PARKLAND MEMORIAL HOSPITAL

MEDICAL GRAND ROUNDS 14 November 1968

METABOLIC ALKALOSIS

CASE 1: [redacted]

This 42 year old [redacted] man was admitted to [redacted] for the sixth time on [redacted] 1968. He had a history of peptic ulcer disease dating back to 1947 with at least two episodes of hematemesis and melena, requiring transfusions. He had persistent epigastric pain for 20 years and admitted to the ingestion of large quantities of milk and baking soda, in addition to commercial antacids. There were three proven episodes of acute pancreatitis in 1965-1966; however, there was no pancreatic calcifications on X-ray nor evidence of chronic pancreatitis.

Since 1965 he had evidence of chronic renal disease with azotemia (BUN 35-90). Bilateral retrograde pyelograms revealed small irregular kidneys and trabeculated bladder. No evidence of urinary tract infection. Creatinine clearances in the past year were between 5-10 ml/min.

Evaluation for hyperparathyroidism was equivocal. In [redacted] 1966 serum calciums were 10.4, 11.4 and 10.6 mg% and serum P 5.3-7.5 at a time when the BUN was 50 mg%. More recently serum calciums have been 8.0-9.5 mg%.

In the past 5 years he has had repeated episodes of pneumonia. Skin tests for histoplasmosis and PPD have been positive. Sputum was AFB positive and grew out photochromogens. Chest X-rays have shown bilateral fiberosis and a linguar infiltrate.

Physical examination:

BP 170/100, P 88, R 40 (shallow). Anasarca. Cardiomegaly without gallop. Shallow diaphragmatic respirations. Coarse rales diffusely.

Laboratory data:

Hgb. 7.7 gm%, Hct 21%. Urine pH 8.0-9.0.
BUN 55 mg%, creatinine 7.0 mg%. Na 147 mEq/L, K 5.5 mEq/L, Cl 78 mEq/L, CO_2 cont. 40 mEq/I pH 7.45, pCO_2 65, pO_2 43, O_2 sat. 82%.

While in the hospital he spontaneously diuresed, reducing his weight from 151 to 133 lbs. Blood chemistries were: Na 142, K 4.4, Cl 96, CO_2 cont. 24, pH 7.42, pCO_2 41, pO_2 71, O_2 sat. 92%.

Summary:

Severe metabolic alkalosis and edema secondary to alkali ingestion in a man with chronic renal disease. The metabolic alkalosis appeared to worsen the associated ventilatory defect, causing hypoxia and hypercapnea.

CASE 2: [redacted]

This 54 year old [redacted] woman was admitted to the hospital in a confused state, complaining of muscle cramps and weakness. She was a chronic alcoholic with vomiting, diarrhea and anorexia for 2-3 weeks. There was a questionable history that she had ingested baking soda.

Physical examination:

BP 100/80, P 80, R 20 (shallow)
Unkempt, malnourished, slightly disoriented woman with scleral icterus. Positive Trousseau sign, but negative Chvostek. Generalized weakness but no muscle paralysis. Hepatomegaly..

Laboratory data:

Blood: Na 142, K 1.0, Cl 64, CO_2 cont. 65, pH 7.57, pCO_2 62.
Urine: pH 6.5, Na 150 mEq/day, K 10 mEq/day, Cl 160 mEq/day

Course in hospital:

Initially given small amount of KCl (80 mEq), which raised serum K to 2.0 mEq/L. Supplemental KCl was then discontinued and she was given 4 L isotonic saline over 2-day period. Urinary excretion of Na and Cl increased, but there was no bicarbonate diuresis. Blood chemistries were Na 132, K 1.8, Cl 65, CO_2 cont. 56, pH 7.52.

She was then given supplemental KCl (120 mEq/day) for 7 days with complete correction of the metabolic alkalosis.

Summary:

Saline-resistant metabolic alkalosis secondary to severe potassium deficiency. The associated hypercapnea was probably due to weakness of respiratory muscles resulting from the severe hypokalemia.

CASE 3: [redacted]

This 40 year old man was admitted with typical primary aldosteronism. Hypertension was first discovered 3 months before admission. He had never had any definite episodes of tetany or muscle paralysis, but for the past 4 years he had repeated episodes of muscle cramps. For 8 years he had severe polyuria and polydipsia (estimated at 6-10 liters daily).

BP 155/100. Physical exam otherwise unremarkable. Blood chemistries: Na 145, K 2.0, Cl 92, CO_2 cont. 33, pH 7.46, pCO_2 45.

Urine: Na 96 mEq/day, K 52 mEq/day, pH 6.0
Urinary aldosterone 77 µgm/day (normal 4-22)
Plasma renin was low (82 ngm%) and did not increase with either low salt diet or diuril.

On [redacted] 1968 the right adrenal was removed and was found to contain a 1.5 cm adenoma.

Summary:

Classical primary aldosteronism with hypokalemia and metabolic alkalosis.

CASE 4: [redacted]

This 19 year old [redacted] girl developed intractable psychogenic vomiting following an illegitimate pregnancy in a younger sister. Her diet consisted almost entirely of celery and mayonaise (2 quarts daily). Her weight decreased from 120 to 75 lbs. On two occasions she was admitted to the hospital with thrombophlebitis following self-mutilation. She denied ingestion of alkali.

