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Emergency Approach to Patients with Acid-Base, Fluid, and Electrolyte Disorders

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Serious electrolyte and acid-base problems are frequently encountered in the critically ill patients in the emergency room as well as in the general wards, and have siginificant potential for morbidity and mortality. Therefore, the followings in this paper were intended to help the physician deal with a fluid, electrolyte, or acid-base emergency. Only the important concepts including definition, etiology, signs and symptoms, diagnosis, therapy, and several leading questions asked often by the physician are provided. The details in this field may not be covered enough in this paper. Therefore, for further informations, the general textbooks should be considered as the references.

METABOLIC ACIDOSIS

1) Definition

pH<7.35, pCO_2<40, HCO_3^-<24.

Metabolic acidosis may be acute (e.g., organic acidosis and/or diabetic ketoacidosis) or chronic (e.g., inorganic acidosis such as chronic renal failure) and results from the retension of fixed acid or the loss of alkali.

2) Etiology

a. Increased anion gap; $Na^+-(Cl^-+HCO_3^-)>12\pm2$ mEq/L
   1) Ketoacidosis – diabetic ketoacidosis, alcoholic ketoacidosis, starvation ketoacidosis
   2) Renal failure – ARF or CRF
   3) Poisons: aspirin, ethylene glycol, methyl alcohol, paraldehyde
   4) Lactic acidosis in shock, or in severe tissue hypoxia

b. Normal anion gap; $Na^+-(Cl^-+HCO_3^-)<12\pm2$ mEq/L
   1) Severe diarrhea – GI HCO_3^- loss, decreased urinary anion gap (i.e., $Na^++K^+-Cl^-$), or increased urinary NH_4 excretion
   2) Renal tubular acidosis – Type 1, 2 and 4
   3) Carbonic anhydrase inhibitor

3) Signs and Symptoms

There are no specific signs or symptoms—shock, tachypnea, fever, and sepsis frequently accompany metabolic acidosis.

a. Arterial blood: decreased pH, decreased pCO_2, decreased HCO_3^-
b. Electrolytes: required to calculate anion gap
c. BUN and creatinine: elevated in volume depletion, especially in the ratio of BUN/creatinine, and renal failure
d. Blood glucose: may indicate diabetes
e. Toxicology screen and solvent screen, as well as lactate and alcohol level determinations should be considered.

4) Diagnosis

1) Diagnosis is based on determination of pH, pCO_2, and HCO_3^-
2) Most patients seen in the emergency room with severe metabolic acidosis have increased anion gap acidosis. By obtaining determinations of pH, pCO_2, electrolytes, glucose, and BUN as

well as a brief histiory, the diagnosis can usually be made quickly.

5) Thrapy

1) If pH<7.1~7.2 and HCO_3^-<8~10 mEq/L, then $NaHCO_3$ is indicated: (desired HCO_3^- −observed HCO_3^-) (0.4) (weight in kg)=mEq HCO_3^- needed.
   Give one-third of calculated dose IV; then recheck blood gases and electrolytes.
2) If pH>7.1~7.2 and HCO_3^->8~10, $NaHCO_3$ is probably not indicated−concentrate on correcting the underlying cause of acidosis.

6) Questions Asked Often in Metabolic Acidosis

1) Is the respiratiory response normal in the analysis of arterial blood gases?
2) Is there a reason for the plasma anion gap to be lower than normal?
3) When should $NaHCO_3$ therapy be started?
4) Is the therapy of $NaHCO_3$ in the lactic acidosis beneficial?

7) References

1) DiNubile MJ. The increment in the anion gap. Overextension of a concept. Lancet 1988; 11: 951.
2) Kassirer JP. Serious acid-base disorders. N EnglJ Med 1974; 291:773.
3) Oh MS, Carroll HJ. The anion gap. N Engl J Med 1977; 297:814.

METABOLIC ALKALOSIS

1) Definition

pH>7.45, pCO_2>40, HCO_3^->28

Metabolic alkalosis is usually a chronic disorder. An exception is overzealous administration of HCO_3^- for treatment of acidosis due to cardiac arrest. Whenever metabolic alkalosis is noted in the analysis of arterial blood gases, the initiation factors for a loss of fixed acid or a gain of alkali as well as the maintenance factors such as volume contraction or reduced GFR should be investigated.

