ACIDOSIS, LACTIC

Key Features

ESSENTIALS OF DIAGNOSIS

- Severe acidosis with hyperventilation
- Blood pH below 7.30
- Serum bicarbonate < 15 mEq/L
- Anion gap > 15 mEq/L
- Absent serum ketones
- Serum lactate > 5 mmol/L

GENERAL CONSIDERATIONS

- Characterized by overproduction of lactic acid (tissue hypoxia), deficient removal (hepatic failure), or both (circulatory collapse)
- Occurs often in severely ill patients suffering from
  - Cardiac decompensation
  - Respiratory or hepatic failure
  - Septicemia
  - Infarction of bowel or extremities
- With the discontinuance of phenformin therapy in the United States, lactic acidosis in diabetics is uncommon but occasionally occurs with use of metformin. It must be considered in the acidotic diabetic, especially if the patient is seriously ill

ETIOLOGY

- Tissue hypoxia, eg, cardiogenic, septic, or hemorrhagic shock; seizure; carbon monoxide or cyanide poisoning
- Hepatic failure
- Ischemic bowel
- Infarction of extremities
- Diabetes, especially with metformin use
- Ketoacidosis
Clinical Findings

SYMPTOMS AND SIGNS

- Main clinical feature is marked hyperventilation

- When lactic acidosis is secondary to tissue hypoxia or vascular collapse, the clinical presentation is variable, being that of the prevailing catastrophic illness

- In idiopathic, or spontaneous, lactic acidosis
   - Onset is rapid (usually over a few hours)
   - Blood pressure is normal
   - Peripheral circulation is good
   - No cyanosis

DIFFERENTIAL DIAGNOSIS

Other causes of metabolic acidosis

- Diabetic ketoacidosis
- Starvation ketoacidosis
- Alcoholic ketoacidosis
- Renal failure (acute or chronic)
- Ethylene glycol toxicity
- Methanol toxicity
- Salicylate toxicity
- Other: paraldehyde, metformin, isoniazid, iron, rhabdomyolysis

Diagnosis

LABORATORY TESTS

- High anion gap (serum sodium minus the sum of chloride and bicarbonate anions [in mEq/L] should be no greater than 15). A higher value indicates the existence of an abnormal compartment of anions
Plasma bicarbonate and blood pH are quite low, indicating the presence of severe metabolic acidosis.

Ketones are usually absent from plasma and urine, or at least not prominent.

In the absence of azotemia, hyperphosphatemia occurs in lactic acidosis for reasons that are not clear.

The diagnosis is confirmed by demonstrating, in a sample of blood that is promptly chilled and separated, a plasma lactic acid concentration of 5 mmol/L or higher (values as high as 30 mmol/L have been reported).

Normal plasma values average 1 mmol/L, with a normal lactate–pyruvate ratio of 10:1. This ratio is greatly exceeded in lactic acidosis.

Treatment

Empiric antibiotic coverage for sepsis should be given after culture samples are obtained if the cause of lactic acidosis is unknown.

Alkalinization with IV sodium bicarbonate to keep the pH above 7.2 in the emergency treatment of lactic acidosis is controversial; as much as 2000 mEq in 24 h has been used. However, there is no evidence that the mortality rate is favorably affected by administering bicarbonate.

THERAPEUTIC PROCEDURES

Aggressive treatment of the precipitating cause is the main component of therapy, such as ensuring adequate oxygenation and vascular perfusion of tissues.

Hemodialysis may be useful when large sodium loads are poorly tolerated.

Outcome

PROGNOSIS

Mortality rate of spontaneous lactic acidosis is high.

Early and aggressive treatment of metformin-induced lactic acidosis with hemofiltration improves outcome.

Prognosis in most cases is that of the primary disorder that produced the lactic acidosis.

WHEN TO ADMIT

All patients because of the high mortality rate.

Evidence

PRACTICE GUIDELINES

National Guideline Clearinghouse: Surviving Sepsis Campaign Guidelines for Management of Severe Sepsis and Septic Shock

REFERENCES


Content adapted from CURRENT Medical Diagnosis & Treatment 2008 and CURRENT Consult Medicine 2007.