Polyuria

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DEFINITION

- Polyuria – The production of >3L of urine in 24 hrs.
- Urinary frequency – An increase in the number of times a person voids, irrespective of the net volume produced.
- Nocturia – Urinary frequency that is predominantly confined to nighttime hours (i.e. while supine).

POLYURIA RESULTS FROM ANY PROCESS THAT INVOLVES :

Sustained increase in water intake (Polydipsia).

Decreased ADH secretion (Central DI).

Decreased peripheral ADH sensitivity (Nephrogenic DI).

Solute diuresis.

MECHANISMS OF POLYURIA

Excess Intake of Water	Excess Tubular Solute (a.k.a. osmotic diuresis)			Defect in Tubular Reabsorption of Water (a.k.a. Diabetes Insipidus)	Polyuria Mimics (↑ urine frequency without ↑ urine volume)
	Excess Na^+	Excess glucose	Other		
Primary Polydipsia (a.k.a. "psychogenic polydipsia")	Conventional diuretics*	Poorly controlled diabetes mellitus*	Mannitol	Central DI (Low production of ADH) • Idiopathic • Post-neurosurgery • Trauma • Infiltrative diseases • Hypoxic injury • Tumor	UTI
Administration of a large volume of IV fluids		SGLT2 inhibitors (e.g. gliflozins)		Nephrogenic DI (Renal resistance to ADH) • Hereditary • Lithium • Hypercalcemia*	BPH Detrusor overactivity (a.k.a. "overactive bladder")

* Most common causes of true polyuria (though polyuria mimics are more common overall)

* There are also other proposed mechanisms of by which hypercalcemia causes polyuria

CAUSES

1. Physiological
2. Pathological
3. pharmacological
4. Iatrogenic

I. PHYSIOLOGICAL POLYURIA

Anxiety.

Protein rich diet **(Protein by-products are excreted in your urine, and the more protein you eat, the more your body needs to excrete, which means an increase in urination.)**

Compensatory mechanism to increased fluid intake.

II. PATHOLOGICAL POLYURIA

Polyuria caused by a disease of the kidney or disorder anywhere in the body.

1) Endocrine- DM, Central DI, Hyperparathyroidism .

2) Renal- ATN (diuretic phase), pyelonephritis, UTI, RTA2 ,BPH ,nephrogenic DI

3) Decreased Aldosterone by the adrenal gland in Addison's Disease.

4) Neurologic causes: neurologic damage

The damage of the hypothalamic ADH-producing neurons (by trauma or tumor), their axons or the posterior pituitary leads to post-traumatic central DI .

III. PHARMACOLOGICAL POLYURIA

Polyuria caused by administered medication such as Diuretics, Lithium, Glucocorticoids, high doses of vitamin D.

IV.IATROGENIC

excessive IVF, mannitol infusion, radiocontrast media .

CLINICAL APPROACH TO POLYURIA

HISTORY

1- PP

2- PC (polyuria) you should ask about duration, onset, any recent clinical factors that may cause polyuria (IV fluids, tube feedings, resolution of urinary obstruction, head trauma, surgery)

 # you should ask about thirst, fatigue, wt loss, bone pain.

3- Review of systems

Seek symptoms suggesting possible causes, including dry eyes and dry mouth (Sjögren syndrome) and weight loss and night sweats (cancer).

4- PMH

You should ask about any conditions associated with polyuria , including diabetes mellitus, psychiatric disorders and hyperparathyroidism.

History of transurethral resection of prostate → post obstructive diuresis

History of neurosurgery → central diabetes insipidus

5- DH

If the patient took any drugs → that cause polyuria like diuretics, lithium.

6- FH

Of polyuria and excessive water drinking should be noted, if there is anyone in the family have diabetes mellitus or diabetes insipidus.

7-SH

Caffeine and alcohol consumption and high protein diet.

PHYSICAL EXAMINATION

The general examination should note signs of obesity (as a risk factor for diabetes mellitus), Dehydration or cachexia, weakness and wasting of the body due to severe chronic illness that may reflect an underlying cancer or eating disorder.

The head and neck examination should note dry eyes or dry mouth (Sjögren syndrome).

Skin examination should note the presence of any hyperpigmented or hypopigmented lesions and ulcers.

DIAGNOSIS ON EXAMINATION

- Failure to thrive → DM,DI
- Fever → UTI
- Not oriented → Schizophrenia
- Pallor → Sickle cell anemia ,CRF
- Edema → Renal failure

INVESTIGATION

24 hour urine output

↓

More than 2.5 liters per day

↓

POLYURIA

↓

Further investigations

URINALYSIS

will help determine if there is glucose in the urine and thus may suggest DM

Osmolality of the urine

- Low urine osmolality <300 mosm/kg → DI

-high urine osmolality >600 mosm/kg → Less likely to be DI

White blood cells count >5-10 → UTI

Specific gravity <1.005 → DI

BLOOD TESTS

Serum electrolytes especially calcium (elevated level of calcium may cause polyuria like hyperparathyroidism).

Blood glucose

Plasma osmolality
High plasma osmolality >300 mosm/kg ⟶ suggest DI

OTHER TESTS

1) Vasopressin response test

To differentiate central DI from nephrogenic DI

2) X-ray of the skull

3) Ct scan for the brain if suspected pituitary diabetes insipidus

4) Renal biopsy if suspected renal disease

COMPLICATION

Fatigue

Dizziness

Tachycardia

Hypotension shock

MANAGEMENT

Admit to hospital if significantly dehydrated, whatever the likely cause.

Fluid balance and electrolyte disturbance will need to be corrected.