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Student scientific circle

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Poliuria - Polydipsia Syndrome

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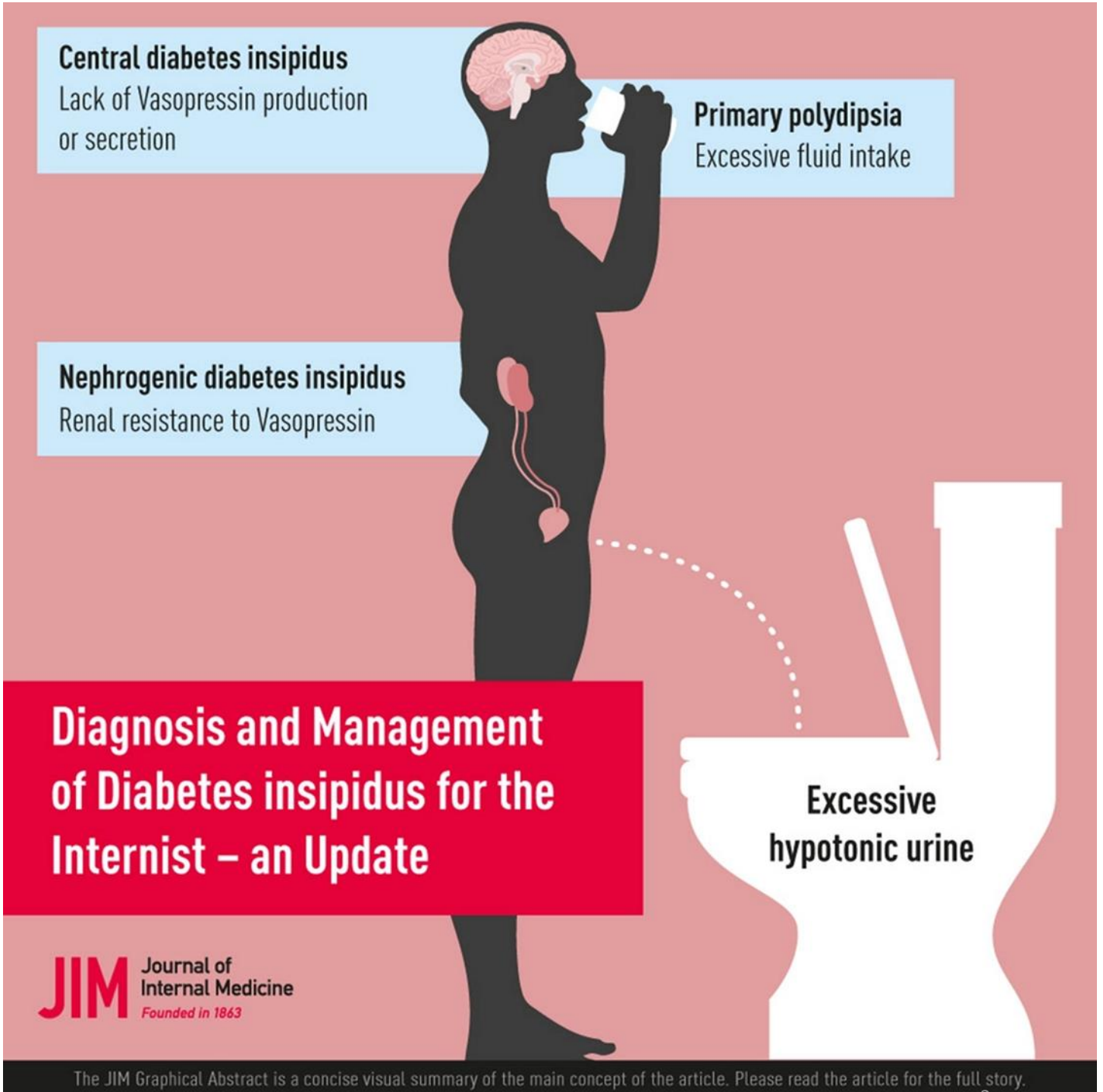
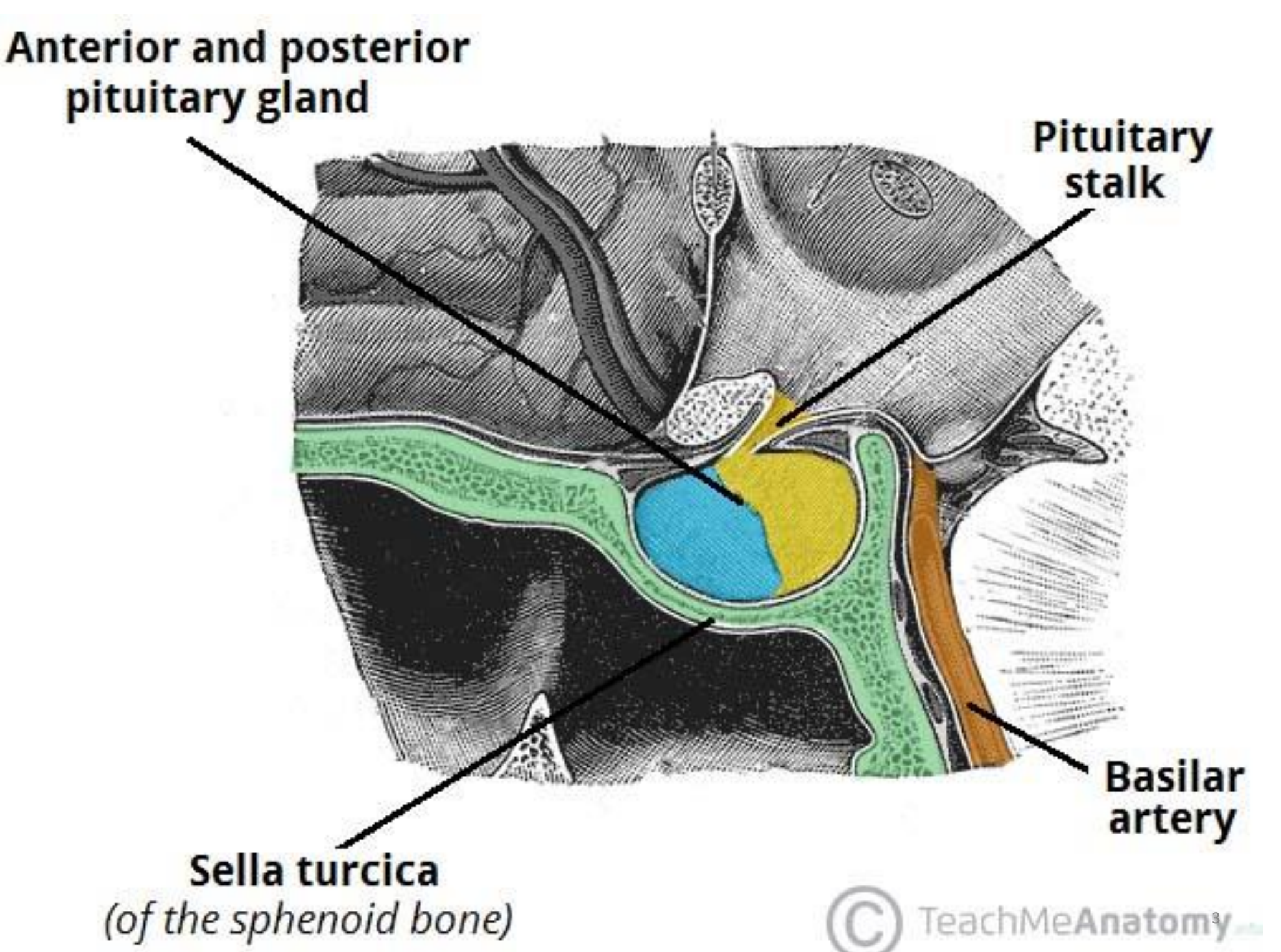
Poliuria - Polydipsia Syndrome




