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PARKLAND MEMORIAL HOSPITAL
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GRAND ROUNDS [Floyd C. Rector]

PATHOLOGIC WATER DRINKING

Case 1: [redacted] 52 y/o W/M [redacted]

Following a head injury in 1930 the patient developed malaise, weakness, headaches, progressive polydipsia and polyuria with daily urine volumes in excess of 4 gallons. In 1932 the diagnosis of diabetes insipidus was made. Treatment with pitressin reduced urine output from 4 gallons to less than 2 gallons daily without improvement of other symptoms. He entered the Army in 1942 and diagnosis of diabetes insipidus was reaffirmed. While in the Army he was restarted on pitressin but urine output remained high. In 1956 he was again treated with pitressin but became confused and agitated after 3 days. He was told that he had an allergic reaction to pitressin. During the next few years he continued to have polyuria with urine volumes in range of 10 to 16 L daily. In addition to polyuria and polydipsia, he had frequent attacks precipitated by extreme anxiety. During these attacks he would become tense, feel pains shooting down both arms, he was unable to speak, complained of dizziness and had diplopia and blurring of vision.

On admission to the hospital his urine output was 7 to 10 L daily. Examination revealed a frightened, insecure man, but otherwise was normal. Electrocardiogram, chest X-rays, and skull films were normal. Blood chemistries were: Na 135 mEq/L; K 4.1 mEq/L; Ca 9.4 mg%; BUN 16 mg%.

Water deprivation test

	Urine flow ml/min	Urine Osm mOsm/Kg	Serum Na mEq/L
[redacted] AM	8.0	69	136
[redacted] PM	2.5	148	138
[redacted] PM	1.0	403	138
[redacted] PM	0.75	520	135
[redacted] PM	0.5	609	133
[redacted] PM	0.4	672	133

Impression:

Psychogenic water drinker with normal response to water deprivation.

Course in hospital:

Patient was reassured that he did not have a serious organic diseage. Daily urine volumes thereafter fell to approximately 2 liters. Random urine specific gravities rose to 1.017 to 1.020.

Case 2: [redacted]. 8 y/o W/M [redacted]

Seven months before admission to the hospital this 8 y/o boy began drinking large quantities of water (3 glasses every hour) associated with marked polyuria and urinary frequency (q 1 hour during the day and q 2 hours during the night).

One month before admission he had the mumps. When seen by his family doctor urine specific gravity was 1.002. Diagnosis of diabetes insipidus was considered. The patient was referred to a neurosurgeon who obtained the history of difficulty in writing for one year and episodic fogging or vision for one month. Examination was unremarkable except for positive Babinski and Oppenheim on the right.

The patient was then referred to [redacted] for further evaluation. Physical examination was completely within normal limits; there were no pathological reflexes at this time. Skull X-rays were negative. Visual fields were normal. Electroencephalogram suggested a posterior, midline lesion extending to the left.

On admission blood chemistries were: Na 159 mEq/L; K^+ 4.9 mEq/L; Cl 129 mEq/L; CO_2 26 mEq/L; BUN 2 mg%; urine specific gravity was 1.002.

After 8 hours of water deprivation urine volume averaged 5 ml/min., urine specific gravity was 1.010 and serum Na was 145 mEq/L.

Hickey-Hare test:

1) 20 ml H_2O per Kg body weight given over period of 1 hour. Urine flow was then 3 to 3.6 ml/min.
2) 0.25 ml of 2.5% NaCl per min. per Kg was given for 45 min. (300 ml).

Urine flows were:		Sp. Gr.
15 min	- 3.6 ml/min	1.006
30 min	- 2.0	1.010
45 min	- 2.9	1.008
75 min	- 3.1	
105 min	- 2.3	
135 min	- 3.5	1.007

On the basis of the polyuria in the face of hypernatremia and the failure to respond to hypertonic saline, a diagnosis of organic diabetes insipidus was made and treatment with pitressin tannate in oil instituted. For the 24 hours preceding the first injection of pitressin the urine volume was 8 100 ml. Following the injection of 4 units of pitressin tannate daily for 3 days, daily urine volumes were 5300 ml, 5 890 ml and 4200 ml.

