Severe perioperative lactic acidosis: How clinically significant is it?

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■ **BACKGROUND** Lactic acidosis, generally defined as a plasma lactate concentration in excess of 5 mmol/L with a concomitant blood pH less than 7.25, is reported to have a direct association with mortality.

■ **OBJECTIVE** To report a case of unexplained perioperative lactic acidosis and to discuss the etiology, recognition, treatment, and importance of a transient rise in plasma lactate concentration.

■ **SUMMARY** Severe lactic acidosis developed in a 40-year-old man with Crohn's disease during major abdominal surgery. The plasma lactate concentration reached 16.9 mmol/L (normal range 1.5 to 2.2 mmol/L). This condition resolved within 14 hours without harm to the patient.

■ **CONCLUSIONS** When lactate accumulates in the perioperative period, the responsible condition is most often self-limiting. Reversible, subacute, marked lactic acidosis should not be assumed to predict mortality as it does in patients whose plasma lactate concentrations remain chronically elevated during severe systemic diseases such as sepsis.

■ INDEX TERMS: ACIDOSIS, LACTIC; INTRAOPERATIVE COMPLICATIONS; PARENTERAL NUTRITION, TOTAL
■ CLEVE CLIN J MED 1994; 61:314–316

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LACTIC ACIDOSIS, even when severe, does not always lead to death. We report a case of severe but self-limiting lactic acidosis that developed during abdominal surgery in which the patient survived.

CASE HISTORY

The patient, a 40-year-old man, had been found to have Crohn's disease in 1974. He had subsequently undergone gastrojejunostomy, and a revision of the gastrojejunostomy with a splenectomy was done in 1982. In early 1987 he had undergone drainage of a hepatic abscess, a jejunal resection, and a Roux-en-Y gastrojejunostomy. He had received total parenteral nutrition for the past 5 years, and this was continued though the present surgery, which was planned as an exploratory laparotomy for repair of a gastrocolic fistula.

Upon admission, the patient weighed 71 kg and was 193 cm in height. His heart rate was 96 per minute, blood pressure 130/70 mm Hg, respiratory rate 14 per minute, and oral temperature 36°C. A Hickman catheter was in place in the left subclavian vein, and the

TABLE
HOURLY DATA OBTAINED DURING SURGERY

Hour	pH	$PaCO_2$ (mm Hg)	PaO_2 (mm Hg)	Bicarbonate (mmol/L)	Base deficit (mmol/L)	Hematocrit (%)	Lactic acid (mmol/L)	Lowest arterial pressure (mm Hg)	Total estimated blood loss (mL)	Total urinary output (mL)
0	7.39	36	242	22	2	38	—	95/60	0	100
1	—	—	—	—	—	—	—	130/70	400	165
2	7.33	35	267	19	6	36	—	120/60	700	190
3	7.27	36	165	16	9	28	7.3	120/70	900	235
4	7.31	37	156	19	6	31	8.6	120/70	1000	270
5	7.32	33	161	17	7	25	10.8	120/70	1200	330
6	7.25	35	171	15	10	24	12.3	110/60	1400	500
7	7.27	36	137	17	9	28	13.4	110/70	1500	720
8	7.32	35	159	18	7	24	14.6	110/70	1800	820
9	—	—	—	—	—	—	—	120/80	1800	870

central venous pressure was 8 cm H_20. Physical examination revealed no signs of pulmonary disease or cardiac failure. Laboratory studies revealed abnormal serum concentrations of lactate dehydrogenase at 195 IU/L (normal range 100 to 185 IU/L), aspartate aminotransferase at 104 IU/L (7 to 40 IU/L), alkaline phosphatase at 750 IU/L (20 to 110 IU/L), and platelets at 750×10^9/L (150 to 400×10^9/L). The anion gap was 4.9 mEq/L.

