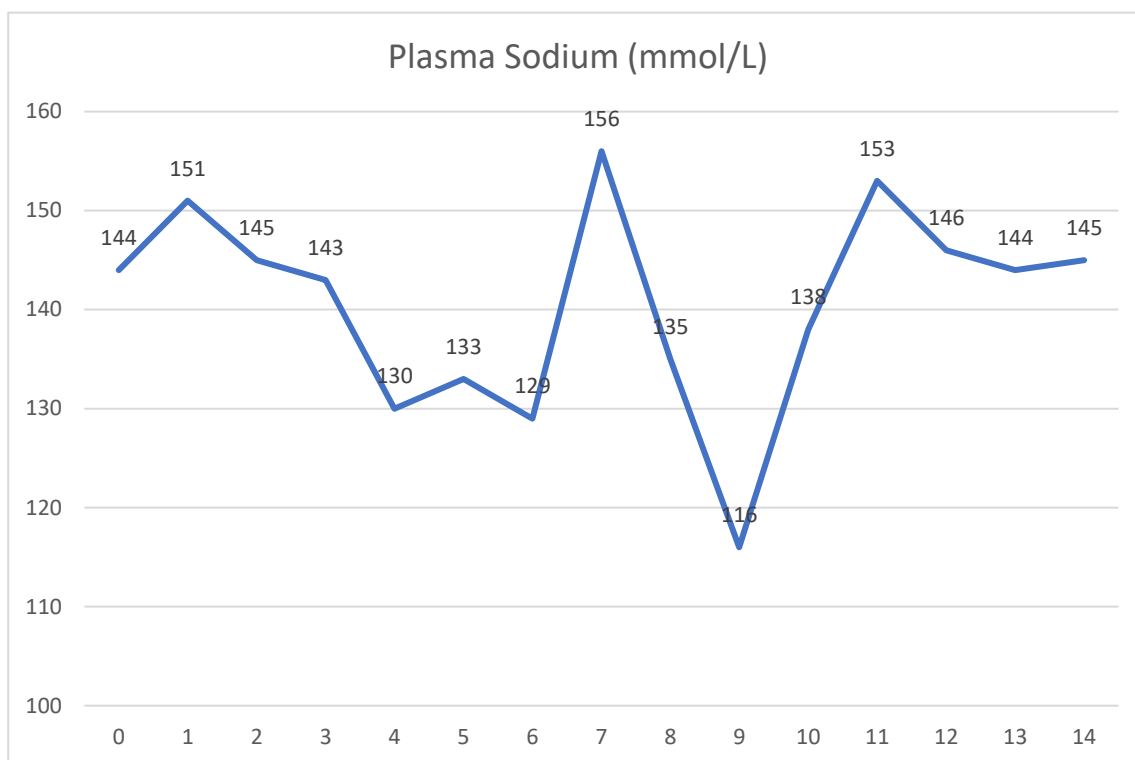
Diabetes insipidus is a combination of signs and symptoms generating a plentiful volume of urine and causing elevated serum osmolality. There are two types of diabetes insipidus: central diabetes insipidus and nephrogenic diabetes insipidus. Central neurogenic diabetes insipidus occurs when the production of the hormone Arginine Vasopressin (AVP) is low. In contrast, nephrogenic diabetes insipidus occurs when the kidneys cannot respond to high levels of the hormone AVP. Postsurgical central insipidus can be categorized into transient, permanent, and triphasic. Transient courses of diabetes insipidus following surgery represent most of the cases. Temporary diabetes insipidus is thought to be caused by temporary dysfunction of AVP-producing neurons as a result of direct surgical trauma or indirect after-surgical edema. The incidence of diabetes insipidus in patients who underwent pituitary surgery is 5%, and 4.6% of these patients will have only transient diabetes insipidus, and only 0.4% became permanent. Transphenoidal surgery is considered a minimally invasive and effective procedure for pituitary adenomas. Diabetes Insipidus after this surgery is not an uncommon complication, even though the reported rate of postsurgical central diabetes insipidus varies widely from 1 to 67%. Postoperative temporary diabetes insipidus gradually resolves up to 6 months.

