**BRIEF CLINICAL UPDATE**

# Co-management of the Neurosurgical Patient for the Hospitalist

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Co-management of surgical patients by hospitalists has become increasingly common since Zuckerman et al[1] first described the effects of an interdisciplinary team approach to the hip fracture patient in 1992. Auerbach et al[2] were the first to report co-management specifically in neurosurgical patients at the University of California San Francisco Medical Center in 2011. A consultative service on neurosurgical patients managed by a small cohort of hospitalists has existed at Ronald Reagan UCLA Medical Center since 2011. We review management of hyperglycemia and hyponatremia in the neurosurgical patient.

### Glycemic Control

Hyperglycemia is common in the neurosurgical population due to steroid treatment, stress hyperglycemia, concomitant type 2 diabetes mellitus as well as the emergence of new onset diabetes. History and physical should concentrate on diabetic complications as well as current glycemic control. A foot examination as well as evaluation of cardiovascular risk factors is important. Hemoglobin A1C should be measured if not obtained in the past three months. Dexamethasone is commonly used to decrease vasogenic edema in patients with intracranial malignancy and may increase blood glucose values. Thus, routine blood glucose monitoring in all patients treated with dexamethasone is important. In rare cases, steroid treatment caused diabetic ketoacidosis.[3] Attention to hyperglycemia is crucial as it causes arterial remodeling and neuronal damage. The mechanism of injury involves hyperglycemia-induced acidosis that enlarges the infarct area.

Hyperglycemia is associated with increased perioperative morbidity and mortality. McGirt et al[4] evaluated surgical morbidity in patients undergoing stereotactic brain biopsy. Diabetes was an independent risk factor for morbidity (OR 3.73) when preoperative glucose exceeded 200 mg/dL. Other studies examined if single hyperglycemic episodes or sustained perioperative hyperglycemia adversely affect neurosurgical outcomes. McGirt et al[5] evaluated hyperglycemia in 97 aneurysmal SAH patients, finding that a serum glucose >200 mg/dL (for two or more days) was associated with a poor outcome at 2 weeks and 10 months. They did not find correlation of individual hyperglycemic episodes with poor neurologic outcome. Another study of 182 craniotomy patients with low-grade glioma found decreased long-term survival in patients with persistent poor glycemic control (defined by post-operative glucose >180 mg/dL 3 or more times).[6] Persistent hyperglycemia was associated with worse five-year survival.

Several studies investigate the association between poor glycemic control and poor postoperative wound healing or surgical site infections. In a review of 918 craniotomy and spine cases, Davis et al[7] found that pre-operative blood glucose levels over 120 mg/dL were associated with an increased length of stay (LOS). Postoperative complications occurred more commonly in the hyperglycemic group.[7] A retrospective chart review of 197,461 lumbar fusion cases also found higher postoperative infection rates in diabetics.[8] Another retrospective review of 2,485 tumor resection craniotomy patients found higher postoperative glucose levels in patients who developed surgical site infections, but the difference was not statistically significant. The duration of surgery was significantly associated with postoperative surgical site infections (p=.047).[9]

In a retrospective evaluation of 290 neurosurgical patients, Guo et al[10] evaluated the relationship between steroid-induced hyperglycemia and hospital LOS. There was no association of increased plasma glucose with LOS. They found steroid-induced hyperglycemia was uncommon, present in only 10 patients. However, the study only included 23 patients with diabetes. Variability in blood glucose monitoring and steroid tapering regiments make the results difficult to generalize.[10] In aggregate, these studies suggest but do not prove a causal relationship between hyperglycemia and poor neurologic outcomes. More studies need to be conducted to fully elucidate this question.

The American Diabetic Association (ADA) recommends treatment to a goal blood glucose <180 mg/dL in critically ill patients. It is important to avoid hypoglycemia that is associated with worse neurologic outcomes. Hyperglycemia in the neurosurgical patient is associated with increased morbidity and mortality. Intraoperative and ICU glycemic control may consist of an insulin drip in patients with blood sugars above the target range. An insulin drip may also be appropriate for initial management of patients with labile or poorly controlled type 1 diabetes or type 2 patients on high-dose steroids. Ongoing titration of the insulin regimen may be required for patients on a steroid taper. For patients who are eating or receiving enteral nutrition, a basal-bolus regimen can provide good glycemic control. Additional sliding scale insulin can be added to the existing basal-bolus regimen. Oral hypoglycemic medication should be held until discharge.

