Hypernatremia - case scenario 2

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Case history 1

- 8 months old baby boy presented with the history of low grade fever and fast breathing of one day duration

- Hospitalized in the ward as bronchiolitis as the saturation in room air was normal and X ray chest showed hyper inflation
- After 4 hours of admission, shifted to PICU in view of cool peripheries and poor sensorium

- On examination

HR: 150/min, RR: 54/min, BP: 60/40mmHg, SPO2: 96 % with 6lit O_2

No chest retractions

Effortless tachypnoea+

- Septic shock

Step 1

- Stabilization of ABC
  - Maintainable airway
  - Breathing – acidotic
  - Circulation – in shock
- Capillary Blood Glucose: 83mg/dL
- Ionized Calcium - 1.25mmol/L
- Serum sodium value 165 mEq/L

- Diabetes mellitus and pan hypopituitarism were ruled out

Step 2: Estimate the volume status

		Diagnosis
1. History of fluid loss	No obvious loss (No diarrhea, but still voiding urine)	Hypovolemia
2. Weight	Pre illness weight is not known (Birth weight 3kg, present weight 6kg. Length: 68cm)	
3. Urine frequency	Voiding urine despite poor intake	
4. Signs of dehydration	Depressed AF. Doughy feel of abdominal skin	
5. Urinary indices	Urine Na < 10 mEq/L FENA < 1.0	

Step 3: Management of hypovolemic hypernatremia

a) Fluid bolus

Isotonic NS 20 mL/kg over 20 minutes (2 boluses given)

b) Deficit calculation – simple strategy based

½ DNS, 900ml @ 38ml/hour [150% maintenance]

(Or)

Formula based (750ml) + insensible water loss (180ml) – 39ml/hour

Step 4: Look for specific diagnosis requiring any specific therapy

- Urine output 50ml/hour (>4ml/kg/hour) – polyuria
  (History of frequent urination and heavy wet nappy since birth)
- Polyuria was missed in this child!

- CRP-negative, urine and blood culture-sterile, CBG- 140mg/dL

Further lab values
- Serum osmolality: 315mOsm/kg
- Urine osmolality: 210mosm/kg

Step 4: Look for specific diagnosis requiring any specific therapy

- Serum AVP levels sent
- Clinical exam: Reassessed- stable vitals. No midline defects
- Diabetes Insipidus – ? Neurogenic/ Nephrogenic DI

- Trial of AVP therapy (2mcg IV) and urine osmolality was rechecked – 810mOsm/kg

- Diagnosis: **Central DI**

Step 5: Monitoring

- Intake/output monitoring
- Output replacement every 4^{th} hourly
- Ensure rate of fall of sodium is 0.5 mEq/hr or 10 mEq/day
- Monitor sodium 4^{th} to 6^{th} hourly and modify the fluids

Step 6: Watch for complications

- NS bolus in case of shock

- New onset seizure – presume as rapid fall of sodium and give 3% saline (take sample ca, sugar)

  (also keep in mind – DD- CNS bleed/thrombosis/hypocalcemia)
- Reducing urine output in a well child – sign of improvement
- Reducing urine output in a sick child – AKI/other organ dysfunction

Step 7: Transition

- Alert at 6 hours of PICU stay
- Started on oral fluids and breast feeds
- Urine output 2ml/kg/hr. S.Cr – 0.4mg/dL
- Sodium level was 145 mEq/L (48 hours of admission)
- Serial monitoring of serum sodium and switched to less intense management

- Nasal AVP was started by the endocrinologist

Case 2

- 15 months old baby boy presented with lethargy, refusal of feeds
- On examination - febrile, Wt: 7kg, Ht: 70cm
- HR- 160/min, Pulse: Central pulse normal. Peripheral pulses weak, CRT> 2 sec. Cold extremities
- RR- 48/min, effortless tachpnoea, SPO_2 – 92% in room air
- BP:60/40mmHg