Anterior and posterior pituitary gland
Pituitary stalk
Basilar artery
Sella turcica
(of the sphenoid bone)
© TeachMeAnatomy

Central Diabetes Insipidus (CDI)

Destruction of AVP- producing neurons in the pituitary or AVP mutations

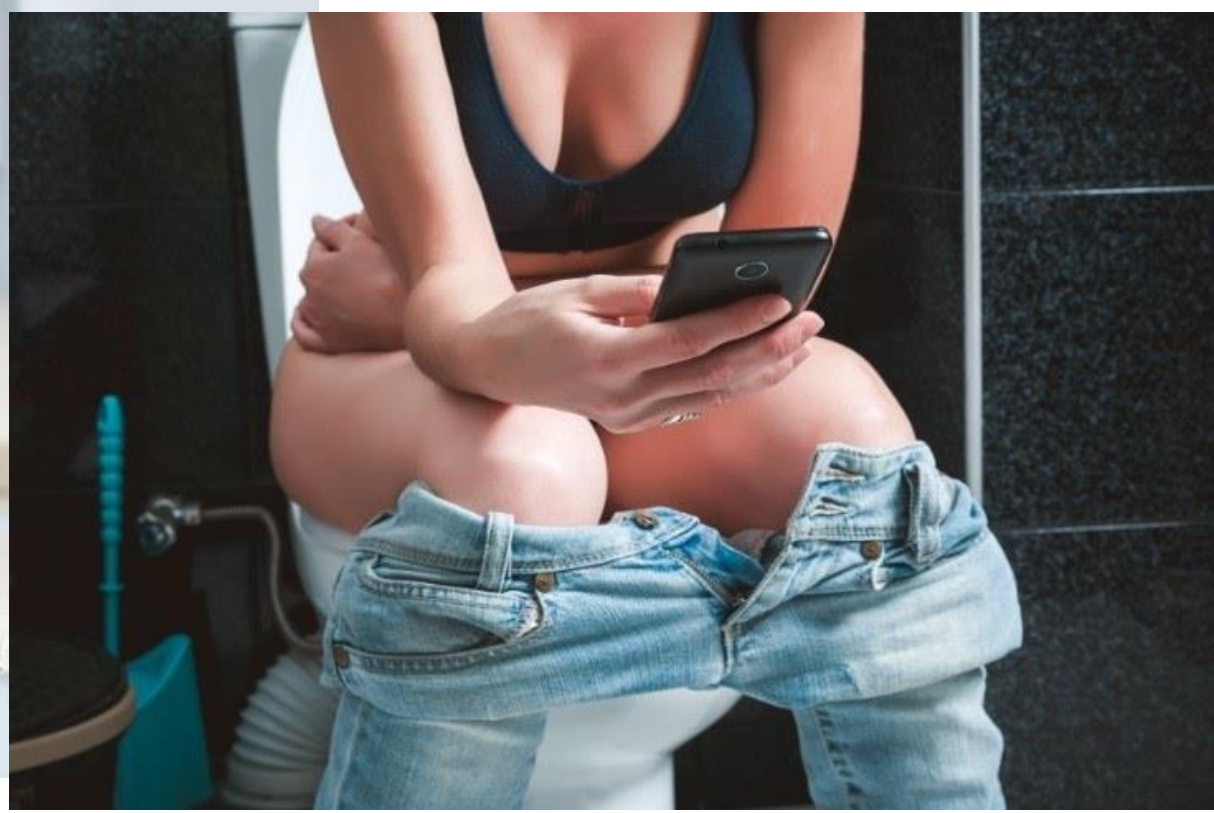
- Acquired
  - Postoprative or trauma
  - Primary brain tumors
  - Metastatic cancer
  - Infiltrative, eg neurosarcoidosis
  - Inflammatory / Autoimmune, eg hypophysites
  - Infections
  - Idiopathic
- Hereditary
  - Affected AVP gene

Nephrogenic Diabetes Insipidus (NDI)

- Acquired
  - Drugs
    - Lithium
    - Cisplatin
  - Electrolyte disorders
    (temporary downregulation
    of aquaporin 2 (AQP2) expression)
    - Hypokalaemia
    - Hypercalcaemia
  - Haematological
    - Multiple myeloma
    - Amyloidosis
    - Sickle cell disease
- Hereditary X-linked
  - AVPR2 or AQP2 genes

Gestational Diabetes Insipidus (GDI)

- Increased AVP degradation by placental vasopressinase



Primary polydipsia (PP)

