Approach to Polyuria

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Definition

Urine output > 40ML/ Kg/ HR

or

Urine output > 3 L/ / day

CAUSES OF POLYURIA

- INCREASED FLUID INTAKE
- INCREASED URINARY SOLUTE EXCRETION
- IMPAIRED URINARY CONCENTRATION

INCREASED FLUID INTAKE

- IATROGENIC
- COMPULSIVE WATER DRINKING (PSYCHOGENIC POLYDIPSIA)

INCREASED URINARY SOLUTE EXCRETION

- **OSMOTIC DIURESIS**

1. DIABETES MELLITUS
2. MANNITOL TREATMENT

- **SALT LOSS**

1. ADRENAL INSUFFICIENCY
2. DIURETICS
3. CEREBRAL SALT WASTING
4. ALDOSTERONE RESISTANCE

IMPAIRED URINARY CONCENTRATION

- **<u>INEFFICIENT ADH ACTION (DIABETES INSIPIDUS)</u>**

1. CENTRAL
2. NEPHROGENIC

- **<u>RENAL DISORDERS</u>**

1. R T A
2. BARTTER SYNDROME
3. GITELMAN SYNDROME

INEFFICIENT ADH ACTION (DIABETES INSIPIDUS)

- **<u>CENTRAL (NEUROGENIC) DIABETES INSIPIDUS:</u>**

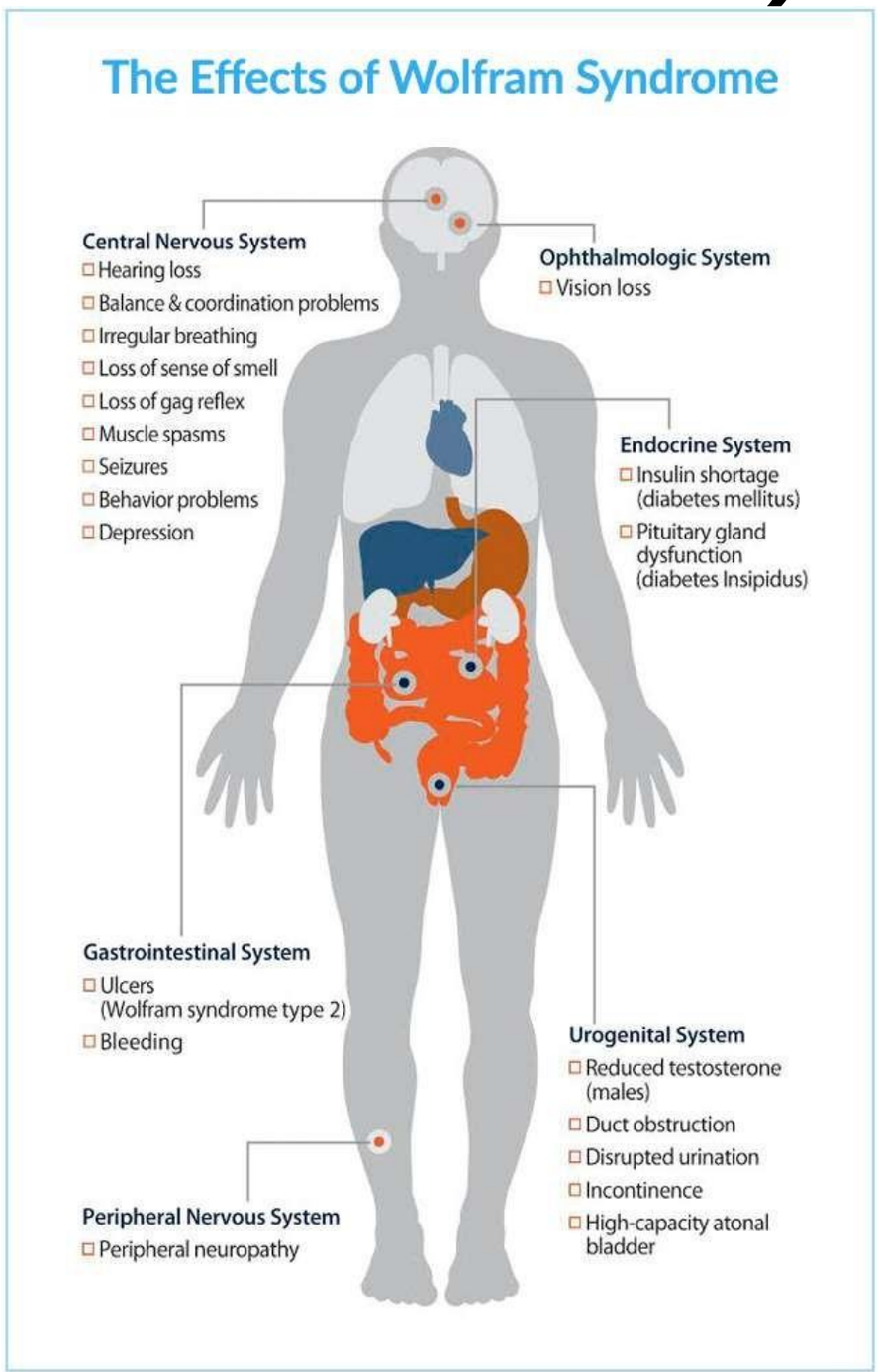
1. ***<u>GENETIC DEFECTS:</u>*** *- AR, AD, WOLFRAM DIDMOAD SYNDROME*

