

Renal Support in Brain Injury

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Acute Kidney Injury and Renal Support

I have no actual or potential conflict of interest in relation to this program or presentation.

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Renal Support in Brain Injury

- Acute traumatic brain injury
- Multisystem organ failure with coma, cerebral edema
- ESRD patients with brain injury

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Pathophysiology to consider

- Fixed volume in cranium
- Impaired cerebral auto-regulation of blood flow
- Loss of integrity of blood brain barrier (BBB)
- Small volume changes result in large pressure changes
- High ICP associated with reduced cerebral blood flow

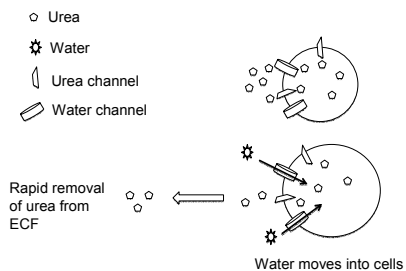
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Effect of RRT on ICP and CPP

- Urea and water pass through BBB via urea transporters and aquaporin channels
- The relative speed of water moving through aquaporin channels is faster than that of urea transporters
- Osmotic gradient can develop
- Outpatient IHD in ESRD patients is associated with elevations in ICP

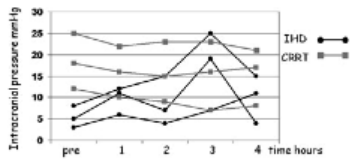
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Osmotic shifts with rapid removal of ECF solute



IHD vs. CRRT and ICP

- ICP elevation occurs much more frequently with IHD than with CRRT



Davenport. Sem. in Dialysis. 2009

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Dialysis Prescription in Brain Injury

- Use CRRT if available
- Use a low dose prescription to start
 - Avoid rapid correction of osmolality
- Anticoagulation
 - Citrate/No anticoagulation if risk of intracranial hemorrhage
- Pay close attention to sodium prescription

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Sodium Prescription

- Hypotonic solutions will worsen ICP
- Example:
 - Na 155
 - HCO₃ 15
 - Urea 30
 - Volume overloaded, oliguric
- Dialysis prescription?
 - Citrate anticoagulation preferred

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Sodium prescription

- Usual Na when patients on citrate anticoagulation
CRRT with 32mmol/L bicarbonate added: ≈ 135
- Target Na bath 155 to avoid osmotic shifts
- Baseline Na bath plus bicarbonate added
 - Look at CRRT bags to assist with calculation
 - 100 ml of 3% saline added to a 5 liter bag of CRRT solution will raise Na concentration in bag approx. 10 mmol/L (51mmol/5 L fluid)
 - Consider adding 200mL of 3% saline per 5 L bag to provide optimal Na and avoid alkalosis

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Mannitol Induced AKI

- Rare complication of aggressive mannitol therapy (most often used for ICP control)
- Risk factors
 - High dose > 1100 g/day in healthy, > 300 g/day if CKD
 - Chronic Kidney Disease
 - CHF
 - Hypovolemia

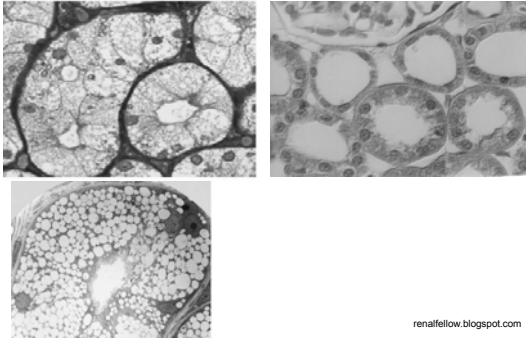
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Mannitol induced AKI - pathology

- Vacuolization and swelling of the proximal tubular cells
- Urinalysis may show vacuole containing cells
- Pathophysiology
 - Tubular swelling may lead to obstruction
 - Arteriolar vasoconstriction

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Osmotic nephrosis



Prevention

- Close monitoring of fluid balance
 - Watch for oliguria
 - Diuretics in combination with mannitol may predispose to AKI
- Monitor osmolar gap
 - Watch for accumulation of mannitol
 - Gap of > 20 mOsm/kg reason for caution
 - Gap of > 55 mOsm/kg associated with AKI
 - Gap may be more useful than absolute osmolality

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Treatment

- If recognized early
 - Hold Mannitol
 - Consider hypertonic saline as alternative for ICP management
 - Most patients recover with conservative management – good prognosis
- If anuric
 - Consider hemodialysis
 - Mannitol is rapidly removed and recovery often quite rapid

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Mannitol use in dialysis dependent patients

- Is it safe?
- Is it effective?
- Safety is not clear – consider if patient already has ESRD or refractory ICP
- Effective in ICP control in patients on CRRT (anecdotal)
- Rapidly cleared –based on osmolar gap

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RRT in Brain Injury - Summary

- Fairly good evidence for CRRT use over IHD
- Gentle prescription with close attention paid to Na balance (frequent monitoring)
- Mannitol induced AKI
 - It happens.
 - Be aware. Good prognosis. Reversible with dialysis.

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