Excessive fluid intake physiologically suppresses AVP secretion

- Psychogenic **($^1/_3$ cases)**
  - Schizophrenia
  - Schizoaffective or bipolar disorders
  - Anxiety

Primary polydipsia

$^1/_3$ cases
Health-conscious individuals
(**Dipsogenic diabetes insipidus),**

$^1/_3$ cases
Beer potomania

$^1/_3$ cases
alth-conscious individuals
psogenic diabetes insipidus),

$^1/_3$ cases
Beer potomania

Diagnosis

1. Excessive urination (> 50 ml/kg body weight/24h)
2. Drinking > 3 L/day
3. Sudden onset in 63% of patients with DI and 27% in patients with PP
4. Clinical symptoms are not specific enough to distinguish DI from PP.

Initial investigations

- Timing and onset of symptoms
- Triggering factors
- History of

- Head trauma
- Headaches
- Vision disturbances
- Psyciatric and dependency disorders
- Family history of the disease

Medical Examination

1. Asses potential dehydration:
   - Blood pressure;
   - Heart rate
2. A visual test including assessment of diplopia and visual field defects
3. Comprehensive skin and lymph nodes assessment (inflammatory or vascular disorders)

Blood tests

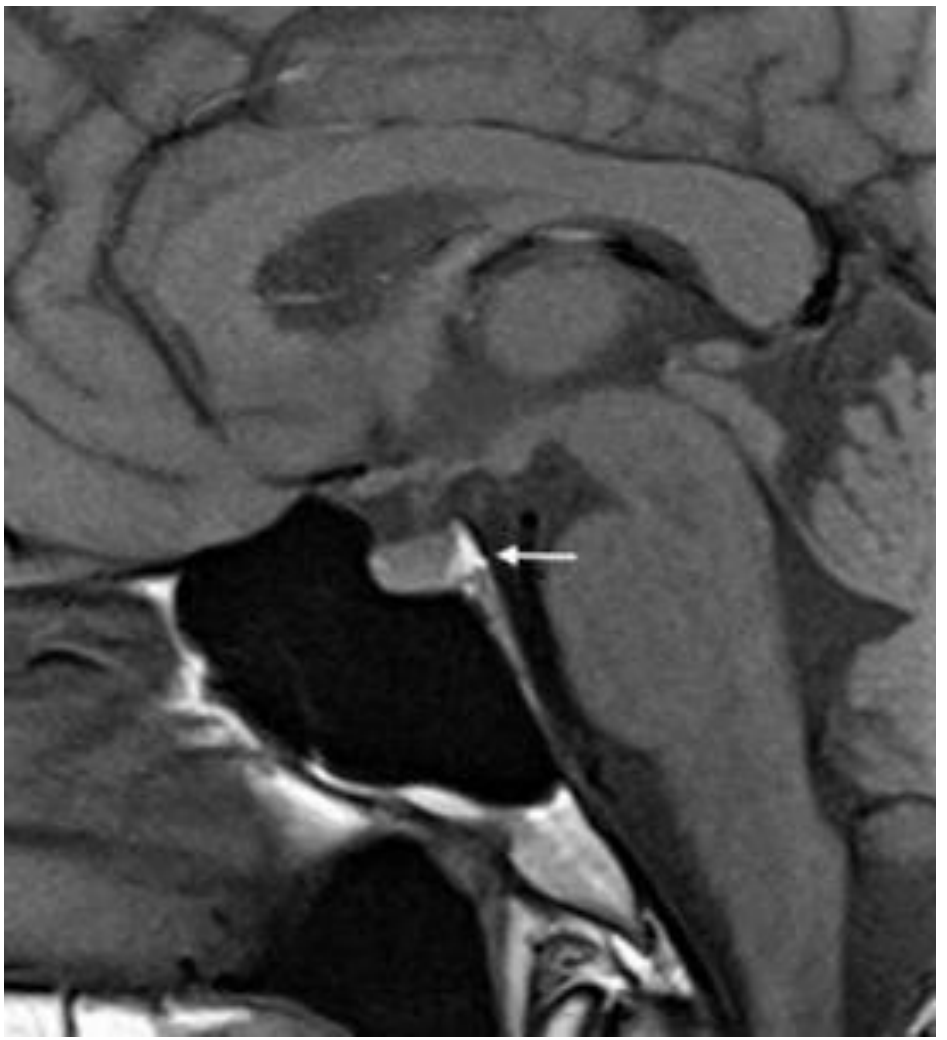
1. Blood tests for glucose, potassium and calcium concentrations
2. **Hyperuricaemia** due to chronic low uric acid clearance in central DI
3. Most polyuria-polydipsia patients will present with **Na and osmolarity levels** within the normal range

