

Objectives

At the conclusion of this lecture, you will be able to:

- Discuss the pathophysiology of fluid and ICF Excess (cellular edema)
- Discuss common, and some less common, causes of ICFVE (cellular edema) disturbances
- Discuss the most common clinical presentations of ICFVE (cellular edema)
- Discuss the rationale for certain treatments of ICFVE such as cerebral intracellular edema

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Pathophysiology of ICFVE (intracellular volume overload)

ICFVE

- Results from the fluid in the blood vessels being hypotonic and hypoosmolar
- If blood is hypotonic in relation to isotonic cells, then water moves into the cells by osmosis

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Pathophysiology of ICFVE (intracellular volume overload) ICFVE "Water intoxication" Total body water is excessive; but total body sodium level is normal ECFVE Water is not moving into the cells. Differs in that both sodium and water are moving into tissue space (third space) Process of peripheral edema

Pathophysiology of ICFVE (intracellular volume overload) • Water intoxication • Expanded intracellular water volumes results in functional impairment of cells • The cells most sensitive to excessive intracellular water volume: cerebral cells

Etiology of ICFVE (intracellular edema) • Excessive water intake (po or IV) • Solute deficit • Excess ADH production • Renal impairment

Etiology of ICFVE (intracellular edema) Excessive water intake (po or IV) Poor solute ingestion (chronic) Psychogenic polydipsia Iatrogenic (in-hospital) D5W; D5W 1/2 NS Dextrose is rapidly metabolized Solute deficit

Etiology of ICFVE (intracellular edema)

Solute deficit

- Low protein diet
- Plain water enemas

Renal impairment

- Decreased water excretion
- (although, RF, like CHF, and cirrhosis, usually involves more intravascular and interstitial volume excess)

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Etiology of ICFVE (intracellular edema)

ADH excess

SIADH (Syndrome of Inappropriate ADH)

- Tumors
 - Brain, lung, pangreas, prostate
- Metabolic
 - Hypothyroidism
- Stress
 - Surgery, anesthesia, pain, head trauma, infection
- Medications
 - _ narcotics

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Pathophysiology of ADH Excess ADH excess (eventual intracellular edema) Occurs in response to stress (trauma, ...) ADH is also known as Vasopressin (Pitressin) Has both vasopressor and antidiuretic hormone (ADH) properties Increases water resorption at the distal renal tubular epithelium (ADH effect). Causes smooth muscle contraction in vascular bed (vasopressor or PVR effects)

Clinical Presentation of ICFVE (intracellular edema) Hx. [those of hypoNatremia] Early Headache N, V; weakness Moderate Behavioral: anxious, irritable, disoriented Late Seizures, coma

Clinical Presentation of ICFVE (intracellular edema) PE VS Elevated BP [Cushing's - like] Bradycardia Respirations: rapid GEN: delirium,... HEENT: fundi - papilledema and any other signs of primary underlying disorder (lung ca.,...)

Volume Overload Treatment of ICFVE (intracellular edema) Treatment of ICFVE (intracellular edema) similar to Tx of extracellular volume overload water removal (after ABCs/O2,Monitor,...) Tx of Mild (early stage)

Restrict water [including ice chips]

Tx of Moderate to severe (CNS signs of IICP)

- Osmotic diuretic Mannitol
- Hyperttonic(3%) saline [usu not prehospital]

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Summary

We have discussed ICFVE:

- The pathophysiology of IC fluid excess and intracellular edema
- How the Tx of this pathophysiology may be complicated by the primary, underlying disorder (eg lung cancer, trauma,...)
- Common, and some less common, causes
- clinical presentations
- Expected complications and rationale for treatments of ICFVE / intracellular edema

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