2. ***<u>MALFORMATIONS:-</u>*** *SEPTO- OPTIC DYSPLASIA, HOLOPROSENCEPHALY, ANENCEPHALY*

3. ***<u>NEUROLOGICAL INSULTS:-</u>*** *HEAD TRAUMA, NEURO SURGERY, INFECTION, BRAIN DEATH*

4. ***<u>INFILTRATIVE DISORDERS:-</u>*** *SARCOIDOSIS, HISTIOCYTOSIS*

5. ***<u>CNS TUMOURS:-</u>*** *CRANIOPHARYNGIOMA, GERMINOMA, PINEOLOMA*



INEFFICIENT ADH ACTION (DIABETES INSIPIDUS)

- **<u>NEPHROGENIC DIABETES INSIPIDUS</u>**

1. ***<u>GENETIC:-</u>*** XL (V2 RECEPTOR DEFECT), AR, AD (AQUAPORIN DEFECT)

2. ***<u>ACQUIRED:-</u>***
   HYPOKALEMIA,
   HYPERCALCEMIA,
   OBSTRUCTIVE UROPATHY,
   NEPHROCALCINOSIS.

APPROACH

1. HISTORY
CLINICAL EXAMINATION
INVESTIGATIONS

HISTORY

- **AGE OF ONSET :-** *CONGENITAL/ ACQUIRED*
- **H/ O FEVER:-** *UTI*
- **FAILURE TO THRIVE:-** *DM, NEPHROGENIC DI, RTA, CAH, BARTTER*
- **H/ O HEAD TRAUMA, NEUROSURGERY:-** *CENTRAL DI*
- **H/ O MENINGITIS:-** *CENTRAL DI*
- **H/ O WEIGHT LOSS:-** *DM, RTA*
- **H/ O RASH, SEBORRHEAS:-** *HISTIOCYTOSIS*
- **H/ O MUSCLE WEAKNESS:-** *HYPOKALEMIA- RTA, BARTTER*
- **H/ O DRUG INTAKE:-** *MANNITOL, DIURETICS, OUTDATED TETRACYCLINES*

HISTORY cntd..