Laboratory data:

Na 137, K 2.3, Cl 80, CO_2 42, pH 7.58, pCO_2 43.
Urine: pH 8, Na 80 mEq/day, K 45 mEq/day, Cl 10 mEq/day
Urinary aldosterone - 50 μgm/24 hr.
Plasma renin - 342 ngm%.

Summary:

Metabolic alkalosis and hypokalemia secondary to psychogenic vomiting and probable alkali ingestion, and associated secondary hyperaldosteronism.

CASE 5: [redacted]

This 25 year old [redacted] man was admitted to [redacted] in the [redacted] of 1955 with paralytic poliomyelitis and was placed in a tank respirator. He was chronically overventilated and became severely hypokalemic and alkalotic. The alkalosis was a combined metabolic and respiratory alkalosis (pH 7.5-7.8, pCO_2 15-30, CO_2 cont. 35 mEq/L, serum K 1.8-3.0). It was not possible to correct the potassium deficiency despite the administration approximately 200 mEq/day - all of the administered potassium was excreted into the urine. When the respirator was correctly adjusted and the respiratory alkalosis reversed, the patient rapidly went into positive potassium balance, correcting both the hypokalemia and metabolic alkalosis.

Summary:

Chronic respiratory alkalosis leading to potassium deficiency and superimposed metabolic alkalosis.

CASE 6:

This 21 year old [redacted] man was admitted to the hospital with severe hyperventilation and tetany. Since returning from the military service 3 months previously he had difficulty finding work and had a tumultous marital life. During the previous one month he had 2-5 episodes of hyperventilation and carpo-pedal spasm daily. These episodes usually terminated spontaneously with the onset of laryngospasm.

On admission blood chemistries were:

Na 138, K 2.2, Cl 90, CO_2 cont. 36, pH 7.66, pCO_2 30.
Urine Na 95 mEq/24 hr., K 40 mEq/L

While in the hospital the patient had repeated episodes of severe hyperventilation and tetany. The administration of large doses of KCl (120-200 mEq daily) failed to correct the hypokalemia. He was transferred to the psychiatry ward and placed under light pentothal anesthesia to control his hyperventilation. KCl supplementation quickly corrected the hypokalemia at that time.

Summary:

Chronic respiratory alkalosis producing potassium deficiency and superimposed metabolic alkalosis.

CASE 7: [redacted]

This 57 year old man was admitted to the hospital with a diagnosis of chronic bronchitis and emphysema. He was a heavy smoker (3-4 pack/day) and had an 8 year history of chronic productive cough. For 6 years he had had increasingly severe dyspnea. Four days before admission he had sudden worsening of dyspnea. Prior to admission he was eating a regular diet and had taken no medications.

Physical exam:

BP 146/80, Pulse 90, Temp 98, R 24/min. No cyanosis. Chest was hyperresonant and had increased AP diameter. Breath sounds were distant and there were rhonchi in both bases. Heart was unremarkable. No cyanosis or edema.

Laboratory data:		Measured	Predicted
vitalometry:	FVC	1.8	4.05
	FEV 0.5	0.5	2.25
	FEV 1.0	0.63	3.1
	$(FEV_{0.5}/FVC)$ X100	26	50
	$(FEV_{1.0}/FVC)$ X100	35	75

Blood gases:

pH 7.47, pCO_2 47 mm Hg., CO_2 cont. 38 mEq/L, pO_2 46, O_2 sat. 83% , Na 133 mEq/L, K 3.7 mEq/L, Cl 87 mEq/L.

Summary:

Uncomplicated chronic obstructive bronchopulmonary disease associated with metabolic alkalosis.

CASE 8: [redacted]

This 79 year old [redacted] man has been followed in the [redacted] since 1962 with a diagnosis of chronic obstructive bronchopulmonary disease. When first seen blood chemistries were Na 141 mEq/L, K 4.6 mEq/L, Cl 89 mEq/L and CO_2 35 mEq/L. In the intervening years he received bronchodialators, digitalis, Naqua and KCl.

In [redacted] 1966 he developed acute bronchitis and was admitted to the hospital. Initial blood gas studies were: pH 7.61, pCO_2 37 mm Hg, pO_2 18 and O_2 sat. 41%. Repeat studies were pH 7.42, pCO_2 51, pO_2 48, O_2 sat. 83%. On 100% O_2 pH 7.46, pCO_2 46, pO_2 168, O_2 sat. 99%. While in hospital he received a 1 gram salt diet, digitoxin, Naqua, and KCl. At time of discharge pH 7.48, pCO_2 51, pO_2 52 and O_2 sat. 90%. Na 137 mEq/L, K 4.4, Cl 88, CO_2 37.

In [redacted] 1968 he developed a flu-like syndrome, pedal edema, and worsening of his dyspnea. On admission pH 7.42, pCO_2 50, pO_2 56, O_2 sat. 89%. Na 139, K 4.1, Cl 96, CO_2 37.

Summary:

Chronic obstructive bronchopulmonary disease associated with chronic metabolic alkalosis. Cor pulmonale with salt retention stimulus, low salt diet plus diuretics may have contributed to maintenance of the alkalosis. On one occasion the high blood pH was clearly due to relative overventilation.

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