Case Report

Hemodialysis for Lactic Acidosis

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Abstract

Lactic acidosis (Type A) is common in critically ill patients and usually treated by correcting the underlying etiology. We present the case of a young female who presented with life-threatening lactic acidosis secondary to hematological malignancy. Timely initiation of hemodialysis was lifesaving. The case highlights the importance of considering Type B lactic acidosis (in this case secondary to a hematological malignancy) and also initiating renal replacement therapy when routine measures are ineffective.

Keywords: Hematological malignancy, hemodialysis, hyperlactatemia, lactic acidosis, malignancy

Introduction

Lactic acidosis is very commonly encountered in the critical care units. Treatments are generally focused on improving oxygen delivery and restoring tissue perfusion. We present a patient with grossly elevated lactate levels associated with lymphoma which improved only after initiation of dialysis.

Case Report

A 21-year-old female patient was transferred from an outside hospital to our tertiary Critical Care Unit (CCU) with worsening dyspnea and unresponsiveness (Glasgow Coma Scale 3/15 on arrival) requiring intubation. The patient was noted to be in severe shock despite fluid resuscitation and vasopressor support. Initial arterial blood gas (ABG) analysis revealed severe metabolic acidosis (pH: 6.725, $PaCO_2$: 13.6 mmHg, HCO_3: 1.7 mmol/l), but the lactate level was too high to measure (using Rapidlab 1265-SIEMENS™). She was given intravenous sodium bicarbonate (bolus followed by infusion), and repeat ABG after an hour showed pH of 7.005 with bicarbonate of 4.6 mmol/L [Table 1]. Due to persistent metabolic acidosis with severe hyperlactatemia, renal replacement therapy was initiated. After initial dialysis, pH improved to 7.179 with a measurable ABG lactate of 21.72 mmol/L. Dialysis was continued and lactate got reduced to 13.98 mmol/L in <24 h. The patient also had hypoglycemia requiring dextrose infusion. Complete hemogram done showed pancytopenia, and ultrasound abdomen revealed enlarged kidneys which was similar to earlier computer tomography report. With the above features, Type B lactic acidosis, secondary to malignancy, was considered as a possible etiology, and ā hematologist was consulted and bone marrow biopsy was performed. Over the next 48 h, the patient's hemodynamic and clinical status improved and was extubated. Bone marrow biopsy revealed lymphoma.

Discussion

Lactic acidosis (Type A) is commonly encountered in CCU and is usually secondary to tissue hypoperfusion and reversed by correction of hemodynamic status. In the case illustrated, we encountered a patient with Type B lactic acidosis secondary to hematological malignancy. The cause of lactic acidosis in malignancy is unclear and is likely multifactorial. Otto Warburg showed that malignant cells have altered metabolism and have increased glycolysis and end up producing lactate which is known as "Warburg effect."[1] Increased glycolysis is thought to be secondary to overexpression or aberrant expression of glycolytic enzyme, hexokinase in the glycolytic pathway.[2] This may be due to insulin-like growth factor-binding protein participation in signaling the glycolytic pathway of malignant cells.[3] Lactate produced is converted into glucose in the liver and kidneys. Hence, impaired liver function is often implicated

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Table 1: Arterial blood gas report

Date	Day 1	Day 1	Day 1	Day 1	Day 1	Day 1	Day 2	Day 3	Day 4	Day 5
Time (h)	10:20	11:58	13:18	14:06	18:27	21:03	7:34	5:00	4:27	5:00
pH	6.725	6.603	7.005	6.906	7.236	7.179	7.324	7.516	7.492	7.462
$PaCO_2$ (mmHg)	13.6	24.8	19	24.6	18.2	14.9	18.5	32	30.3	30.9
PaO_2 (mmHg)	224	315.6	469	66.9	89.6	89.0	124.2	96.6	94.1	101
HCO_3 (mmHg)	1.7	2.4	4.6	4.8	7.6	5.4	9.4	25.3	22.7	21
Lactate (mmol/L)	N/R	N/R	N/R	N/R	21.62	21.72	13.98	5.87	6.34	6.54

N/R: Not readable

as another likely mechanism[4] for lactic acidosis. However, there have been reports of lactic acidosis in patients with normal liver function[5] suggesting other causes. The hyperactivity of malignant cells and local hypoxia is also been considered to be the reason for increased lactate in the absence of global hypoxia or hypoperfusion.[6] Thiamine deficiency can also cause hyperlactatemia.[7] Pyruvate dehydrogenase requires thiamine for conversion, and therefore, deficiency of thiamine may lead to accumulation of pyruvate which is converted into lactate. Embolization of malignant cells into the microvasculature can occur and may cause a state of hypoperfusion and increase anaerobic metabolism-producing lactate.[8]

Regardless of the etiology, patients with severe metabolic acidosis often receive intravenous bicarbonate as the first line of treatment. However, bicarbonate supplementation in patients with lactic acidosis is controversial.[9] However, alkali supplementation and even dialysis can be performed temporarily. Prikis *et al*.[10] had reported hemodialysis to be helpful in decreasing lactate concentration. Hemodialysis along with supportive care will help revive and stabilize the patient until definitive treatment is started.

As there are a few reports of malignancy causing hyperlactatemia and as it has been associated with a poor prognosis, it is considered an oncologic emergency.[11] Our case report highlights the importance of considering evaluation for malignancy and initiating early dialysis in patients with high lactate levels who do not respond to standard resuscitative measures.

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Conflicts of interest

There are no conflicts of interest.

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