Fluid and electrolyte disturbances in children

Block 11

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Introduction

- Children with disorders of Na^+ balance present with complaints referable to ECF :
- Volume expansion (volume overload / oedema) or
- Volume contraction (dehydration)
- Loss of >10 % of ECF volume $\rightarrow$ at risk of hypovolaemic shock

Physiology

- TBW (total body water) constitutes
  - 60 % in an adult
  - 70 % in an neonate
  - > 80 % in a premature
- TBW varies inversely with fat content
- TBW is distributed into 2 major compartments: Extra- and intracellular

Physiology

- In infants ECF consists of ± 1/3 of TBW
- ECF consist of
  - Plasma volume and
  - Interstitial fluid
- Na^+ is the predominant cation in the ECF
- Na^+ is the major component and major determining osmolyte in the serum

Physiology

- Na^+ cannot freely move across cell membranes
- Water can move freely across cell membranes

  $\rightarrow$ thus Na^+ determines water distribution in the body compartments
- S-Osmolarity = 2 x S-Na^+ + S-urea + S-glucose
- Normal S-Osmolarity = 270-280 mOsmol/L

Hyponatraemia

- Defined as S-Na^+ sodium <130 mmol/L
- Differentiate between 2 groups
  - ↑ Total body Na^+
  - ↓ Total body Na+
- Causes of ↓ S-Na^+
  - External loss of Na
  - Gain of excess water -fluid overload

Conditions associated with a deficit or an excess of total body Na^+

Condition	Deficit (hypovolemic)	Excess (hypervolemic)
Gastro-intestinal	Diarrhoea Vomiting (pyloric stenosis)	Cirrhosis
Renal	Medications (diuretics) Renal tubular diseases	Nephrotic syndrome Acute renal failure

Conditions associated with a deficit or an excess of total body sodium (cont)

Condition	Deficit (hypovolaemic)	Excess (hypervolaemic)
Cardiac		Congestive heart failure
Adrenal	Addison's disease Congenital adrenal hyperplasia	Conn's syndrome Cushing's syndrome Steroid treatment
Skin	Cystic fibrosis	
Iatrogenic	Diuretic therapy (hydrochorothiazide)	Hypotonic IV fluid

Volume contraction

Severe dehydration
Sunken eyes
Loss of skin turgor
Dry mucous membranes
Limp/ non-responsive

Volume overload
Nephrotic Syndrome

Generalised oedema

Assessment of Na balance and ECF volume

- History
  - Underlying disease
  - Food and fluid intake, vomiting
  - Diarrhoea, stool frequency, nasogastric tube drainage
  - Weight loss & urine output
- Physical findings
- Laboratory results
  - Urine osmol, U-Na^+,Cl, K^+, Creatinne, $FeNa^+$ %,
  - S-UKE, glucose and osmol

Clinical features of ↓ S-Na+ with hypervolaemia

- Periorbital oedema
- Swollen extremities
- Ascites
- Signs of hypoproteinaemia

- Depends on the underlying cause
- Associated with stimulation of renin-angiotensin system

Special investigations

- blood and urine biochemistry are necessary to diagnose hyponatraemia and related disturbances

Changes in body weight during treatment of a child with fluid and electrolyte provide practical guide - thus

Weigh the child on admission
Weigh again at frequent intervals
Monitor urine output

Treatment of Hypo-Na^+ or Hyper-Na^+

Focused on how to avoid the neurological complications which can potentially occur

- During the course of untreated dysnatraemias

or

- Which may develop after inappropriate correction of these disorders

Modern approach to rehydration therapy

- Keep things simple: aim for oral rehydration
- Assess circulation – if shocked –
- Resuscitate with isotonic fluid bolus: 0.9% NaCl IV
- Once shocked has been treated effectively
- Continue rehydration according to estimated dehydration: Mild/moderate/severe
- Oral rehydration (NG tube if necessary)

- Armon K, Stephenson T, MacFaul R, Eccleston P *et al*. An evidence and consensus-based guideline for acute diarrhoea management. Arch Dis Child 2001; 85: 132 - 142

Rate of fluid replacement

- Depends on
  - underlying cause
  - severity of dehydration
  - ongoing losses
- Acute isotonic dehydration
  - Rehydrate over 4-6 hrs
- Reassess frequently -
  - Weigh the child every 12-24 hours

For severe dehydration due to gastroenteritis

- Replacement fluid ½ Darrows in 5% Dextrose IV (½ D/D)*
- Give more if stool output is very high
- Start oral rehydration solution* once shock has been treated
- Review regularly