MRI investigation

1. **Absence of the ‘bright spot’**

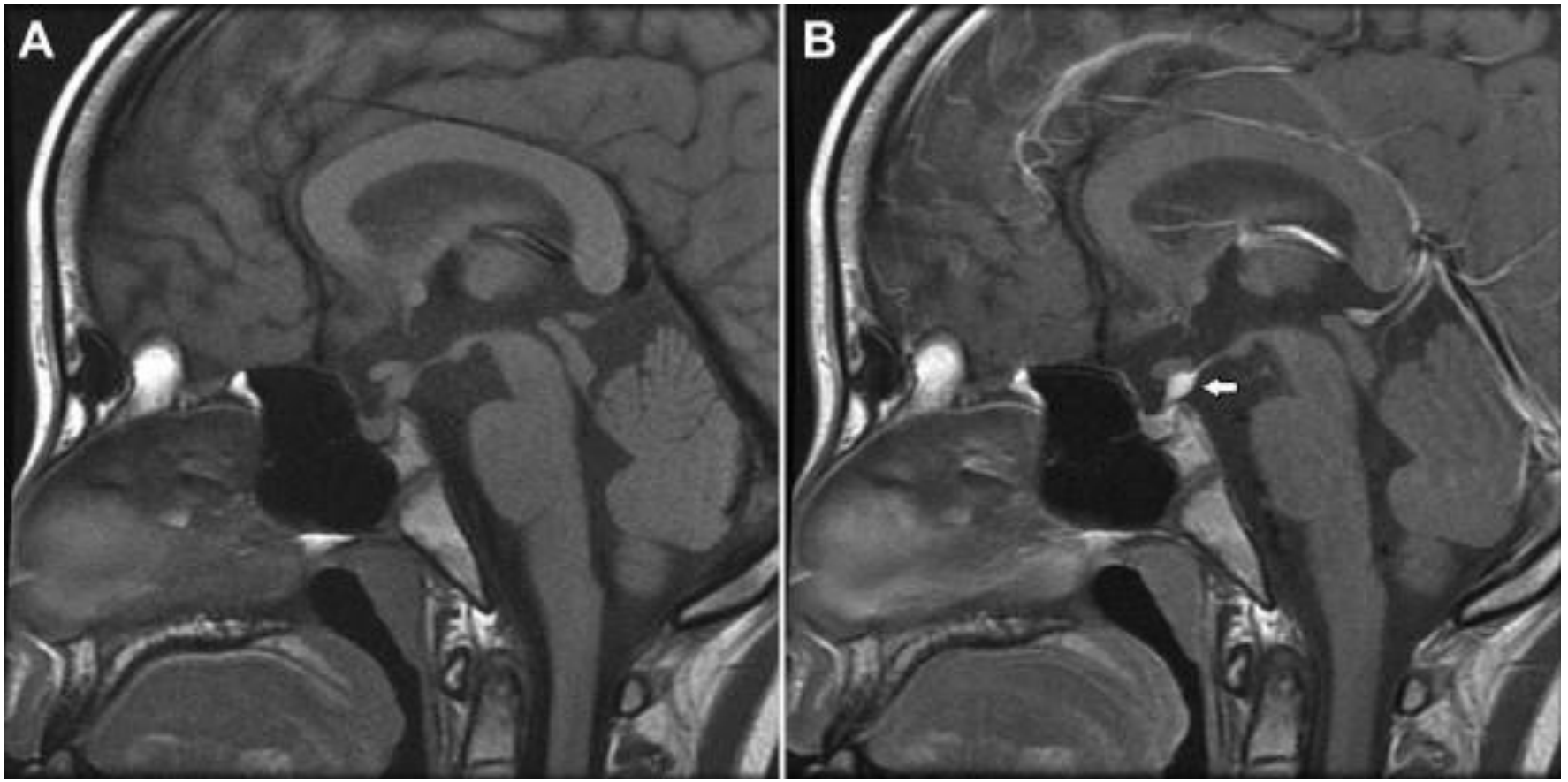
PP – 39%

Central DI – 70%



The above image is a non-contrast T1 MRI image of a normal pituitary gland. The white arrow point towards the ‘bright spot’ seen in the posterior pituitary. This finding is a result of phospholipid-rich granules that store arginine vasopressin (AVP) and oxytocin.