2) Etiology

a. ECF volume contraction
   1) Nasogastric suction and vomiting
   2) Diuretics
b. Mineralocorticoid excess syndrome—primary hyperaldosteronism
c. Excessive alkali administration
d. Post hypercapnic alkalosis
e. Potassium depletion − extremely rare as a single cause

3) Signs and Symptoms

Often not helpful

a. Arterial blood: increase pH, increased HCO_3^-, increased pCO_2
b. Electrolytes: decreased K, increased HCO_3^-. Urine chloride level is low (<20 mEq/L). Exceptions are Bartter's syndrome or primary hyperaldosteronism.

4) Diagnosis

based on determination of pH, pCO_2, and HCO_3^- with history of medication, vomiting, recent weight loss (ECF depletion)

5) Therapy

Correct ECF volume depletion with normal saline IV as well as the correction of electrolyte imbalance, especially hypokalemia with KCl (Caution: 5 mEq of KCl IV push may be lethal!). Exception is mineralocorticoid excess, which should be suspected if patient is cushingoid or taking steroid drugs.

6) Questions Asked Often in Metabolic Alkalosis

1) What are the key points to evaluate the ECF volume status wheather contracted or not in the presence of metabolic alkalosis?
2) What tests should be done if the ECF volume is not contracted?

7) References

1) Harrington JT. Metabolic alkalosis. Kidney Int 1984; 26:88.

2) Seldin DW, Rector FC, Jr. Symposium on acid-base homeostasis: the generation and maintenance of metabolic alkalosis. Kiney Int 1972; 1: 306.

RESPIRATORY ACIDOSIS

1) Definition

$pH < 7.35$, $pCO_2 > 40$.

Respiratory acidosis, which may be acute or chronic, is invariably due to hypoventilation.

2) Etiology

a. Pulmonary disease: e.g., COPD, severe asthma
b. Central hypoventilation, drug intoxication.
c. Mechanical
   1) Obesity
   2) Foreign body obstructing major airway
   3) Respiratory muscular failure, muscular dystrophy, hypokalemia

3) Signs and Symptoms

Dyspnea (sometimes not present), drowsiness, confusion, tremor, coma. Syptoms depend upon rate of change as well as upon absolute value of pCO_2.

a. Arterial blood: decreased pH, increased pCO_2.
b. Electrolytes: HCO_3^- (normal if acute, elevated if chronic)

4) Diagnosis

based on determination of pH, pCO_2, HCO_3^-.

5) Therapy

Identify and treat underlying etiology; in general, respiratory measures are sufficient to correct respiratory acidosis.

6) Questions Asked Often in Respiratory Acidosis

Is that justified to try high oxygen concentration in the treatment of hypoxia and respiratory acidosis in COPD patients?

7) References

1) Robin ED. Abnormalities of acid-base regulation in chronic pulmonary disease, with special reference to hypercapnea and extracellular alkalosis. N Engl J Med 1963; 268:917.

RESPIRATORY ALKALOSIS

1) Definition

$pH > 7.45$, $pCO_2 < 40$.

Respiratory alklosis, which may be acute or chronic, is invariably due to hyperventilation.

2) Etiology

a. Psychogenic hyperventilation – the most common cause of respiratory alkalosis seen in an emergency room.
b. Hyperventilation as a result of hypoxia (pulmonary embolus, alveolar capillary block, CHF)
c. CNS hyperventilation (salicylate ingestion, stroke)
d. Miscellaneous: exercise, beriberi, fever, thyrotoxicosis, hepatic coma, delirium tremens.

3) Signs and Symptoms

a. May be asymptomatic
b. Paresthesias, lightheadedness, and carpal pedal spasm may be associated with hyperventilation.
c. Arterial blood: increased pH, decreased pCO_2.
d. Electrolytes: HCO_3^- (normal if acute, decreased if chronic)

4) Diagnosis

based on determination of pH, pCO_2, HCO_3^-

5) Therapy

Identify and treat etiology; in general, respiratory measures are sufficient if hypoxia is the cause.

Psychogenic hyperventilation can be treated with paper bag rebreathing or sedative medication (diazepam 5~10 mg IM or orally).

6) Questions Asked Often in Respiratory Alkalosis

What is the pathogenetic mechanism of paresthesia or carpopedal spasm in acute respiratory alkalosis?