- Continuous dribbling of urine, poor urinary stream and palpable urinary bladder
- Septic shock
- Posterior urethral valve

Steps

- Stabilization of ABC
  - Maintainable airway
  - Breathing – acidotic
  - Circulation – in shock
- Capillary Blood Glucose: 102mg/dL
- Ionized Calcium - 1.21mmol/L
- Serum sodium value 165 mEq/L. S.Creatinine 1.2mg/dL, BUN 30mg/dL
- Urine sodium 12mmol/L, FeNa <1

- Hypovolemic hypernatremia with AKI (on a CKD)

Steps

- Received fluid bolus NS 10ml/kg over 20 min and antibiotics (after taking urine and blood culture)
- Followed by 150% maintenance (44ml/hour)
- Urine output was 6ml/kg/hour (polyuria)

- USG – Bilateral enlarged kidneys with hydrourteronephrosis with bladder wall thickening – suggestive of PUV (later proven by MCU)

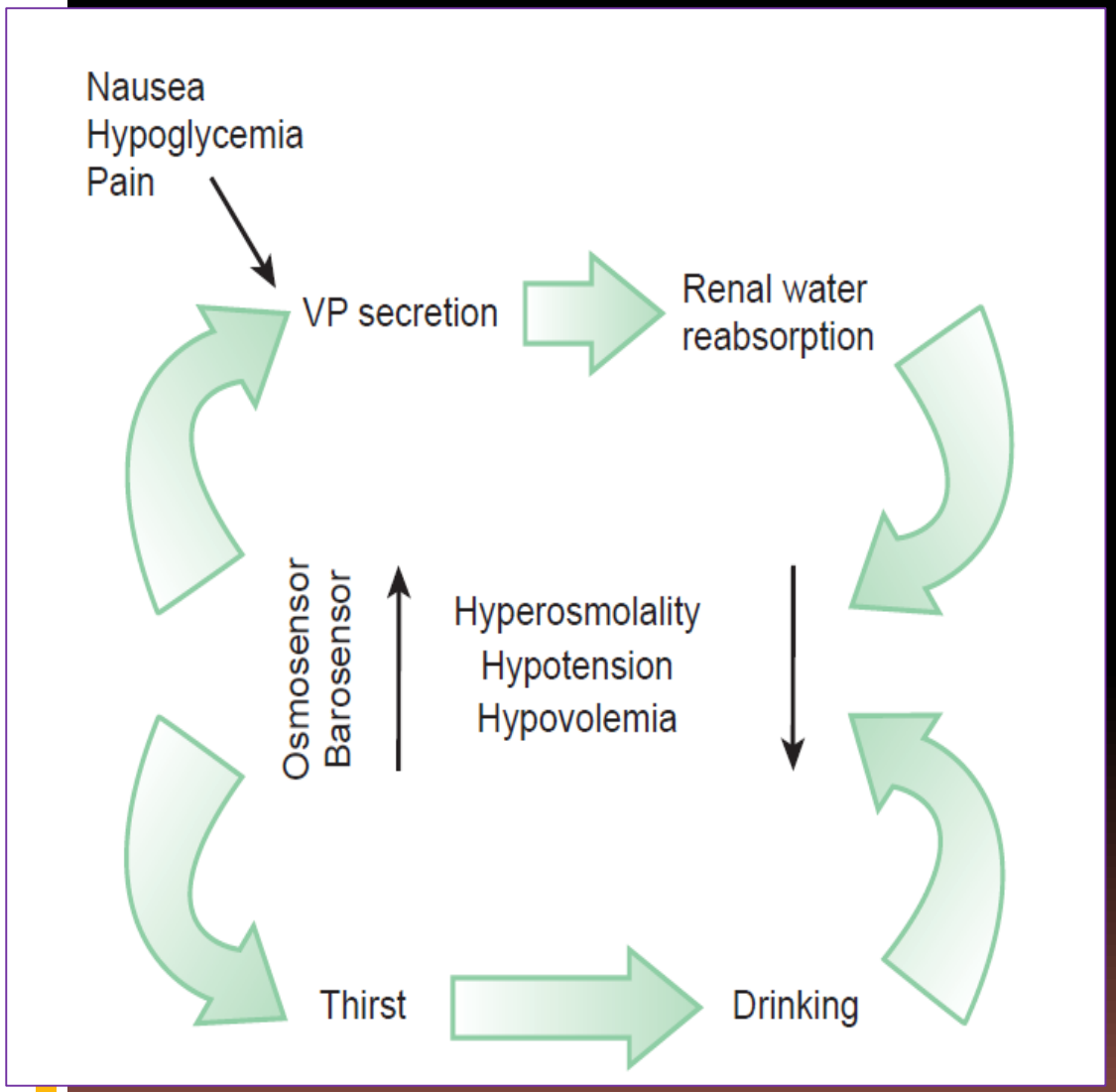
- Secondary cause of nephrogenic DI – tubular resistance to vasopressin