- **SYMPTOMS OF INCREASED ICT:-** *CNS TUMOURS*
- **H/ O POLYURIA, SHOCK IN NEWBORN PERIOD:-** *CAH*
- **H/ O CONSTIPATION, PARAESTHESIA:-** *HYPERCALCEMIA*
- **H/ O PSYCHOLOGICAL PROBLEMS:-** *PSYCHOGENIC POLYDIPSIA*
- **H/ O ABDOMINAL CRAMPS, ARTHRALGIA etc., :-** *SICKLE CELL ANAEMIA*

CLINICAL EXAMINATION

- **ANTHROPOMETRY:-** *TO R/ O FTT: DM, DI, RTA, CAH*
- **FEVER:-** *UTI*
- **MENTAL RETARDATION:-** *CNS MALFORMATIONS*
- **NEUROLOGICAL DEFICITS:-** *CNS PATHOLOGIES*
- **GENITAL AMBIGUITY:-** *CAH*
- **MIDLINE DEFECTS:-** *CENTRAL DI*
- **FEATURES OF RICKETS:-** *RTA, RENAL FAILURE*
- **ACIDOTIC BREATHING:-** *RTA*
- **RASH, SEBORRHOEA, EAR DISCHARGE:-** *HISTIOCYTOSIS*
- **HYPER-PIGMENTATION:-** *ADRENAL INSUFFICIENCY*
- **MUSCLE WEAKNESS, NECK FLOP:-** *HYPOKALEMIA- RTA, BARTTER.*

** ALSO LOOK FOR SIGNS OF DEHYDRATION AND SHOCK*

INVESTIGATIONS

24 HOUR URINE OUTPUT

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> 40 ML/ KG/ HR OR > 3l/ DAY

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POLYURIA

↓

FURTHER INVESTIGATIONS

COMPLETE URINE EXAMINATION

- WBCs :- UTI
- SUGAR :- DM
- SPECIFIC GRAVITY :-
  < 1.005- DI
- URINE OSMALILITY :- < 300 mOsm/ kg- DI

OTHER INVESTIGATIONS

- UREA, CREATININE
- SERUM ELECTROLYTES
- CALCIUM
- BLOOD GAS ANALYSIS
- BLOOD GLUCOSE
- PLASMA OSMOLALITY

- HIGH PLAMSA OSMOLALITY > 300 mOsm/ kg
- LOW URINE OSMOLALITY < 300 mOsm/ kg
- URINE SPECIFIC GRAVITY < 1.005
- SERUM SODIUM > 145 mmol/ L

CENTRAL DI

- SERUM OSMOLALITY< 270
- URINE OSMOLALITY > 600 mosm/ kg
- URINE SPECIFIC GRAVITY > 1.010

DI
UNLIKELY

HIGH PLASMA OSMOLALITY < 300 mOsm /kg

WATER DEPRIVATION TEST

SERUM OSMOLALITY > 270 mOsm/ kg

WATER DEPRIVATION TEST

- DETERMINES ABILITY OF KIDNEYS TO CONCENTRATE URINE
- USEFUL IN THE DIAGNOSIS OF DI
- REQUIRES CAREFUL SUPERVISION BECAUSE DEHYDRATION AND HYPERNATREMIA MAY OCCUR

METHOD

- BEGIN THE TEST AFTER 24 HOUR PERIOD OF ADEQUATE HYDRATION AND STABLE WEIGHT
- OBTAIN A BASELINE WEIGHT AFTER BLADDER EMPTYING
- RESTRICT FLUIDS FOR 7 HOURS
- MEASURE BODY WEIGHT, URINE SPECIFIC GRAVITY AND VOLUME HOURLY
- CHECK SERUM SODIUM, URINE AND SERUM OSMOLALITY EVERY 2ND HOURLY
- TERMINATE THE TEST IF WEIGHT LOSS APPROACHES 5%.