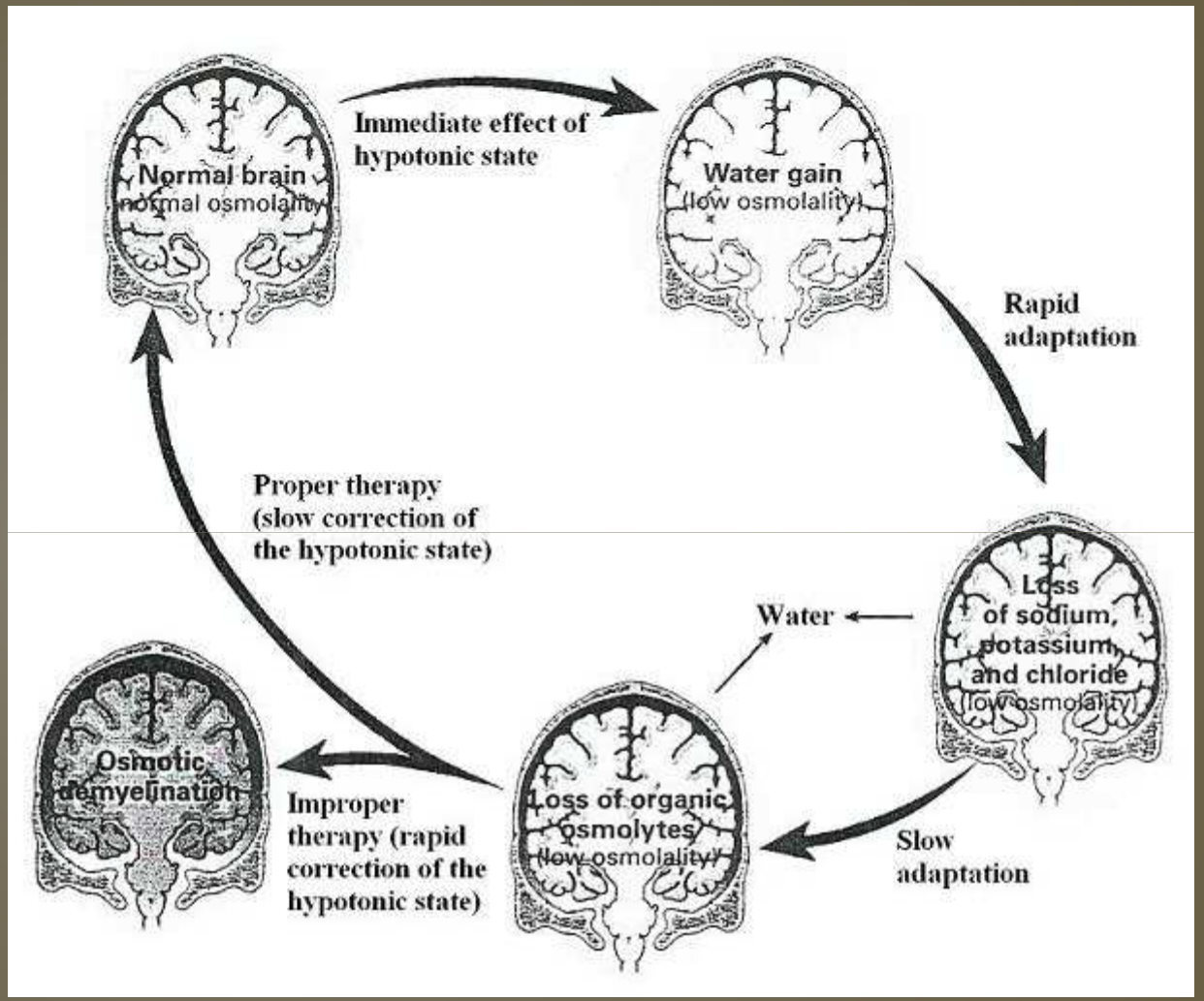
* (Study contents of ½ D/D and oral rehydration solution)

Treatment of hyponatraemic dehydration

- First treat shock
  - Restore circulating blood volume
  - Bolus isotonic fluid (0.9% NaCl) 20 ml/kg IV rapidly
  - Repeat once more if shock is still present
  - Then start IV rehydration ± Na^+ replacement
  - NOTE : Na^+ is replaced only if S-Na^+ <120mmol/L + patient is symptomatic