Pitressin injections were then discontinued and 4 days later the water deprivation test repeated. The following results were obtained:

	Urine Flow cc/hr	Urine Osmolarity mOsm/L	Serum Sodium mEq/L
10:00 AM	60	161	145
12:30 PM	55	249	146
2:30 PM	20	448	147
4:30 PM	16	565	148
overnight		623	

Interpretation:

Essentially normal response to water deprivation. Therefore, the polyuria most likely due to psychogenic polydipsia.

Retrospective psychological history revealed that the patient was a very bright, sensitive individual who was seriously disturbed by the sadistic behavior of his teacher. In addition, his father had diabetes mellitus and frequently discussed his own symptoms of polyuria and polydipsia in the presence of the patient.

On admission the hypernatremia, low urine specific gravity and the failure to respond to hypertonic NaCl probably represented a transient failure to secrete ADH or to respond to ADH after prolonged pathologic water drinking.

Case 3: [redacted] 34 y/o W/M - [redacted]

Patient was in a racing car accident in [redacted] 1970, suffered spinal cord compression at C 6-7 and thereafter was quadriplegic. He was transferred to [redacted] for physiotherapy and did well until [redacted] 1970 when he became depressed and exhibited marked behavioral changes. He developed persistent spiking fever, polydipsia and polyuria with urine volumes up to 18 liters/day. He appeared to have decreased sweating. There was a urinary tract infection which cleared with antibiotics, but the fever persisted. The cause of the fever was never established and was eventually attributed to autonomic dysfunction.

Blood chemistries were: Na 136 mEq/L; K 4.3 mEq/L; Ca 8.2 mg%; BUN 7 mg%; creatinine 0.5 mg%. Intravenous pyelogram did not show any evidence of urinary tract obstruction. Urine volumes ranged between 7 and 18 liters/day and urine osmolalities were approximately 100 mOsm. Urine sodium excretion was as high as 800 mEq/day, but was roughly equal to intake.

He was considered to have diabetes insipidus plus renal salt wastage secondary to pyelonephritis. He was given 10 to 30 units pitressin daily which decreased urine volumes to 4 liters/day, but resulted in a fall in serum sodium to 116 mEq/L accompanied by somnolence and confusion. Urine sodium excretion increased above intake (400 mEq vs 300 mEq). Discontinuation of pitressin and fluid restriction resulted in decreased urine volume to 2 liters daily and maintenance of sodium balance on 100 mEq sodium diet.

Impression:

Psychogenic water drinking secondary to quadriplegia. The high rate of salt excretion was due to excessive water intake and salt loads and was not due to primary renal salt wastage.

Case #4: 48 y/o W/F

This woman was first hospitalized on [redacted]-70 because of seizures. At that time: Serum Na 102 mEq/L; K 2.9 mEq/L; Ca 9 mg% and urine sp. gravity 1.006. EKG, skull X-rays, IVP and brain scan were normal. She was discharged with a diagnosis of grand mal epilepsy and was placed on Dilantin and phenobarbital. She was readmitted to that hospital on [redacted]-70 after two seizures and at that time her laboratory data were: Serum Na 116 mEq/L; BUN 6 mg%. She was discharged, but returned to the hospital on [redacted]-70 after 3 to 4 episodes of generalized seizures. At that time the history was obtained that she had been drinking 5 to 6 gallons of water daily for at least two years.

On that admission the laboratory data were: Serum Na 103 mEq/l; BUN 3 mg% and urine specific gravity of 1.001. During the first few hours in the hospital she excreted 1000 to 1300 ml urine per hour, containing 67 mEq/L Na. She was considered to be a salt waster and was given 2000 ml of 3% NaCl. This increased her serum Na to 130 mEq/L and osmolality to 270 mOsm/kg. Urine osmolality was 465 mOsm/Kg at that time suggesting the possibility of inappropriate secretion of ADH.

On the first day of hospitalization she had a urine output of 18,000 ml and an intake of 19,000 ml. During the next 3 to 4 days she was able to take food and liquids orally. She excreted large volumes of urine containing 90 mEq/L of sodium. She was given 1 mg Florinef and urine sodium concentration fell to 5 mEq/L. On a 15 mEq sodium diet for 5 days urine sodium excretion was 50 to 60 mEq/L and fell to 10 mEq after 5 mg of DOCA I.M. During this time her fluid intake was not rigidly controlled and she drank 6 to 7 liters of H_2O daily. Subsequently, she was transferred to a locked ward and restudied. On a 15 mEq sodium diet with fluids restricted to 1500 ml per day she had no evidence of salt wastage.