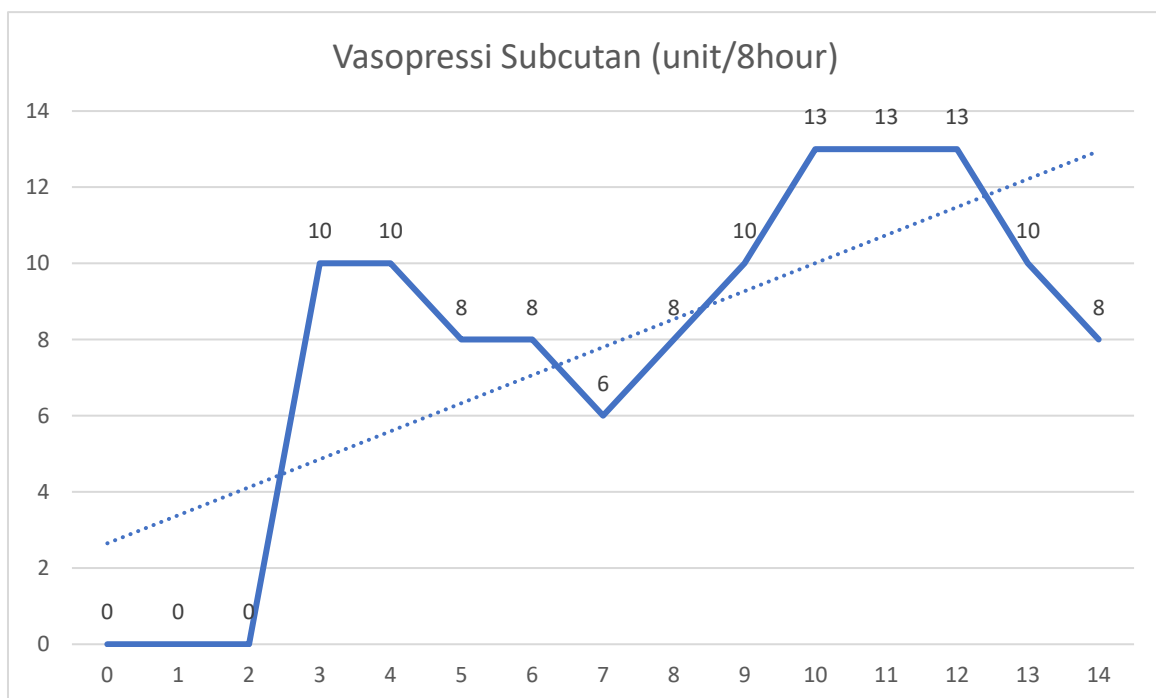
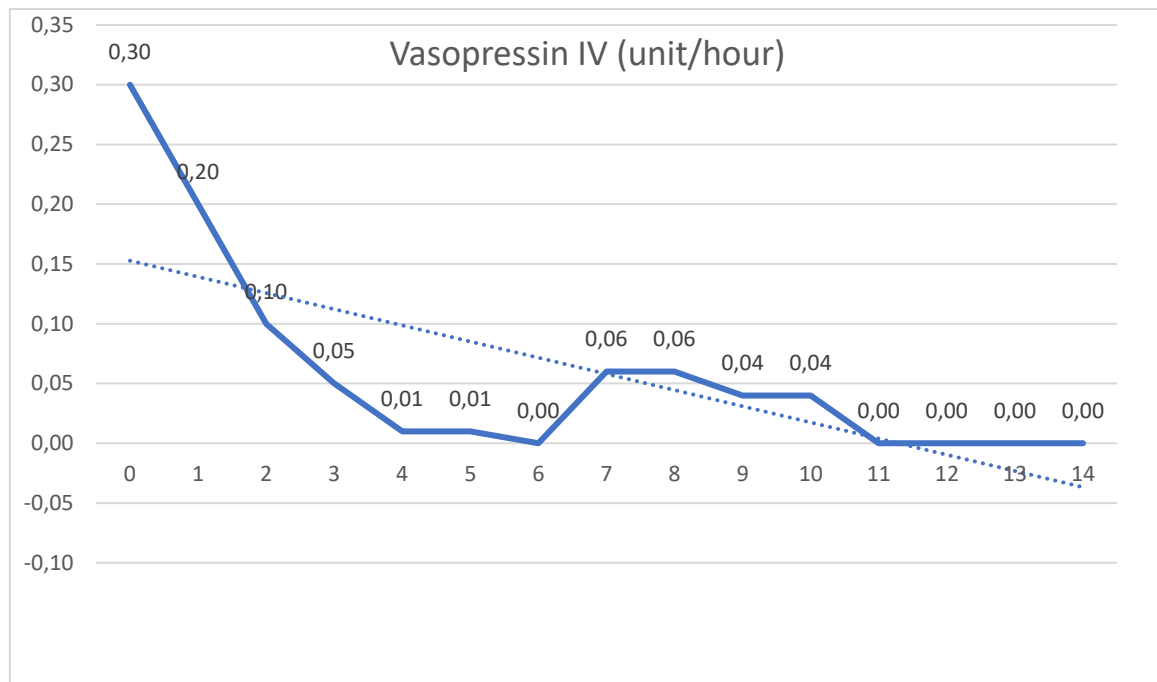
Case Presentation