Na^+ repletion for symptomatic ↓S-Na

- Calculate Na^+ deficit:
  - 0.6 x weight x [desired S-Na^+ minus actual S-Na^+]
  - Desired S-Na^+ = 125 mmol/L - to avoid over correction
- Add calculated Na^+ deficit to replacement fluid
- Correct S-Na^+
  - At a rate to increase S-Na ≤ 1 mmol/L per hour
- Replace Na^+ deficit slowly over 48 hours to prevent neurologic complications
- Normal daily dietary Na^+ requirement 1-2 mmol/kg


Normal brain
normal osmolality
Immediate effect of
hypotonic state
Water gain
(low osmolality)
Rapid
adaptation
Loss
of sodium,
potassium,
and chloride
(low osmolality)
Water
Slow
adaptation
Loss of organic
osmolytes
(low osmolality)
Improper
therapy (rapid
correction of the
hypotonic state)
Osmotic
demyelination
Proper therapy
(slow correction of
the hypotonic state)

Management of ↓ S-Na^+ associated with hypervolaemia

- Depends on the underlying cause
- With ↓ GFR - ↓ S-Na^+ develops due to fluid overload
- Treatment
  - Restrict fluid and NaCl intake
  - Diuretics

Hypernatraemia

- = Serum sodium >150 mmol / L
- > 90 % caused by diarrhoeal losses of water
- Other causes
  - Too concentrated milk formula
  - Incorrect reconstitution of ORS
  - Salt added to infant foods
  - Child abuse (enforced thirst, lack of feeding)
  - Diabetes insipidus (central/nephrogenic causes)

Clinical features of ↑S-Na & hypovolaemia

- Dehydration is more subtle in fat babies
- Hypotension is a late phenomenon
- Skin feels doughy
- Intracellular dehydration→ CNS signs
- Irritability, lethargy, seizures and coma

Pathophysiology

- With gradual ↑ S-Na^+ brain cells produce idiogenic osmoles (adaption)
- → Compensatory ↑ intracellular osmolality and prevents cellular dehydration
- Aggressive treatment with hypotonic fluids may cause cerebral oedema
- → Coma, convulsions and death

Clinical features of hypernatraemic dehydration

- Neurological
  - Irritability, lethargy and thirst
- Dehydration
  - Sunken fontanel / eyes
  - Dry mucous membranes, ↓ turgor
  - Tachypnoea- to compensate for metabolic acidosis
- CVS
  - ↑ pulse and ↓ BP
  - ↓ central venous pressure
  - ↑ Capillary refill time

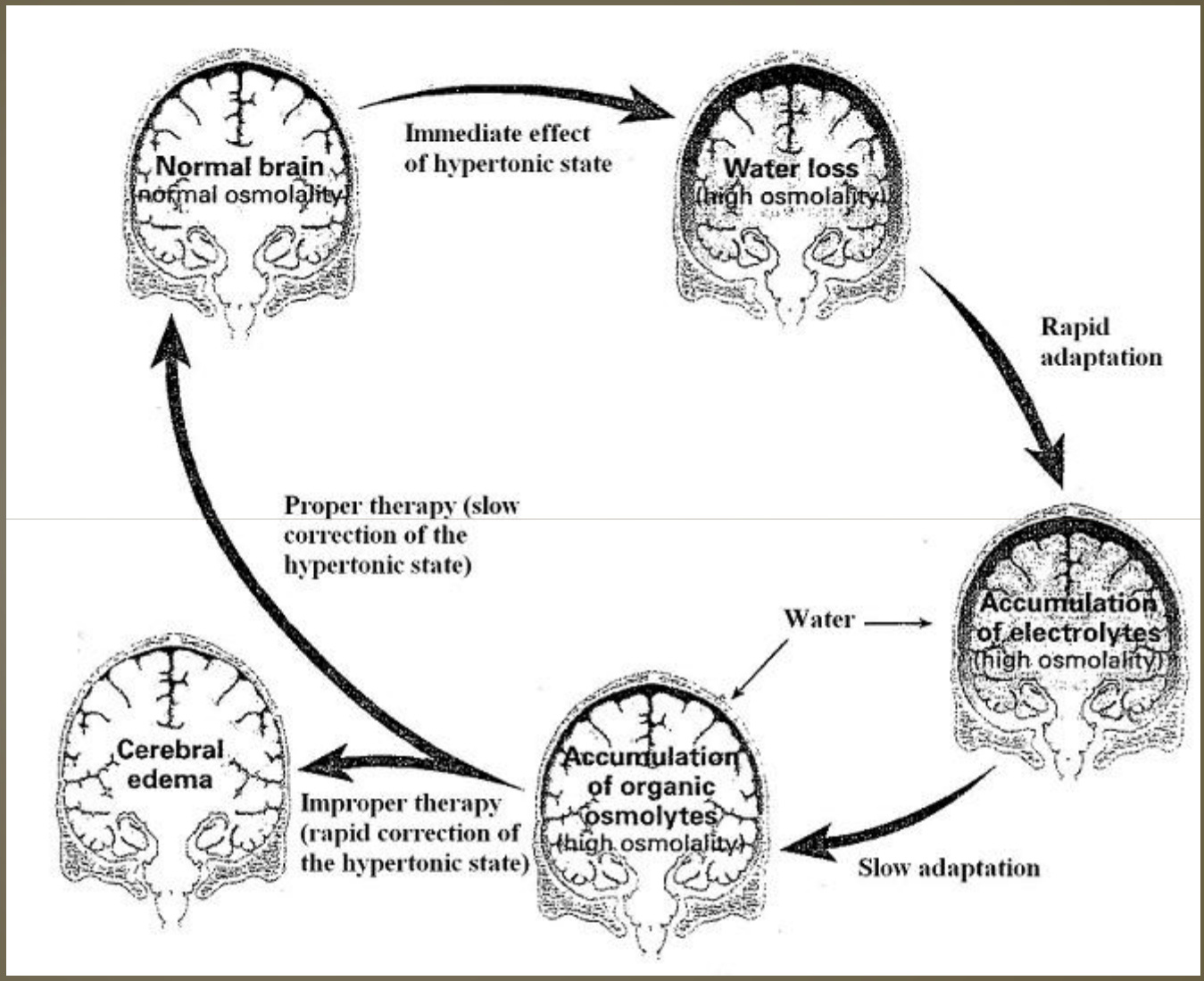
Management of Hypernatraemic Dehydration