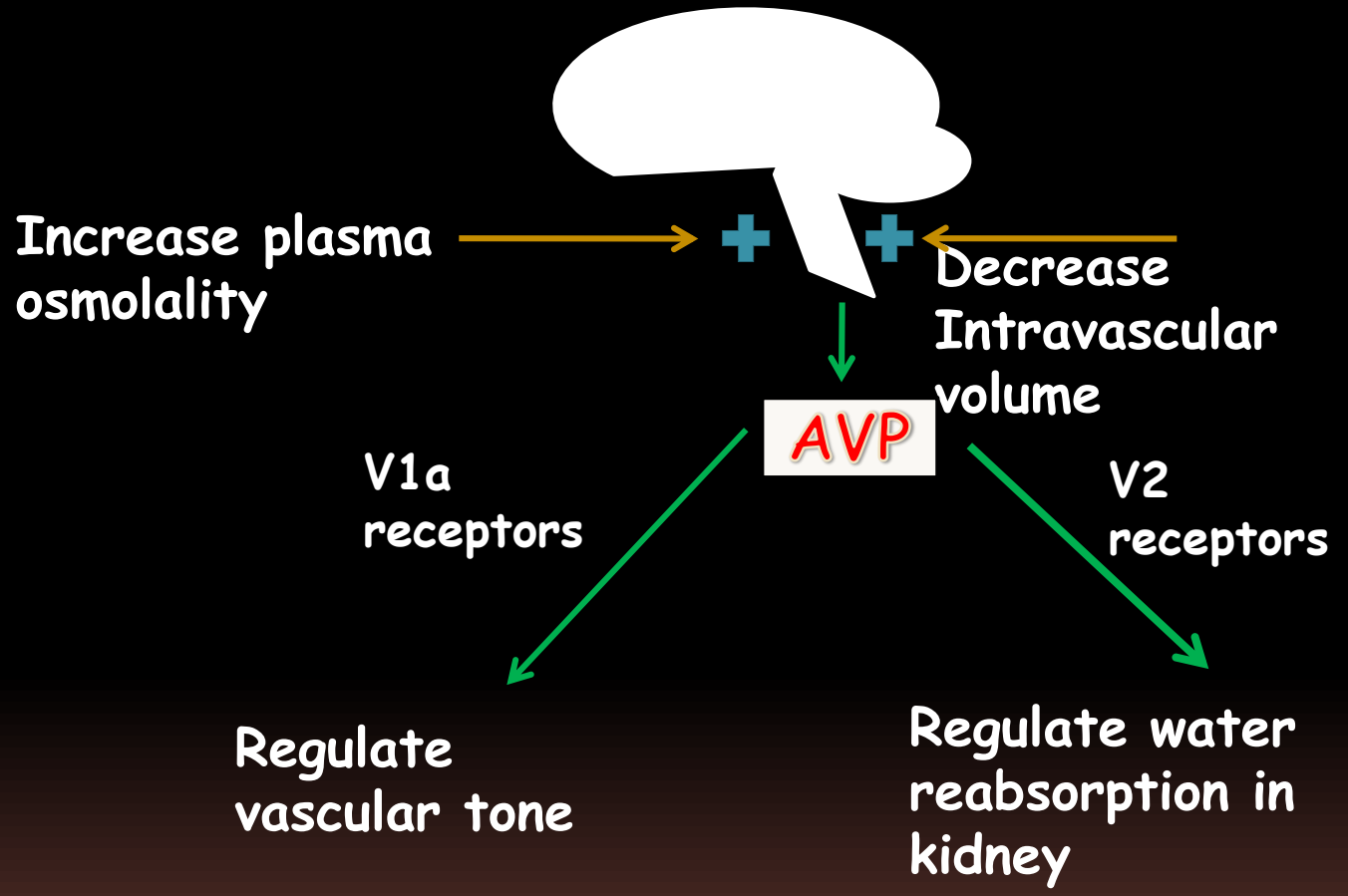
Diabetes Insipidus

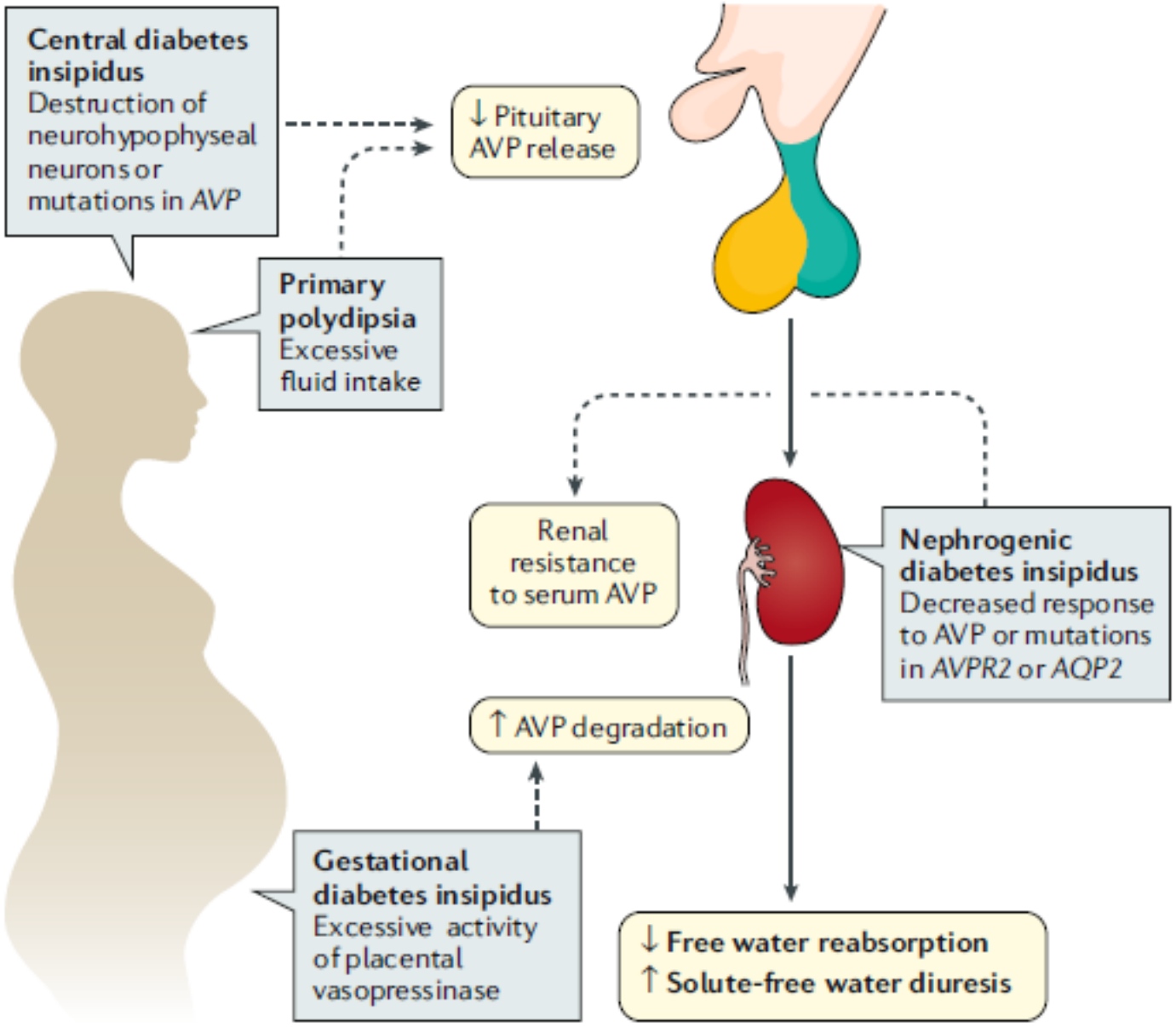
- It is a heterogeneous clinical syndrome of disturbance in water balance, characterized by