A 36-year-old female patient presented chief complaints of headache and blurred vision, which gradually worsened one year ago. After undergoing several examinations, the patient was diagnosed with pituitary adenoma. The patient underwent a sublabial transsphenoidal

hypophysectomy. The duration was three long hours and uneventful.

On Day 0, the patient arrived at the intensive care unit (ICU) intubated, hemodynamically stable, and sedated. The patient is then monitored and weaned; a brain protection strategy and strict fluid balance urine collection and pain management are applied. On day 1, the patient was then extubated. The patient was examined for several parameters, such as electrolytes, kidney function, and blood glucose level. The patient began to significantly increase urine output (>5 milliliters/kilogram body weight/hour). Increased urine production is accompanied by a simultaneous decrease in urine-specific gravity (<1.005) and an increase in serum sodium level up to 151 mmol/liter. The patient was diagnosed with postsurgical diabetes insipidus. The patient started receiving intravenous vasopressin at a dose of 0.3 units/hour and titrated according to urine production until the target urine output was reached after the third day of care. After urine is reached, the dose of vasopressin slowly decreases, and the administration begins to transition to the subcutaneous route. On day 7 of treatment, when the intravenous vasopressin dose had been discontinued and the vasopressin dose at the 8u/h point, there was a significant urine production spike and an increase in the plasma sodium level to 156 mmol/liter. On the eighth day of treatment, the administration of vasopressin was again given intravenously and subcutaneously until a decrease in urine production towards the target was achieved. Finally, on days 11 to 14, vasopressin is administered only subcutaneously until the patient is discharged from the ICU. The patient was successfully discharged to the ward with a tapering-off subtotal dose.





Discussion

The patient developed polyuria within the initial hours of treatment. Polyuria is a hallmark sign of diabetes insipidus. The clinician should be aware of other polyuria causes, such as

postoperative hypovolemia, hyperglycemia, and the use of diuresis drugs. This differential diagnosis must be excluded. In this case, the differential diagnosis was excluded through proper fluid balance calculations, monitoring

serum electrolytes and glucose levels, and ensuring the absence of diuretic use. Confirmation of the postoperative central diabetes insipidus is made based on findings of high urine output (5 ml/kg BW/hour), urine specific gravity (<1.005), response to vasopressin, average blood glucose level, and absence of diuretic use.

Diabetes Insipidus is the body's inability condition to concentrate urine due to defective production of the antidiuretic hormone (central diabetes insipidus) or nephrogenic diabetes insipidus (NDI), which corresponds to the insensitivity of the kidney to the antidiuretic effects of vasopressin. Diabetes insipidus (DI) is a syndrome characterized by polyuria (>30ml/kg/24H) of hypotonic urine, equivalent polydipsia, and hypernatremia. The patient shows elevated urine volume (108 cc/kg/24H) and blood sodium levels (144-151mmol/L). The primary therapy was the titrated vasopressin dose, in conjunction with electrolytes and fluid management. Vasopressin titration is based on patient clinical condition, urin output, fluid management, oral intake, and laboratory measures (natrium blood level and urin osmolarity).

Transient Diabetes Insipidus must be closely monitored after neurosurgical operations, especially in regions adjacent to the pituitary DI. Transient Management with good monitoring is the key. The risk of morbidity comes from the risk of untreated dehydration, electrolyte imbalance

Intravenous vasopressin provides a rapid effect with lower doses. At the same time, subcutaneous administration requires caution in critically ill patients because absorption is

slow, resulting in a slow effect and the need for higher doses. The conversion of the administration route needs to consider the patient's pharmacology, route, and hemodynamics.

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