

METABOLIC ALKALOSIS (PRIMARY BASE BICARBONATE EXCESS)

Metabolic alkalosis is characterized by a high pH (loss of hydrogen ions) and high plasma bicarbonate caused by excessive intake of sodium bicarbonate, loss of gastric/intestinal acid, renal excretion of hydrogen and chloride, prolonged hypercalcemia, hypokalemia, and hyperaldosteronism. Compensatory mechanisms include slow, shallow respirations to increase CO₂ level and an increase of bicarbonate excretion and hydrogen reabsorption by the kidneys.

CARE SETTING

This condition does not occur in isolation but rather is a complication of a broader problem that may require inpatient care in a medical-surgical or subacute unit.

RELATED CONCERNS

Plans of care specific to predisposing factors

Fluid and electrolyte imbalances

Renal dialysis

Respiratory acidosis (primary carbonic acid excess)

Respiratory alkalosis (primary carbonic acid deficit)

Patient Assessment Database (Dependent on Underlying Cause)

CIRCULATION

May exhibit: Tachycardia, irregularities/dysrhythmias
Hypotension
Cyanosis

ELIMINATION

May report: Diarrhea (with high chloride content)
Use of potassium-losing diuretics (Diuril, Hygroton, Lasix, Edecrin)
Laxative abuse

FOOD/FLUID

May report: Anorexia, nausea/prolonged vomiting
High salt intake; excessive ingestion of licorice
Recurrent indigestion/heartburn with frequent use of antacids/baking soda

NEUROSENSORY

May report: Tingling of fingers and toes; circumoral paresthesia
Muscle twitching, weakness
Dizziness

May exhibit: Hypertonicity of muscles, tetany, tremors, convulsions, loss of reflexes
Confusion, irritability, restlessness, belligerence, apathy, coma
Picking at bedclothes

SAFETY

May report: Recent blood transfusions (citratd blood)

RESPIRATION

May exhibit: Hypoventilation (increases PCO₂ and conserves carbonic acid), periods of apnea

TEACHING/LEARNING

History of Cushing's syndrome; corticosteroid therapy
Discharge plan considerations: **DRG projected mean length of inpatient stay depends on underlying cause**
May require change in therapy for underlying disease process/condition.
Refer to section at end of plan for postdischarge considerations.

DIAGNOSTIC STUDIES

Arterial pH: Increased, higher than 7.45.

Bicarbonate (HCO₃): Increased, higher than 26 mEq/L (primary).

Paco₂: Slightly increased, higher than 45 mm Hg (compensatory).

Base excess: Increased.

Serum chloride: Decreased, less than 98 mEq/L, disproportionately to serum sodium decreases (if alkalosis is hypochloremia).

Serum potassium: Decreased.

Serum calcium: Usually decreased. Prolonged hypercalcemia (nonparathyroid) may be a predisposing factor.

Urine pH: Increased, higher than 7.0.

Urine chloride: Less than 10 mEq/L suggests chloride-responsive alkalosis, whereas levels higher than 20 mEq/L suggest chloride resistance.

ECG: May show hypokalemic changes including peaked P waves, flat T waves, depressed ST segment, low T wave merging to P wave, and elevated U waves.

NURSING PRIORITIES

1. Achieve homeostasis.
2. Prevent/minimize complications.
3. Provide information about condition/prognosis and treatment needs as appropriate.

DISCHARGE GOALS

1. Physiological balance restored.
2. Free of complications.
3. Condition, prognosis, and treatment needs understood.
4. Plan in place to meet needs after discharge.

Because no current nursing diagnosis speaks clearly to metabolic imbalances, the following interventions are presented in a general format for inclusion in the primary plan of care.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Electrolyte & Acid/Base Balance (NOC)

Display serum bicarbonate and electrolytes WNL.

Be free of symptoms of imbalance, e.g., absence of neurological impairment/irritability.

