UCLA Health

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Abstract Form

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Project Title:	Differentiating Type A and Type B Lactic Acidosis in Septic Oncologic Patients

Research Category (please check one):							
☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction:
Type B lactic acidosis is a complication of hematologic malignancies whose exact pathogenesis is unclear. Current theories include increased anaerobic metabolism and pyruvate production from high malignant cell turnover rate (1). Type A lactic acidosis is caused by global versus focal hypoperfusion and hypoxia usually in septic states. However, the etiology of lactic acidosis in a patient with both possible sepsis and oncologic emergency can be difficult to elucidate, especially in a critical care setting. We present a patient with newly diagnosed lymphoma whose initial clinical picture suggested sepsis but ultimately was found to have type B lactic acidosis complicated by tumor lysis syndrome.

Case Report:
A 55-year-old male with a history of HIV/AIDS and cryptococcal meningitis complicated by residual bilateral lower extremity spastic hemiparesis presented with oral intolerance, nausea, and emesis for two weeks prior to admission. Vitals were significant for tachycardia to the 110s, and labs notable for elevated lactate of 5.2. Imaging was concerning for malignancy given diffuse abdominal and retroperitoneal lymphadenopathy with normal tumor markers of CEA, CA19-9, and AFP. The patient was presumed septic with urine culture growing Enterococcus faecium and started on appropriate antibiotics. However, the lactate continued to rise to 7.8 and uric acid to 8.3 concerning for tumor lysis syndrome. After receiving a total of 12 liters of fluids over three days, the patient became acutely tachypneic and required escalation to BiPAP for oxygen support, which resolved with IV diuresis. Biopsy of an omental nodule resulted as stage 4 diffuse large B-cell lymphoma and the patient was initiated on inpatient chemotherapy with R-EPOCH. The lactic acidosis and tumor lysis labs resolved on C1D12 of chemotherapy initiation. At the time of writing, the patient had undergone six cycles of R-EPOCH with PET/CT showing complete response.

Discussion:
Differentiating between sepsis causing type A lactic acidosis versus an independent type B lactic acidosis as a cause of this patient's fluctuating lactate levels relied mainly on infectious workup and imaging. This specific patient's elevated lactate was likely secondary to the Warburg effect in the setting of newly diagnosed lymphoma. The pathophysiology is theorized to be increased anaerobic metabolism and pyruvate production from high turnover of malignant cells, glycolysis acceleration via type II hexokinase enzyme overexpression, or impaired pyruvate metabolism due to liver metastases if present (2). In this case, the patient did not present with liver metastases and so the more likely mechanism was thought to be enhanced glycolytic activity in malignant cells that exceeded the normal lactic acid clearance rate in the setting of extensive disease. Despite the positive urine culture growing 50,000 CFU Enterococcus, the clinical picture was not consistent with sepsis. In patients with a hematologic malignancy, a high degree of clinical suspicion should be maintained for underlying sepsis given their immunocompromised status, particularly with concomitant neutropenia. However, in patients who present with an otherwise stable clinical picture and negative infectious workup, the elevated lactate can be attributed to the Warburg effect. Future studies should focus on elucidating this phenomenon in patients with solid malignancies which has prognostic implications.

References:

(1) Liu QS, Harji F, Jones A, Tarnower AC. Type B lactic acidosis: a rare oncological emergency. BMJ Case Rep. 2020 Mar 31;13(3):e233068. doi: 10.1136/bcr-2019-233068. PMID: 32234853; PMCID: PMC7167422.
(2) El Imad T, El Khoury L, Geara AS. Warburg's effect on solid tumors. Saudi J Kidney Dis Transpl. 2014 Nov;25(6):1270-7. doi: 10.4103/1319-2442.144266. PMID: 25394449.