Conclusion:

1. Pathologic water drinking
2. Salt wastage secondary to large water intake and over-expansion of ECV
3. A transient period of "apparent inappropriate secretion of ADH" during correction of hyponatremia.

Case #5: [redacted]. 43 y/o W/M [redacted]

This man had epilepsy since 1 year of age, treated for several years with Dilantin, phenobarbital and mysoline. He had only a first grade education, cannot read or write, has lived all his life with his parents and has never worked. He has drunk large volumes of fluids for several years. In 1963 he was first noted to have hypertension. In [redacted] 1970 he was seen in the emergency room with BP of 210/120

and serum Na 130 mEq/L. He was started on Naqua and Ismelin. He was brought to the hospital 36 hours later after having had 6 to 8 grand mal seizures.

Admission blood chemistries:

Na 113 mEq/L; K 2.6 mEq/L; BUN 7 mg%; creatinine 1.0 mg%. Urine sp. gravity 1.010.

Course in hospital:

He was treated with isotonic salt solutions and serum Na rose to 135 mEq/L. Urine volumes ranged between 3 and 6 liters per day on ad lib fluids. On the 8th hospital day he was again started on Naqua and low-salt diet for his hypertension. Two days later the serum Na had fallen to 112 mEq/L and the patient was lethargic and confused. The hyponatremia corrected after stopping Naqua and salt replacement.

In [redacted] 1971 he was seen in EOR for a seizure and again was noted to be hypertensive and was given Naqua and Ismelin. During the next few days he had increased seizure activity, had frequent vomiting and became more lethargic. Ten days after starting Naqua he was brought to the hospital in coma. On admission: serum Na 103 mEq/L; K^+ 3.4 mEq/L; Ca 8.9 mg%; BUN 22 mg%; urine osmolality 257; urine Na 29 mEq/L. He was initially treated with 750 ml 5% NaCl and then stabilized on a regular diet with serum Na of 137.

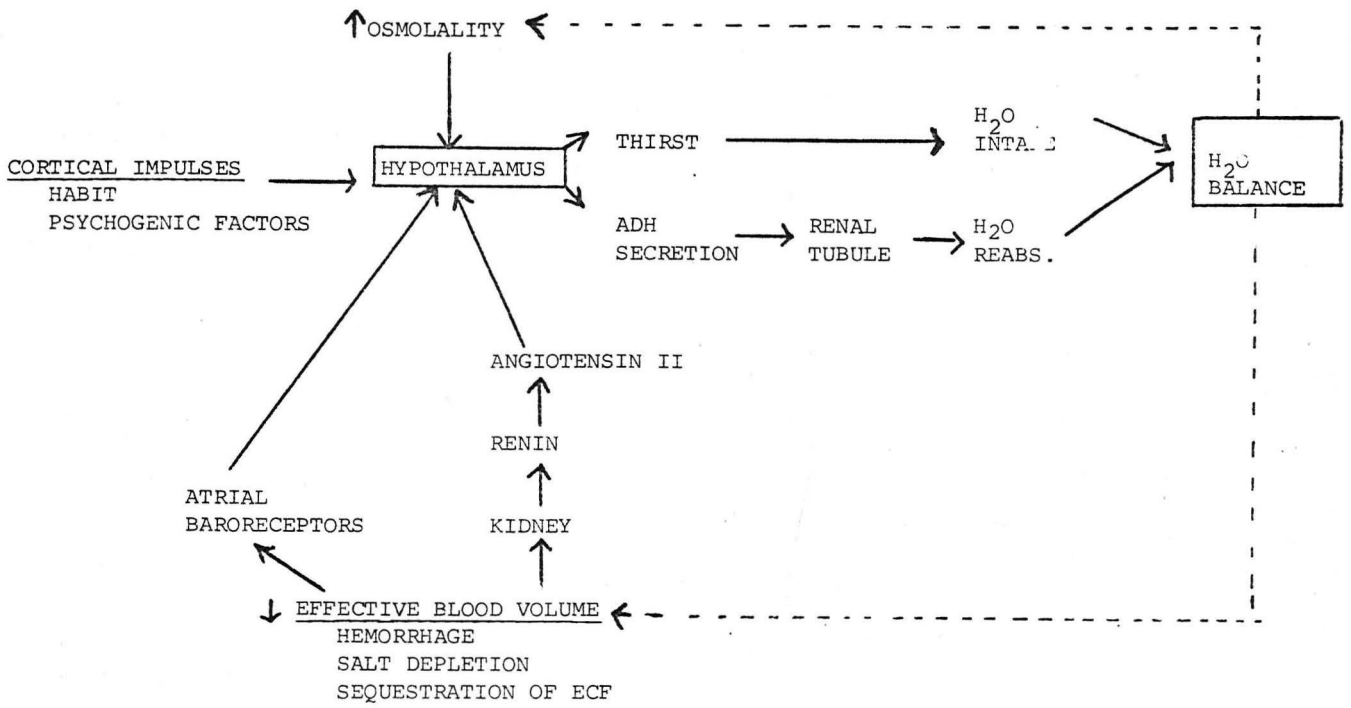
Additional studies:

1) On 150 mEq Na diet and ad lib fluids urine volumes ranged between 5 and 7 L daily.
2) On 15 mEq Na diet urine Na excretions fell to 8 mEq/day within 4 days. No evidence of salt wastage.
3) On 150 mEq Na diet ad lib fluid and Naqua serum sodium fell to 125 mEq/L.
4) On 50 mEq Na diet ad lib fluid and Naqua serum sodium fell to 113 mEq/L.
5) After 12 hours of water deprivation Uosm was 120 mOsm/Kg.
6) After 5 units pitressin tannate in oil Uosm rose to 318 mOsm/Kg.

Conclusions:

1) Pathologic water drinking
2) Thiazide-induced hyponatremia
3) Impaired ADH released
4) Vasopressin-resistant concentrating defect.

REGULATION OF WATER BALANCE AND OSMOLALITY



POLYDIPSIC AND POLYURIC STATES

I OSMOTIC DIURESIS

- A. Glycosuria
- B. Urea loads

II WATER DIURESIS

- A. Increased water ingestion
  1. Psychogenic water drinking
  2. Quadriplegia - autonomic dysfunction versus psychogenic factors
  3. Potassium deficiency
  4. Salt depletion - (? increased renin)

- B. Impaired secretion of vasopressin
  1. True diabetes insipidus
  2. Potassium deficiency
  3. Prolonged psychogenic water drinking

- C. Resistance of renal tubule to vasopressin
  1. Hereditary nephrogenic diabetes insipidus
  2. Potassium deficiency
  3. Hypercalcemia
  4. Obstructive uropathy
  5. Prolonged water diuresis
     a. diabetes insipidus
     b. psychogenic water drinking

Diagnostic Tests in Polyuric States

Disorder	Serum Osmolality and Sodium	Hypertonic Saline Infusion	Water Deprivation	Vasopressin
Pathologic Water Drinking	<270 mOsm/kg <135 mEq/L	urine ↓ flow	Uosm > Posm	Uosm > Posm
True Diabetes Insipidus	>295 mOsm/kg >145 mEq/L	no change	Uosm < Posm	Uosm > Posm
Nephrogenic Diabetes Insipidus	>295 mOsm/kg >145 mEq/L	no change	Uosm < Posm	Uosm < Posm
Comments		Saline diuresis may occur in pathologic water drinker	a) Must monitor weights and serum Na to detect cheating. b) Pathologic H_2O drinkers may have defect in vasopressin release or response	a) Pathologic water drinker may be vasopressin resistant

EXCRETION OF SOLUTE-FREE WATER

I Delivery of Filtrate to Distal Diluting Segment
- A. GFR
- B. Proximal reabsorption - controlled by ECV

II Sodium Reabsorption in Ascending Limb of Henle

III Water permeability of Distal Nephron
- A. ADH
- B. Hydrocortisone
- C. Calcium

IMPAIRED WATER EXCRETION FOLLOWING THIAZIDES

1. Inhibition of Distal Sodium Reabsorption
2. Salt depletion and ↓ ECV
3. Stimulation of Proximal Sodium Reabsorption
4. Decreased Distal Delivery of Filtrate
5. Decreased Solute-free Water Formation and Excretion.