7) Reference

1) Krapf R, Beeler I, Hertner D, Hulter HN. Chronic respiratory alkalosis. N Eng J Med 1991; 324:1394.

MIXED ACID-BASE DISORDERS

The term mixed acid-base disorders refers to a clinical condition in which two or more primary acid-base disorders coexist. The general pattern of presentation of mixed acid-base disturbances is that of one obvious disturbance with what appears to be an inappropriate (excessive or inadequate) degree of compensation. The "inappropriateness" of the compensatory process is actually the effect of a separate primary disorder. The appropriate degrees of compensation for primary acid-base disorders have been determined by analysis of data from large numbers of patients, and are expressed in the form of equations as in following Table 1.

HYPERNATREMIA

1) Definition

Serum sodium>145 mEq/L. Hypernatremia reflects free water depletion only (rarely sodium gain).

2) Etiology

a. Excessive water loss
   1) Central (pituitary) or peripheral (nephrogenic) deabetes insipidus
   2) Osmotic diuretics
   3) Hypertonic tube feeding

b. Decreased water intake: almost always seen in comatose or semicomatose patients who are unable to respond to thirst stimulus.

3) Signs and Symptoms

a. Cardinal symtoms is thirst. If thirst is absent, basis is CNS lesion present with hypernatremia.

b. Other symtoms: lethargy, fatigue, somnolence, irritability related to brain cell shrinkage.

c. Laboratory studies: Na>145 mEq/L

4) Dianosis

based on elevated sodium level

5) Therapy

1) If salt and water deficit are of such magnitude that there is circulatory impairment, isotonic saline should be administered until the hemodynamics are corrected.

2) If hypernatremia is moderate (<160~165 mEq/L) and patient is conscious, he or she can usually respond to thirst sensation by drinking H_2O via oral route.

If hypernatremia is severe, or patient is comatose, calculate free water deficit and replace

Table 1. Patterns of Acid-Base Compensation

Type of Disorder	Degree of compensation	Duration required for compensation
Metabolic acidosis	$pCO_2 = HCO_3^- \times 1.2 \pm 2$	12~24 hrs
Metabolic alkalosis	$pCO_2 = HCO_3^- \times 0.7 \pm 0.5$	12~24 hrs
Acute respiratory acidosis	$HCO_3^- = pCO_2 \times 0.07 \pm 1.5$	Within minutes
Chronic respiratory acidosis	$HCO_3^- = pCO_2 \times 0.4 \pm 3$	3~ 5 days
Acute respiratory alkalosis	$HCO_3^- = pCO_2 \times 0.2 \pm 2.5$	Within minutes
Chronic respiratory alkalosis	$HCO_3^- = pCO_2 \times 0.5 \pm 2.5$	2~3 days

slowly as below.

Normal volume TBW (L)=0.5×normal body weight (kg)

Normal serum (Na)×TBW/measured serum (Na)=current TBW

Body water deficit=normal TBW-current TBW

The plan would be to give half the calculated dose of salt-free solution over the first 12 to 24hrs and reassess.

3) Dangers: too-rapid correction produces CNS damage.

6) Questions Asked Often in Hypernatremia

1) Is hypernatremia due to Na gain?
2) Is the administration of isotonic saline for the treatment of severe hypernatremia indicated?

7) References

1) Rose BD. New approach to disturbances in the plasma sodium concentration. Am J Med 1986; 81:1033.

HYPONATREMIA

1) Definition

Serum sodium<135 mEq/L

2) Etiology

a. With ECF volume depletion – vomiting, diarrhea
b. With ECF volume excess – CHF, liver failure or renal failure (with low effective arterial volume, and kidneys respond by reabsorbing salt and H_2O).
c. With normal ECF volume status – SIADH due to CNS disorders, pulmonary lesions, and multiple other etiologies.

3) Signs and Symptoms

depends on the status of ECF volume

Laboratory data – spot urine sodium<10~30 mEq/L in hypovolemia and ECF volume excess with low EAV.

– spot urine sodium>20~30 mEq/L in SIADH and diuretic trials in ECF volume excess.

4) Diagnosis

based on decreased sodium level, but exclude out the presence of hyperglycemia or pseudo-hyponatremia such as hyperlipidemia related low serum sodium concentration.