Before surgery the patient received midazolam 4 mg and morphine 7 mg intramuscularly. Another 4 mg of midazolam was given intravenously (IV). We gave tubocurarine 3 mg IV and then induced anesthesia with thiopental 300 mg and fentanyl 10 μg/kg; succinylcholine 100 mg IV was given to facilitate endotracheal intubation. We maintained anesthesia with nitrous oxide and oxygen in a ratio of 60:40, enflurane (1% to 2% inspired concentration), and pancuronium for neuromuscular blockade. The fractional concentration of inspired oxygen was kept in the range 35% to 40%.

The *Table* presents data obtained at hourly intervals after induction of anesthesia. Three hours into the surgery, an arterial blood-gas measurement revealed a base deficit of 9 mmol/L, and the directly measured plasma lactate concentration was 7.3 mmol/L. The lactate concentration progressively rose to 14.6 mmol/L intraoperatively. We gave a total of 350 mEq of sodium bicarbonate IV in divided doses to treat the acidosis.

The patient's cardiovascular status remained stable; there were no periods of hypotension, and we observed no obvious evidence of sepsis. We monitored the esophageal temperature throughout the operation, and the maximum decrease was 1.2°C. The patient underwent an exploratory laparotomy, extensive lysis of adhesions, resection of gastrocolic and biliary colic fistulas, and a transverse colectomy.

After the endotracheal tube was removed, the patient was transferred to the recovery room, where he remained overnight. His urine output was maintained at 0.5 to 1 mL/kg/hour, and the central venous pressure was kept at 8 to 12 cm H_2O. A postoperative arterial blood-gas measurement revealed a pH of 7.32, $PaCO_2$ 26 mm Hg, PaO_2 138 mm Hg, bicarbonate 14 mmol/L, and a base deficit of 11 mmol/L. The plasma lactate concentration reached a peak of 16.9 mmol/L. The base deficit disappeared and the plasma lactate concentration declined to 3.8 mmol/L by the next morning. No further episodes of acidosis were observed.

DISCUSSION

Lactic acidosis is generally defined as a plasma lactate concentration in excess of 5 mmol/L with a concomitant blood pH of less than 7.25.[1,2] The plasma anion gap is increased. An increased plasma lactate concentration can be due to increased production of lactate, decreased consumption, or both.

Our patient's preoperative elevations in lactate dehydrogenase, aspartate aminotransferase, and alkaline phosphatase concentrations can be attributed to the long-term administration of total parenteral

nutrition[3]; these measurements give evidence of impaired liver function, which may have impaired this patient's lactate metabolism. Long-term administration of total parenteral nutrition can also lead to electrolyte and acid-base disorders, but our pre-anesthetic measurements showed no acidosis or abnormal anion gap.

Transient episodes of focal low-flow states and hepatic hypoperfusion and the effects of anesthesia may have contributed to the increase in plasma lactate concentration, but global hypoxia did not occur. We therefore concluded that unidentified regional tissue ischemia (probably intestinal) caused type A lactic acidosis. The intestines were manipulated during the operation, and this could have contributed further to the lactic acidosis, as the intestines are a major producer of lactate.[4] The normal acid-base state 14 hours postoperatively was an important indicator that this patient's lactic acidosis was transient and reversible.

Elevated plasma lactate concentrations have been observed during abdominal surgery,[5] but their significance remains unknown. We presume that if lactate accumulates in the perioperative period, the responsible condition is most often self-limiting. Transient lactic acidosis needs to be clearly distinguished from the chronic states in which a direct association with mortality has been shown.[2,6,7] It is worth mentioning that the plasma lactate concentration can increase 7- to 10-fold before measurable acidosis occurs.[8]

In summary, severe perioperative lactic acidosis occurred during major intestinal surgery in a patient receiving long-term total parenteral nutrition. This marked increase in plasma lactate concentration gradually normalized within 14 hours and was not associated with morbidity or mortality for this patient. We feel that reversible, subacute, marked lactic acidosis should not be assumed to predict mortality as it does in patients whose lactate concentrations remain chronically elevated during severe systemic diseases such as sepsis.

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