
A Case of Severe Type B Lactic Acidosis in a Patient with Acute Lymphoblastic Leukemia

Author (Escientific Publishers)*

Suspended

... diseases (e.g., diabetes), drugs, toxins, inborn errors in metabolism, and hereditary/miscellaneous disorders. In comparison, type A

... without other evidence of tumor lysis syndrome. His overall clinical

Citation: Escientific Publishers. (2022). A Case of Severe Type B Lactic Acidosis in a Patient with Acute Lymphoblastic Leukemia. *Journal of Medical Research and Case Reports* 4(1).

presentation was felt to be most consistent with severe type B lactic acidosis portending a poor prognosis.

sepsis after administration of vincristine/prednisone and was noted to have changes in mental status characterized as increased

Suspended

months later, maintenance therapy (prednisone, mercaptopurine, methotrexate, and vincristine) was initiated but patient subsequently developed double vision thought to be secondary to 6th nerve palsy after about 3 months of treatment. Lumbar puncture confirmed recurrence of malignant cells in the cerebrospinal fluid despite treatment with intrathecal methotrexate prompting hydrocortisone to be added. Bone marrow biopsy done about 3 months later confirmed systemic relapse and cerebrospinal fluid showed persistence of malignant cells for which cranial radiation therapy was started. The patient was then hospitalized 2 months later with

Profound lactic acidosis with gradually increasing anion gap in correlation with increasing LDH was thought to be secondary to type B lactic acidosis related to progressive ALL although the pathophysiologic mechanism for this is unknown. It was felt that type A lactic acidosis was clinically unlikely as patient remained without abdominal pain, hematochezia, signs or symptoms of ischemic disease, and no acute elevation in lactate or acidemia. The gradual rise of lactate and compensated pH as seen in this patient is not typical for type A lactic acidosis. Drug-induced lactic acidosis was also considered as patient reported compliance with metformin 500 mg by mouth

daily although there was low suspicion that this may be the cause of lactic acidosis given stable renal function (glomerular filtration rate = 89). It was, however, discontinued at the time of discharge in case it was contributing. As G6PD deficiency can also elicit type

process or processes responsible for the development of severe lactic acidosis. [7]

Another proposed mechanism relates to tumor cell overexpression

Suspended

dehydrogenase, thiamine deficiency, and tumor lysis syndrome. [7] Liver and kidney dysfunction whether they be from tumor infiltration, ischemic damage, or other causes are thought to be two processes that contribute to the development of type B lactic acidosis as lactate is the end product of anaerobic metabolism and is converted to pyruvate and subsequently glucose by both the liver (90%) and the kidneys (10%) [9] via glycolysis. However, many patients with kidney and liver dysfunction do not develop severe lactic acidosis which suggests that there is likely a more complex

This phenomenon has been reported in patients on total parenteral nutrition without vitamin supplementation although some of these patients also had malignancy. The acidosis in this situation was successfully reversed via addition of thiamine to the alimentation solution. [16] Furthermore, administration of methotrexate in chemotherapy regimens can result in the development type B lactic acidosis because it competes with thiamine transport systems and results in inhibition of pyruvate dehydrogenase and initiation of

anaerobic metabolism as described above. [10 & 17] Nucleoside reverse transcriptase inhibitors (NRTIs) used in the treatment of HIV have also been associated with the development of type B lactic

Until chemotherapy takes effect to treat the underlying malignancy, both intravenous bicarbonate and hemodialysis have been used to control lactic acidosis although its use is controversial. Since

patients. It is the only treatment modality that has consistently led to remission. Thus, treatment of the primary condition (i.e., cancer-directed therapy for leukemia/lymphoma) remains the mainstay of therapy. [8] The resolution of lactic acidosis occurred in 6 out of 7 of the 29 cases that responded to chemotherapy when reviewed by Chan et al. The lactic acidosis resolved in 5 of these 6 cases within 15 hours to 3 days and in the remaining case resolution was noted weeks after chemotherapy was introduced but within 2 days after salvage chemotherapy was initiated. [2]

such extreme elevations in LDH and severe lactic acidosis. Rather, he was started on salvage chemotherapy (vincristine and prednisone) to treat the underlying cause which was felt to be progression of his ALL. However, this was stopped after one cycle and goals of care were shifted to comfort care within weeks after discharge due to patient's poor performance status.

Conclusion

Overall, type B lactic acidosis appears to be a rare but severe and often lethal complication of hematological malignancies. The exact pathophysiology that governs its development still remains unclear

9. Van der Beek A, de Meijer PHEM, Meinders AE. (2001). Lactic acidosis: pathophysiology, diagnosis and treatment. *Neth J Med* 58(3): 128-136.
10. Sillos EM, Shenep JL, Burghen GA, Pui CH, Behm FG, et. al.

Suspended

case of lactic acidosis caused by thiamine deficiency. *Korean J Intern Med* 73(4): 443-447.

7. Friedenber AS, Brandoff DE, Schiffman FJ. (2007). Type B lactic acidosis as a severe metabolic complication in lymphoma and leukemia: a case series from a single institution and literature review. *Medicine* 86(4): 225-232.
8. Lee HS, Kim HJ, Choi S, Kim CK, Lee NS, et. al. (2010). A case of type B lactic acidosis in acute leukemia. *Yonsei Med J* 51(3): 460-462.
- acidosis. *AIDS Patient Care STDS* 15(12): 611-614.
20. Monier PL, Wilcox R. (2004). Metabolic complications associated with the use of highly active antiretroviral therapy in HIV-1-infected adults. *Am J Med Sci* 328: 48-56.
21. Shaer A, Rastegar A. (2000). Lactic acidosis in the setting of antiretroviral therapy for acquired immunodeficiency syndrome. A case report and review of literature. *Am J Nephrol* 20(4): 332-338.

Citation: Ami K. Patel and Nancy Andrea. (2022). A Case of Severe Type B Lactic Acidosis in a Patient with Acute Lymphoblastic Leukemia. *Journal of Medical Research and Case Reports* 4(1).

22. Tiefenthaler M, Amberger A, Bacher N, Hartmann BL, Margreiter R, et. al. (2001). Increased lactate production follows loss of mitochondrial membrane potential during apoptosis of human leukaemia cells. *Br J Haematol* 114(3): 574-580.
23. Forsythe SM, Schmidt GA. (2000). Sodium bicarbonate for the treatment of lactic acidosis. *Chest* 117(1): 260-267.
24. Máttar JA, Weil MH, Shubin H, Stein L. (1974). Cardiac arrest in the critically ill: II. Hyperosmolal states following cardiac arrest. *Am J Med* 56(2): 162-168.
25. Bersin RM, Chatterjee K, Arieff AI. (1989). Metabolic and hemodynamic consequences of sodium bicarbonate administration in patients with heart disease. *Am J Med* 87(1): 7-14.
26. Bellingham AJ, Detter JC, Lenfant, C. (1971). Regulatory mechanisms of hemoglobin oxygen affinity in acidosis and alkalosis. *J Clin Invest* 50(3): 700-706.
27. Fall PJ, Szerlip HM. (2005). Lactic acidosis: from sour milk to septic shock. *J Intensive Care Med* 20(5): 255-271.

Benefits of Publishing with EScientific Publishers:

- ❖ Swift Peer Review
- ❖ Freely accessible online immediately upon publication
- ❖ Global archiving of articles
- ❖ Authors Retain Copyrights
- ❖ Visibility through different online platforms

Submit your Paper at:

<https://escientificpublishers.com/submission>