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Supraphysiologic Vasopressin-Induced Receptor Desensitization: A Case Report

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Background: Arginine vasopressin (AVP) is a nonapeptide released from the neurohypophysis in response to increases in plasma osmolality, hypovolemia, hypotension, and angiotensin II. V2 receptors, expressed on the basolateral membrane of the renal collecting ducts coupled to the adenylyl cyclase-cyclic adenosine monophosphate (cAMP)-protein kinase A (PKA) pathway. PKA increases the synthesis and shuttling of aquaporin 2 water channel containing vesicles (AQMCV) from cytoplasmic vesicles to the luminal surface of the renal collecting ducts (and inhibits the endocytosis of the vesicles), where they are inserted into the apical cell membrane. As a result, AVP increases free water reabsorption from the filtrate and decreases serum osmolality. There are different protocols for CDI management in post neurosurgical patient, including IV or SQ injections as well as continuous vasopressin drip. Here we discussed a case where prolonged IV drip could probably have caused loss of response to IV DDAVP injections.

Case: Patient is a 46-year-old male admitted with AMS with imaging revealed ruptured anterior communicating artery aneurysm associated with subarachnoid and intra parenchymal hemorrhage. The patient underwent emergent aneurysm coiling and EVD placed. The patient was subsequently started on heparin infusion due to a large frontal lobe clot which was found on cerebral angiogram. The next day, the patient developed acute diabetes insipidus with hypernatremia and polyuria. Repeat head CT demonstrated increased cerebral edema around the frontal clot. The patient underwent right frontal craniotomy for clot evacuation. He patient was subsequently started on vasopressin infusion (ranging from 0.5-6 units/hour) by neurosurgery team for urinary output less than 200 mL/hour. Urinary output and sodium level improved and remained stable on infusion. When endocrinology attempted transition to DDAVP IV injections patient responded with severe polyuria to initial 0.5 mcg injection. Eventually patient required 1 mcg IV every 4 hours dosing to maintain normal urine output and required a slow wean off to the standard dose of 0.5 mcg every 8 hours over 14 days.

Discussion: we are presenting a case of CDI associated to SAH who was started on IV infusion of vasopressin, required slow weaning. phenomenon called for slow re-sensitization of the receptors with gradual decreasing of DDAVP dosing. Animal studies have shown that V1 and V2 receptors levels are sensitive to hormone-induced downregulation of hormone receptors. Hypervasopressinemia in rats whose blood vasopressin concentration was experimentally elevated by IV infusion to extraphysiologic levels caused significant desensitization of kidney vasopressin receptors. In another study, IM injection of DDAVP to rats led to a total loss of kidney V receptors. Thus, it is likely that administration of high dose DDAVP IV in this patient induced downregulation of kidney V2 receptors.

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