Sagittal T1-weighted MRI of the brain of a boy with Langerhans cell histiocytosis and central DI

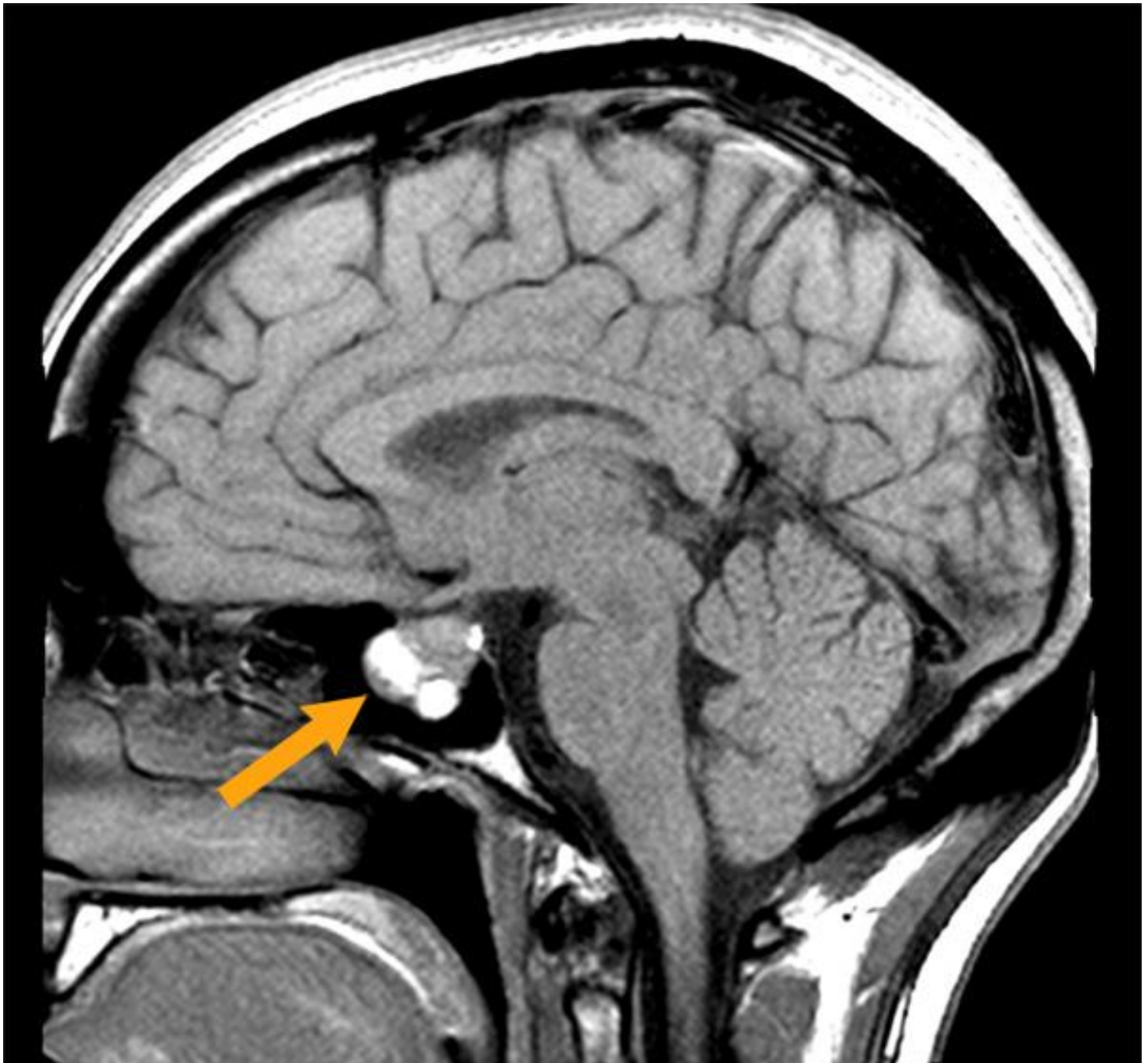


A. The normal posterior pituitary hyperintense signal is absent.

B. Thickened pituitary stalk (arrow) is visible after gadolinium enhancement (diameter > 3,5 mm) It may point towards inflammation or infiltrative diseases