*Hyponatremia*

Hyponatremia is a key issue in the postoperative neurosurgical patient as appropriate salt balance allows for cerebral perfusion. Baroreceptors throughout the body and osmoreceptors in the hypothalamus control the release of ADH that lead to hyponatremia. Cerebral edema can occur with hyponatremia when neuronal size increases due to water exiting the intracellular area.[12] In extreme cases, these changes can result in cerebral ischemia and CVA.

History in the patient with hyponatremia should concentrate on intravascular volume, past history of malignancy, end-stage liver disease, heart failure, CNS disease, and medications that cause hyponatremia. Physical examination should concentrate on an assessment of volume status, peripheral edema, and ascites. A broad differential diagnosis of hyponatremia pertinent to the neurosurgical patient includes pneumonia, medication-induced, the syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting (CSW). Hyponatremia is common in neurosurgical patients due to traumatic brain injury, intracranial infections, tumor, and hemorrhage. Aspiration pneumonia and hospital-acquired pneumonia can occur in the neurosurgical patient, commonly leading to hyponatremia. Hyponatremia occurs in up to 29% of patients with pneumonia and is more common in a hospitalized population.[13] Medications that can contribute to hyponatremia include thiazide diuretics, selective serotonin reuptake inhibitors (SSRIs), non-steroidal anti-inflammatory drugs (NSAIDs), opiates, and proton pump inhibitors (PPIs).[11] Post-operative hyponatremia is common due to volume depletion, administration of hypotonic fluid, pain, and nausea.

Initial evaluation of the hyponatremic patient should include assessment of volume status and plasma osmolality. Patients with normal or high serum osmolality may have hyperglycemia, renal failure, and pseudohyponatremia. Labs should assess adrenal and thyroid function. Adrenal insufficiency is a rare complication that can occur following Trans-Nasal Trans-Sphenoidal (TNTS) resection of a pituitary adenoma. The etiology of hypo and hypervolemic hyponatremia includes CSW, cirrhosis, and heart failure while euvolemic etiologies includes SIADH and primary polydipsia.[11] SIADH is common with intracranial mass lesions, while CSW is common after subarachnoid hemorrhage.[14] Symptoms of hyponatremia can range from nausea and weakness to generalized seizures and coma.

Distinguishing between the syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting (CSW) is crucial as treatment differs markedly. Volume status differentiates CSW from SIADH as CSW is a volume-contracted state.[15] Incorrect diagnosis and treatment of CSW as SIADH can lead to worsening hyponatremia and hypovolemia. The pathophysiology of cerebral salt wasting is still being investigated. CSW involves loss of salt and water resulting in a volume-depleted state. Differentiation of these clinical entities can be challenging but includes an assessment of central venous pressure and volume status. Central venous pressure is a validated method to assist in the diagnosis of

CSW.[16] Extracellular volume assessment should include review of daily weights and orthostatics. Helpful laboratory studies include serum and urine osmolality, urine sodium, hematocrit, bicarbonate, and fractional excretion of urate. CSW patients may have orthostatic changes, low CVP, and tachycardia. Lab findings in a patient with CSW include increased hematocrit, bicarbonate, and bun-to-creatinine ratio.

Treatment of CSW includes fluid replacement, oral salt tabs, and fludrocortisone. CSW typically resolves in a self-limited fashion 3-4 weeks after the initial insult.[15] Isotonic fluids (NS) may be employed to treat CSW. Several recent studies have used hypertonic saline to treat CSW in the neurosurgical population. Sodium levels should be followed every six hours with a goal correction of .5mEq/L/h.[14] Fludrocortisone acts on the renal tubule to increase sodium recovery. Fludrocortisone is typically initiated at a dose of .2 mg po bid.[17] Adverse effects of fludrocortisone include hypokalemia and hypertension. Hypokalemia can be profound and potassium levels should be closely followed.

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