NON-EDEMATOUS HYPONATREMIC STATES

A. PATHOLOGIC WATER DRINKING -

1. ECV expanded
2. high urine sodium
3. urine osmolality - usually hypotonic, but may be isotonic or hypertonic.

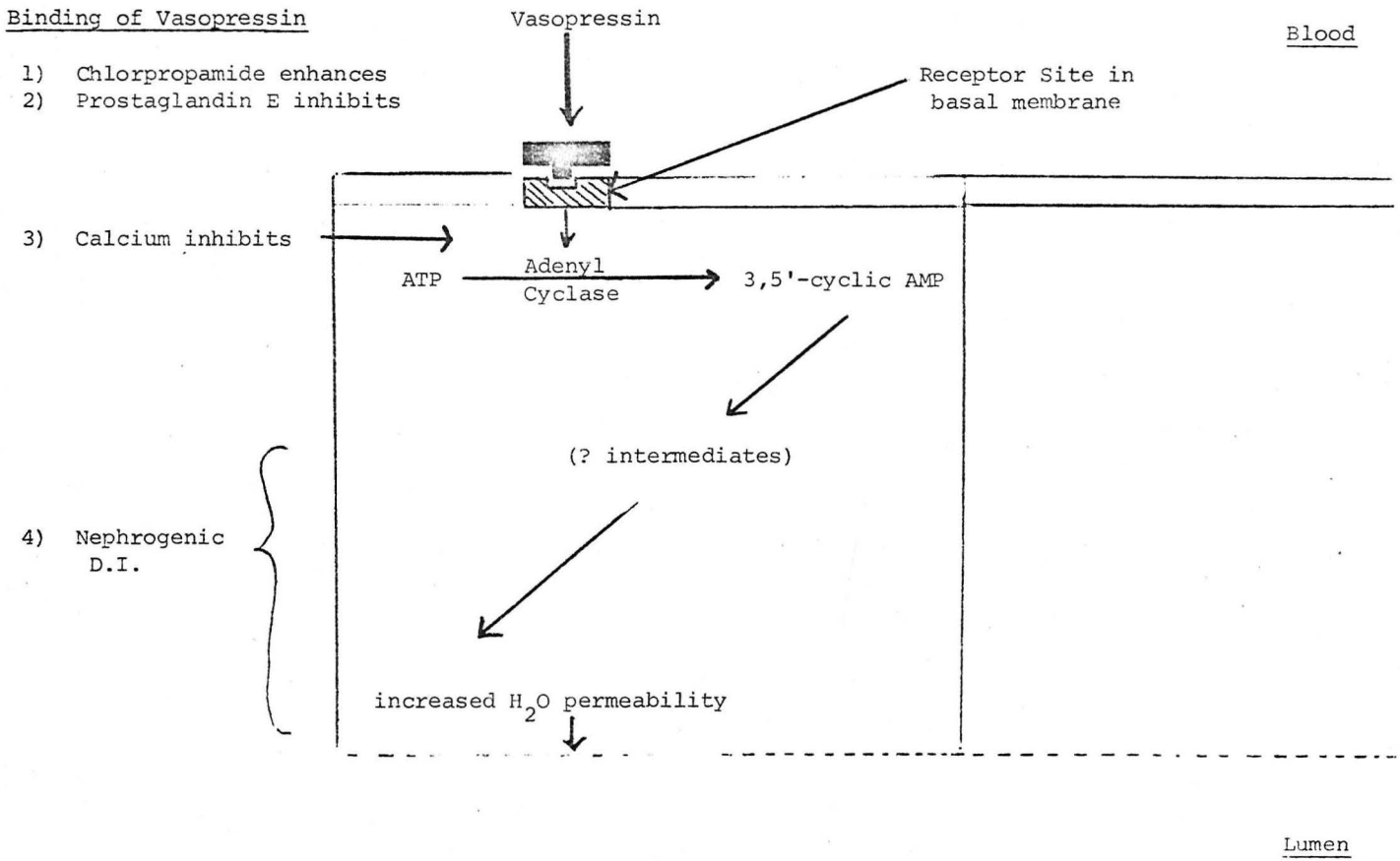
B. SALT DEPLETION (↑ renin → ↑ ADH + ↑ thirst)

1. ECV usually contracted, but may be re-expanded to normal by H_2O.
2. In absence of renal disease urine sodium is usually low; may rise as ECV re-expanded with H_2O.
   In presence of renal disease urine sodium is high.
3. Urine osmolality usually hypertonic.

C. SYNDROME OF INAPPROPRIATE SECRETION OF ADH

1. ECV expanded
2. high urine sodium
3. urine osmolality - hypertonic.

CELLULAR ACTION OF VASOPRESSIN



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