First treat shock(if present) with IV isotonic fluid e.g. 0.9% NaCl (Na = 154 mmol/L)

- Bolus of 20ml / kg IV stat
- May repeat once if patient is still in shock
- After successful treatment of shock continue

For severe hypernatraemic dehydration

- Continue with
  - ½ Darrows in 5% Dextrose IV at slower rate – rehydrate over 24-48 hours
- Start oral rehydration slightly later – patient may be very thirsty and drink too much
- Review regularly
- Increase oral rehydration gradually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of organic
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(high osmolality)
Slow adaptation

Oedema in children

Two major groups, i.e.

- With proteinuria
  - E.g. nephrotic syndrome
- Without proteinuria
  - ↑ Vascular permeability e.g.
    - Premature babies, septicaemia
  - ↑ Hydrostatic pressure e.g.
    - Heart failure and hypertension
  - ↓ Oncotic pressure e.g.
    - Malnutrition & cirrhosis

Pathophysiology of oedema

Two mechanisms

- ↑ capillary leak of water from vascular compartment to interstitium
- ↑ retention of Na^+ and water by the kidney

Patients with oedema may have

- ↓ intravascular volume
- normal intravascular volume
- ↑ effective ECF volume

- Must differentiate between these conditions as this influence therapy

Use of colloid solutions

- Colloid solutions are not used
  - to treat oedema
  - for rehydration
  - to restore albumin levels
- Limited indications e.g.
  - Salt free albumin infusion in nephrotic syndrome only used for severe hypovolaemia associated with hypovolaemic shock and oliguria

Treatment of oedema associated with hypervolaemia

- Treat the underlying condition
- Oedema will settle spontaneously
- Inappropriate colloid infusions may cause acute circulatory overload and pulmonary oedema

Polyuria

- Clinical disorders of ↓ urinary concentration
  - ↓ ADH
    - Central diabetes insipidus (failure to synthesize or secrete ADH)
  - Normal or ↑ADH
    - Renal insensitivity to ADH =nephrogenic diabetes insipidus NDI

Clinical features of CDI

- Rare
- Patients have polydipsia > for cold water
- Severe polyuria and nocturia
- Symptoms of ↑ S-Na^+ will only develop when patient does not have access to water

Nephrogenic DI

- Congenital NDI
  - Rare inherited condition
  - Renal tubules insensitive to ADH
  - X-linked dominant
- Clinical features
  - dehydration, vomiting, fever, constipation
  - ↓ growth ± mental retardation
- Laboratory investigations
  - ↑ S-Na^+, ↓ U-osmol and dehydration

Acquired NDI

- More common than inherited NDI
- Urine may be relatively hypertonic
- Have moderate polyuria and polydipsia

Other causes of ↓ urinary concentration

- Drugs

  Aminoglycocides, furosemide, vancomycin
- Diuretic phase of acute tubular necrosis
- Renal tubular diseases
- Adrenal insufficiency
- Chronic kidney disease