1. Polyuria (urine output > 4 ml/kg/hr and > 6 ml/kg/hr in neonates)
2. Polydipsia (water intake > 2 $L/m^2/d$)
3. Failure to thrive

Water balance - physiology







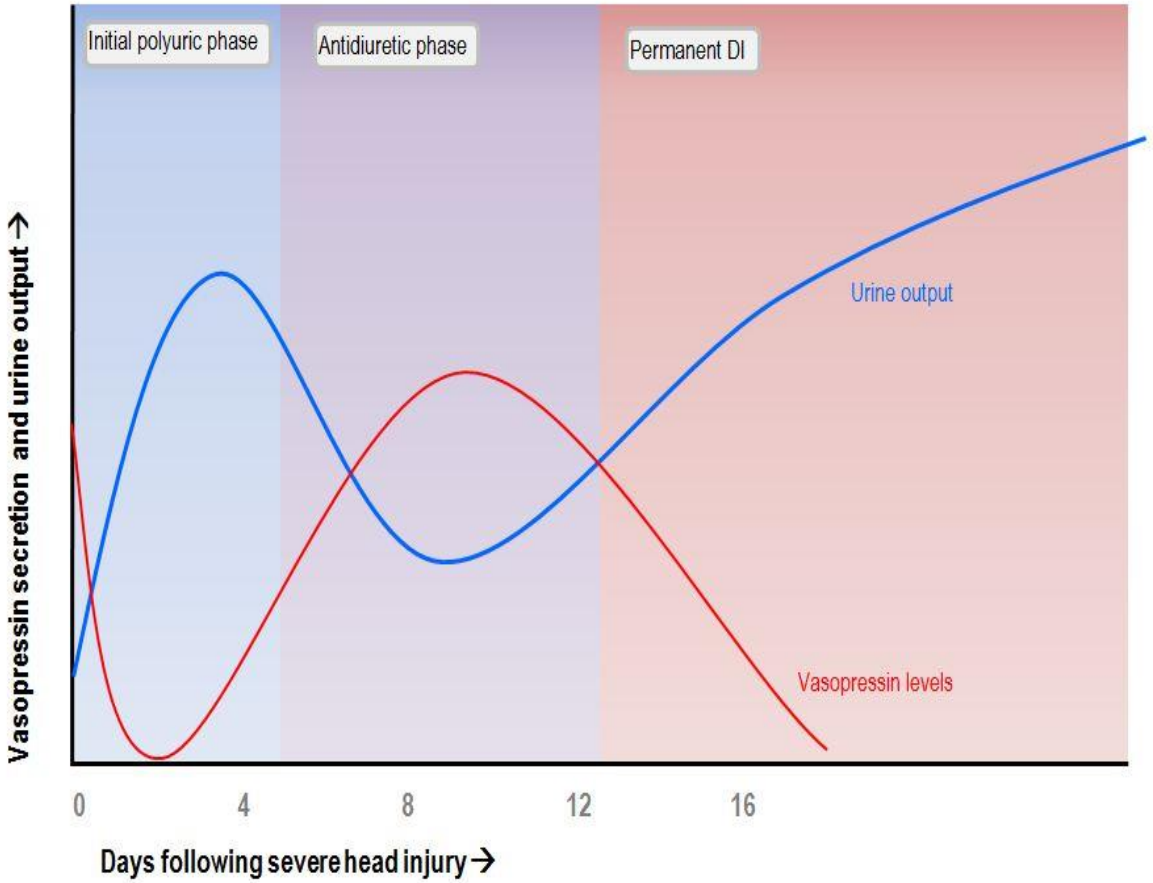
Central DI causes

- Idiopathic (10%)
- Familial CDI (Wolfram syndrome)
- Congenital hypopituitarism
- Congenital malformations (holoprosencephaly, septooptic dysplasia, encephalocele)
- Primary or secondary CNS tumours (craniopharyngioma, germinoma)
- Infiltrative diseases (LCH)
- Neuro surgical interventions and trauma

Mirjam CN. Diabetes insipidus. NATURE REVIEWS : 2019; 5:54

Post neurosurgery -Triphasic response

- Phase 1 - transient DI (lasting 12-48 hr) - local edema interfering with normal vasopressin secretion
- Phase 2 – SIADH (lasting up to 10 days) - unregulated vasopressin release from dying neurons
- Phase 3 - Permanent DI - > 90% of the neurons have been destroyed



Nephrogenic Diabetes Insipidus causes

Inherited

Familial X-linked recessive inheritance (vasopressin V2 receptor gene defect)

Autosomal recessive inheritance (aquaporin-2 gene defect)