5) Therapy

1) Stop or restrict PO water intake
2) NaCl if ECF volume-depleted
3) Hypertonic saline may be necessary for patients with very low Na (<115 mEq/L) who are semicomatose or convulsing.

mEq Na needed=(desired Na—observed Na) (0.6) (weight in kg)

4) Danger: rapid correction may induce neurological catastrophe such as central pontine myelinosis. Therefore, slow correction of sodium less than 0.75~1.0 mEq/hr is more acceptable

6) Questions Asked Often in Hyponatremia

Is rapid or slow correction of hyponatremia more hazardous?

7) References

1) Anderson RJ. Hospital-associated hyponatremia. Kidney Int 1986; 29:1237.
2) Schrier RW. Treatment of hyponatremia. N Engl J Med 1985; 312:1121.
3) Laureno R, Karp BI. Pontine and extrapontine myelinosis following rapid correction of hyponatremia. Lancet 1988; 1:439.

HYPERKALEMIA

1) Definition

Serum K>5.5 mEq/L

2) Etiology

a. K shift from ICF—acidosis

b. Low renal K excretion

1) Reduced nephron mass with GFR<10~20 ml/min

2) Decreased Na delivery – CHF

3) Decreased mineralocorticoid bioactivity – hyporeninemic hypoaldosteronism

4) Excessive K load – accidental or intentional ingestion of excessive K, rhabdomyolysis, burns, tumor lysis syndrome

3) Signs and Symptoms

a. Cardiac arrhythmia

b. Arterial pH: acidosis often accompanies hyperkalemia.

c. EKG: peaked T wave, prolonged P-R, widened QRS, and ventricular fibrillation depending on the degree of hyperkalemia

4) Diagnosis

based on elevated serum K level in the absence of pseudohyperkalemia due to thrombocytosis or leukocytosis or hemolysis.

5) Therapy

1) In severe hyperkalemia, calcium gluconate 1 ampule IV push, 1-2 min—immediate action to block cardiotoxic effect on heart

2) $NaHCO_3$ 1-2 ampule IV push to correct acidosis and drive K into cells

3) Glucose and insulin—500 ml of 10% glucose with 10 units regular insulin. This also drives K into cells—lasts as long as infusion runs

4) Kayexelate (Na: K exchange resin): rectal 50~100 g kayexelate+50~100 g sorbitol in 250 ml H_2O. Retain 45~60 min, evacuate, and repeat if necessary. Do not use oral kayexelate in emergency situations—it takes too long to work.

5) Dialysis, preferably hemodialysis, should be performed if hyperkalemia cannot be controlled by the methods outlined.

6) Questions Asked Often in Hyperkalemia

1) How do you exclude out the possibility of pseudohyperkalemia?

2) Is hyperkalemia due to high K intake alone?

3) When is hyperkalemia an emergency?

7) References

1) Williams ME, Rosa RM, Epstein FH. Hyperkalemia. Adv Intern Med 1986; 31:265.

HYPOKALEMIA

1) Definition

Serum K<3.5 mEq/L

2) Etiology

a. Shifting of K into cells – alkalosis

b. Nonrenal K loss – gastrointestinal loss such as diarrhea, vomiting, fistula

c. Renal K loss – RTA, diuretics, primary hyperaldosteronism. Bartter's syndrome

d. Inadequate intake of K

3) Signs and Symptoms

a. Neuromuscular – weakness, hyporeflexia, paralysis (K usually<2 mEq/L)

b. Cardiovascular; increased sensitivity to digitalis, arrhythmias

c. EKG changes; flat T and U waves, and depressed S-T segments

d. Laboratory findings – spot urine K<20 mEq/d in extra-renal K loss, and>20 mEq/d in renal K loss

4) Diagnosis

based upon decreased K level

5) Therapy

1) Hypokalemia is generally not an emergency, and oral therapy is usually adequate.

2) In severe hypokalemia, i.e., <2.0 mEq/L with arrhythmia or paralysis, one can give 20~50

mEq/h, but dilute in as much as possible. Monitor EKG and reassess serum K level frequently. Never give KCl undiluted, as IV-push fatalities have been reported.

6) Questions Asked Often in Hypokalemia

How does renin and aldosterone profile in the presence of both hypokalemia and hypertension help for the differential diagnosis?

7) References

1) Lowenstein J. Hypokalemia and hyperkalemia. Med Clin North Am 1973; 57:1535.