ACTIONS/INTERVENTIONS	RATIONALE
Metabolic Alkalosis Management (NIC) Independent Monitor respiratory rate, rhythm, and depth. Assess level of consciousness and neuromuscular status, e.g., strength, tone, movement; note presence of Chvostek's/Trousseau's signs. Monitor heart rate/rhythm.	Hypoventilation is a compensatory mechanism to conserve carbonic acid and represents definite risks to the individual, e.g., hypoxemia and respiratory failure. The CNS may be hyperirritable (increased pH of CNS fluid), resulting in tingling, numbness, dizziness, restlessness, or apathy and confusion. Hypocalcemia may contribute to tetany (although occurrence is rare). Atrial/ventricular ectopic beats and tachydysrhythmias may develop.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Metabolic Alkalosis Management (NIC)</p> <p>Independent</p> <p>Record amount and source of output. Monitor intake and daily weight.</p> <p>Restrict oral intake and reduce noxious environmental stimuli; use intermittent/low suction during NG suctioning; irrigate gastric tube with isotonic solutions rather than water.</p> <p>Provide seizures/safety precautions as indicated, e.g., padded side rails, airway protection, bed in low position, frequent observation.</p> <p>Encourage intake of foods and fluids high in potassium and possibly calcium (dependent on blood level), e.g., canned grapefruit and apple juices, bananas, cauliflower, dried peaches, figs, and wheat germ.</p> <p>Review medication regimen for use of diuretics, such as thiazides (Diuril, Hygroton), furosemide (Lasix), and ethacrynic acid (Edecrin).</p> <p>Instruct patient to avoid use of excessive amounts of sodium bicarbonate.</p>	<p>Helpful in identifying source of ion loss; e.g., potassium and HCl are lost in vomiting and GI suctioning.</p> <p>Limits gastric losses of HCl, potassium, and calcium.</p> <p>Changes in mentation and CNS/neuromuscular hyperirritability may result in patient harm, especially if tetany/convulsions occur.</p> <p>Useful in replacing potassium losses when oral intake permitted.</p> <p>Discontinuation of these potassium-losing drugs may prevent recurrence of imbalance.</p> <p>Ulcer patients can cause alkalosis by taking baking soda and milk of magnesia in addition to prescribed alkaline antacids.</p>
<p>Collaborative</p> <p>Assist with identification/treatment of underlying disorder.</p> <p>Monitor laboratory studies as indicated, e.g., ABGs/pH, serum electrolytes (especially potassium), and BUN.</p> <p>Administer medications as indicated, e.g.;</p> <ul style="list-style-type: none"> Sodium chloride PO/Ringer's solution IV unless contraindicated; Potassium chloride; Ammonium chloride or arginine hydrochloride; 	<p>Addressing the primary condition (e.g., prolonged vomiting/diarrhea, hyperaldosteronism, Cushing's syndrome) promotes correction of the acid-base disorder.</p> <p>Evaluates therapy needs/effectiveness and monitors renal function.</p> <p>Correcting sodium, water, and chloride defects may be all that is needed to permit kidneys to excrete bicarbonate and correct alkalosis, but must be used with caution in patients with HF or renal insufficiency.</p> <p>Hypokalemia is frequently present. Chloride is needed so kidney can absorb sodium with chloride, enhancing excretion of bicarbonate.</p> <p>Although used only in severe cases, ammonium chloride may be given to increase amount of circulating hydrogen ions. Monitor administration closely to prevent too rapid a decrease in pH, hemolysis of RBCs. <i>Note:</i> May cause rebound metabolic acidosis and is usually contraindicated in patients with renal/hepatic failure.</p>

ACTIONS/INTERVENTIONS	RATIONALE
Metabolic Alkalosis Management (NIC)	
Collaborative	
Acetazolamide (Diamox);	A carbonic anhydrase inhibitor that increases renal excretion of bicarbonate.
Spironolactone (Aldactone).	Effective in treating chloride-resistant alkalosis, e.g., Cushing's syndrome.
Avoid/limit use of sedatives or hypnotics.	If respirations are depressed, may cause hypoxia/respiratory failure.
Encourage fluids IV/PO.	Replaces extracellular fluid losses, and adequate hydration facilitates removal of pulmonary secretions to improve ventilation.
Administer supplemental O ₂ as indicated and respiratory treatments to improve ventilation.	Respiratory compensation for metabolic alkalosis is hypoventilation, which may cause decreased PaO ₂ levels/hypoxia.
Prepare patient for/assist with dialysis as needed.	Useful when renal dysfunction prevents clearance of bicarbonate.

POTENTIAL CONSIDERATIONS: Refer to Potential Considerations relative to underlying cause of acid/base disorder.