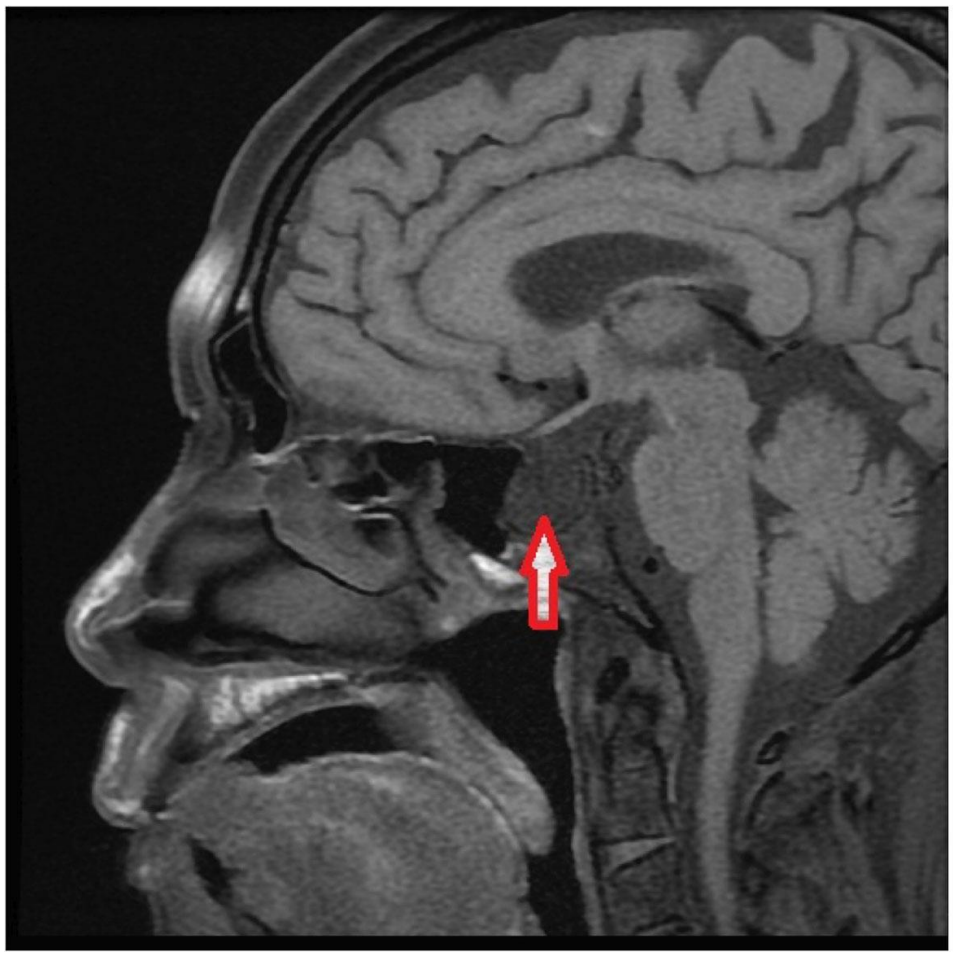
MRI investigation

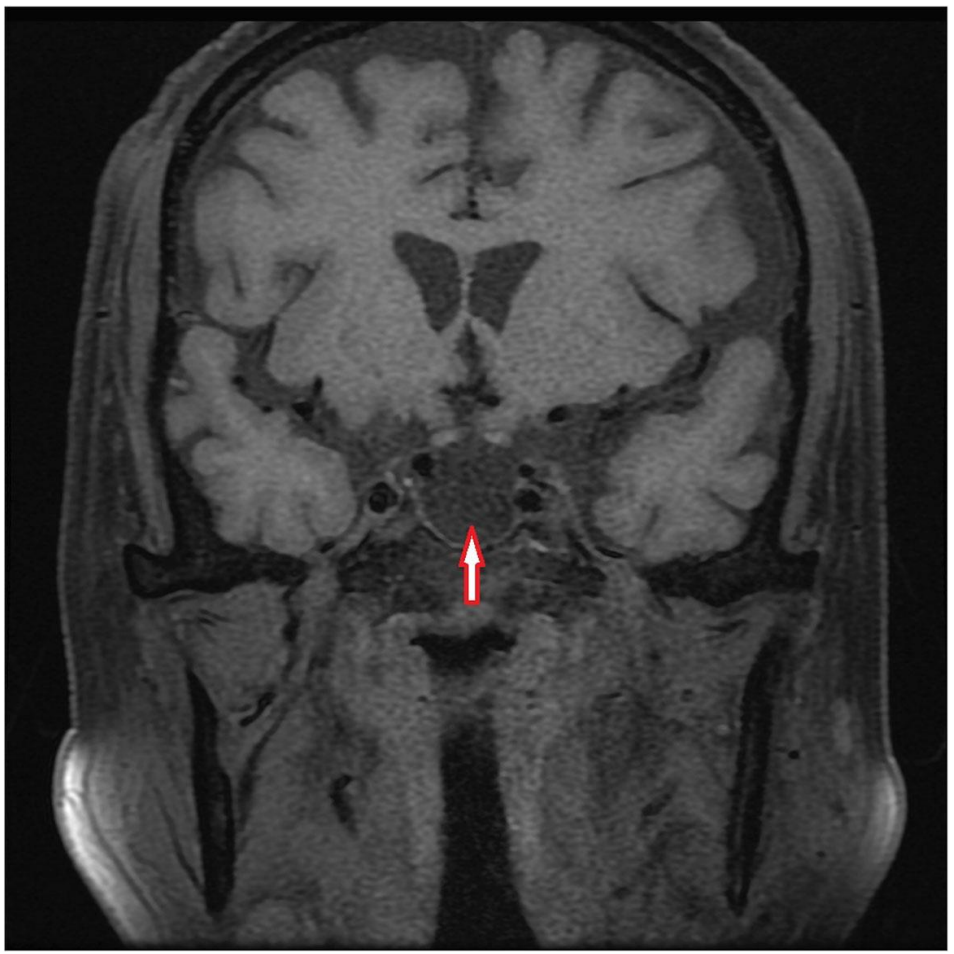
Adenoma



MRI investigation

Infiltrative or inflammatory changes





MRI investigation

These findings should not be used as a sole diagnostic criteria for diagnosing central DI

Diagnostic approach to polyuria–polydipsia syndrome

1. Confirm hypotonic polyuria via 24h urine collection (>50ml/Kg/24h, <800mOsm/kg)

2. Measure plasma sodium / osmolality

- <135 mmol/L / <280 mOsm/kg → **PP**
- Normal range → 3. Measure random copeptin
  - **<21.4 pmol/L** → 4. Measure Arginine-stimulated copeptin
    - **≤3.8 pmol/L** → **central DI**
    - **>3.8 pmol/L** → **PP**
    - 5. Unclear cases: Measure hypertonic saline stimulated copeptin
      - **≤4.9 pmol/L** → **central DI**
      - **>4.9 pmol/L** → **PP**
  - **≥21.4 pmol/L** → **Nephrogenic DI**
- ≥147 mmol/L / >280 mOsm/kg → Central / nephrogenic DI → Measure copeptin
  - **≤4.9 pmol/L** → **central DI**

Management of DI

1. **Central DI:** inranasal or oral desmopressin
2. **Nephrogenic DI** is difficult to treat since the patients have resistance to AVP
   - Submaximal doses of desmopressin
   - Correction of underlying disorder (e.g. hypercalcaemia)
   - Low-sodium diet
   - Thiazide diuretics (induced natriuresis)
   - NSAIDs (block prostaglandin synthesis, thereby increasing non-ADH-dependant water reabsorption