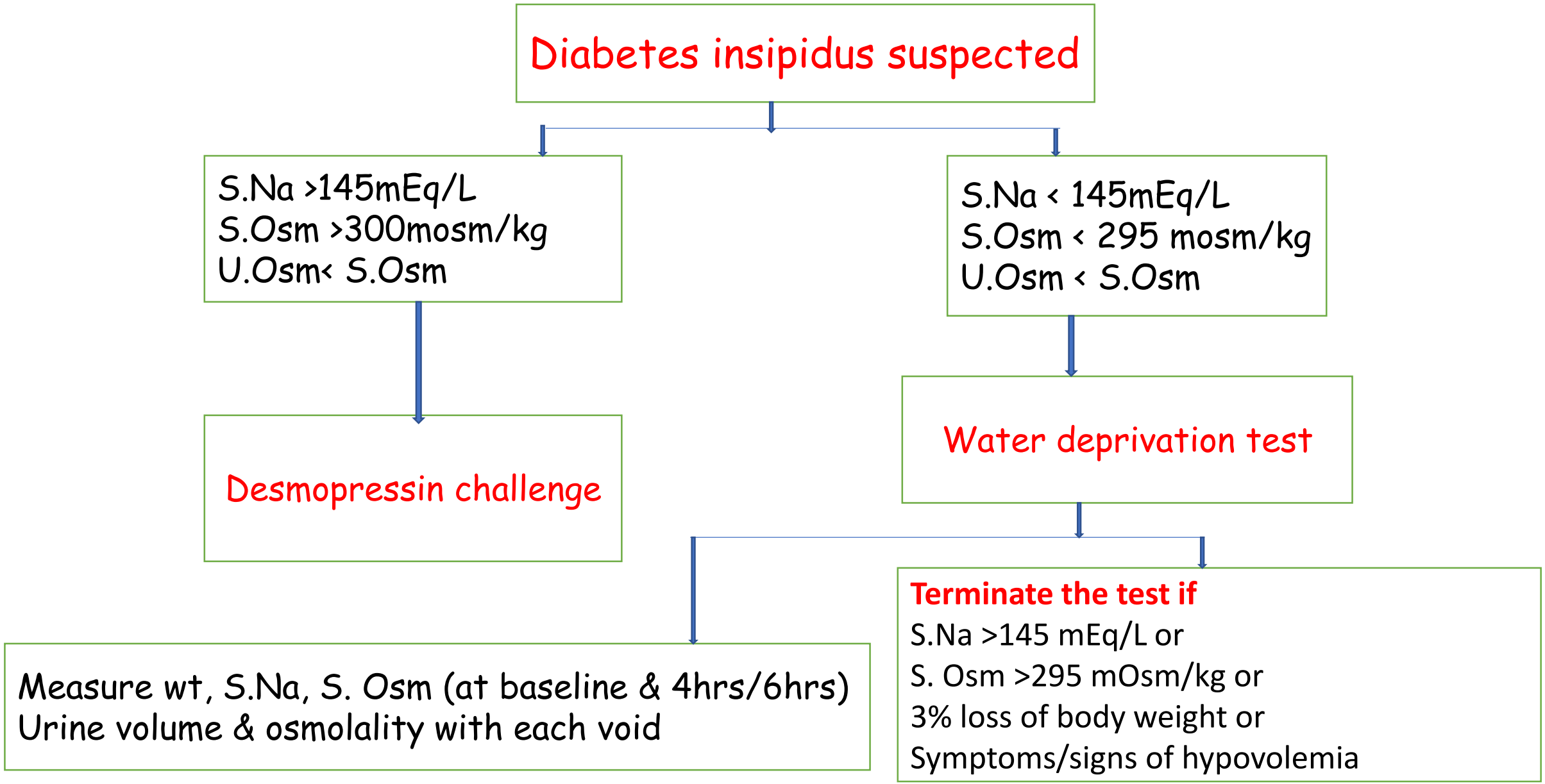
Acquired

Osmotic diuresis (diabetes mellitus)

Metabolic (hypercalcemia, hypokalemia)

Chronic renal disease

Drugs (lithium, amphotericin)


Diabetes insipidus suspected
S.Na >145mEq/L
S.Osm >300mosm/kg
U.Osm< S.Osm
S.Na < 145mEq/L
S.Osm < 295 mosm/kg
U.Osm < S.Osm
Desmopressin challenge
Water deprivation test
Measure wt, S.Na, S. Osm (at baseline & 4hrs/6hrs)
Urine volume & osmolality with each void
Terminate the test if
S.Na >145 mEq/L or
S. Osm >295 mOsm/kg or
3% loss of body weight or
Symptoms/signs of hypovolemia

Role of fluid deprivation and DDAVP tests in polyuria

Urine osmolality (mOsmol/kg)		Diagnosis
After fluid deprivation	After desmopressin	
<300	>750	Central DI
<300	<300	Nephrogenic DI
>750	-	Primary polydipsia
300–750	<750	? Partial central DI/? partial nephrogenic DI

Disease specific management

Aim of treatment for DI is to maintain access to free water and adequate fluid intake (3 $L/m^2/day$)

Central DI

- Continuous IV infusion: 0.0005 unit/kg/hr initially, then double dose q30min to reach desired effect; not to exceed 0.01 unit/kg/hr
- 2.5-10 units IM/SC/intranasally q8-12hr
- Titrate dose on basis of serum sodium, serum osmolality, fluid balance, and urine output

Nephrogenic DI

- Thiazide diuretics (2-3mg/kg/day)
- Indomethacin (2mg/kg/day)is added in patients who have poor response to diuretics.

In secondary NDI access to free water is usually sufficient

Take home points

- Measuring urine output in every child with convulsion/altered sensorium is mandatory to diagnose DI
- Early diagnosis and treatment of specific etiology improves the long term outcome
- Prevention of dehydration and hypernatremia with regular monitoring especially during intercurrent illness

THANK YOU