INTERPRETATION

WHEN WATER IS DEPRIVED

will concentrate urine (to 500- 1400 mOsm/ L

plasma osmolality will be 288- 291 mOsm/ l

urine specific gravity rises to at least 1.010

urine volume decreases significantly

there will be no appreciable weight loss

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NORMAL INDIVIDUALS & PSYCHOGENIC DI

urine osmolality remains < 150- 300 mOsm/ L

plasma osmolality > 300 mOsm/ L

urine specific gravity < 1.005

no significant reduction in urine volume

weight loss upto 5% usually occurs

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CENTRAL OR NEPHROGENIC DI

VASOPRESSIN RESPONSE TEST

TO DIFFERENTIATE CENTRAL DI FROM NEPHROGENIC DI

BASELINE URINE OSMOLALITY IS RECORDED

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VASOPRESSIN INJECTION GIVEN

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URINE OSMOLALITY MEASURED AT 1 AND 4 HOURS AFTER INJECTION

↓

INCREASE IN URINE OSMOLALITY

↓	↓
> 50 % INCREASE FROM BASELINE	**< 50% INCREASE FROM BASELINE**
↓	↓
CENTRAL DI	**NEPHROGENIC DI**

OTHER TESTS

- CENTRAL DI:- MRI OF HYPOTHALAMIC- PITUITARY REGION
- NEPHROGENIC DI:- RENAL IMAGING
- GENETIC STUDIES AS REQUIRED

TREATMENT

CENTRAL DI

- FLUID THERAPY
- VASOPRESSIN ANALOGS
- ACQUEOS VASOPRESSIN

FLUID THERAPY

- With an intact thirst mechanism and free access to oral fluids, a person with complete DI can maintain plasma osmolality and sodium in the high normal range, although at great inconvenience.

- Neonates and young infants are often best treated solely with fluid therapy, given their requirement for large volumes (3 L/ square meter/ day) of nutritive fluid.

VASOPRESSIN ANALOGS

- Treatment of Central DI in older children is best accomplished with the use of DDAVP.

- DDAVP is available in an intranasal preparation (onset 5- 10 min) and as tablets (15-30 min).

- Use of oral tablets requires at least 10 fold increase in the dosage compared with intranasal preparation.

- To prevent water intoxication, patients should have at least 1 hr of urinary breakthrough between doses each day and be advised to drink in response to thirst sensation.

ACQUEOS VASOPRESSIN

- Central DI of acute onset following neurosurgery is best managed with continuous administration of synthetic aqueous vasopressin (Pitressin).

- Under most circumstances, total fluid intake must be limited to 1 L/ square meter/ day during antidiuresis.

- A typical dosage of intravenous vasopressin therapy is 1.5 mU/kg/hr, which results in a blood vasopressin concentration of approximately 10 pg/ml.

- On occasion, following hypothalamic (but not transspheniodal) surgery, higher initial concentrations of vasopressin may be required, which has been attributed to the release of a vasopressin inhibitory substance.

NEPHROGENIC DI

- The treatment of acquired NDI focuses on eliminating, if possible, the underlying disorder, such as offending drugs, hypercalcemia, hypokalemia, or ureteral obstruction.

- Congenital DI is often difficult to treat.

- The main goals are to ensure that intake of adequate calories for growth and to avoid severe dehydration.

- Foods with highest ratio of caloric content to osmotic load (Na <1 mmol/ kg/ 24 hr) should be ingested to maximise growth and to minimise the urine volume required to excrete the solute load.

- Pharmacologic approaches to the treatment of NDI include the use of thiazide diuretics and are intended to decrease the overall urine output.

- Thiazides appear to induce a state of mild volume depletion by enhancing sodium excretion at the expense of water and by causing a decrease in the glomerular filtration rate, which results in proximal tubular sodium and water reabsorption.

- Indomethacin and amiloride may be used in combination with thiazides to further reduce polyuria.

- High dose DDAVP therapy, in combination with indomethacin, has been used in some subjects with NDI. This treatment could prove useful in patients with genetic defects in the V2 receptor associated with a reduced binding affinity for vasopressin.

THANKYOU