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Lactic Acidosis and Atrial Tachycardia: Unusual Presentations of Disseminated Burkitt-Like Lymphoma*

Hazem El-Osta[1#], Abdulrahman Abdulbaki[2], Prakash Peddi[1], Amol Takalkar[3], James Cotelingam[4], Diana Veillon[4], Andres Vargas[5], Nuri Akkus[2], Gerhard C. Hildebrandt[1]

[1]Department of Medicine, Division of Hematology oncology, Louisiana State University Health Sciences Center, Shreveport, USA; [2]Department of Medicine, Division of Cardiology, Louisiana State University Health Sciences Center, Shreveport, USA; [3]Department of Nuclear Medicine, Louisiana State University Health Sciences Center, Shreveport, USA; [4]Department of Pathology, Louisiana State University Health Sciences Center, Shreveport, USA; [5]Department of Medicine, Division of Internal Medicine, Louisiana State University Health Sciences Center, Shreveport, USA.
Email: #helost@lsuhsc.edu

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ABSTRACT

Lactic acidosis is a rare complication of malignancies and is seen more frequently in high grade lymphoma and leukemia. Although, its pathogenesis is not well understood, it remains a surrogate of poor prognosis. Herein, we present a case of Burkitt-like lymphoma presenting with metabolic abnormalities including lactic acidosis and hypoglycemia along with atrial tachycardia. We will discuss the different mechanisms involved in these metabolic disturbances and we will provide insight on novel therapeutic strategies based on our understanding of the underlying pathophysiology.

Keywords: Atrial Tachycardia; Burkitt-Like Lymphoma; B-Cell Lymphoma; Intra-Cardiac Tumor; Hypoglycemia; Lactic Acidosis

1. Introduction

Burkitt's lymphoma is a highly aggressive form of B-cell non Hodgkin lymphoma characterized pathologically by highly mitotic small non cleaved cells with round nuclei and multiple nucleoli giving a "starry-sky" pattern on histology. On molecular level, it is characterized by translocation of the c-myc gene leading to its dysregulation. It comprises 3 distinct clinical forms: endemic (African), sporadic and immunodeficiency-associated primarily seen in acquired immunodeficiency syndrome (AIDS) patients. Burkitt-like lymphoma is a morphologic variant of Burkitt's lymphoma and has pathologic features intermediate between Burkitt's lymphoma and diffuse large B cell lymphoma.

Patients with Burkitt's lymphoma present with rapidly growing masses and bulky lymphadenopathy. The endemic forms presents typically with jaw mass. On the other hand, the sporadic form presents usually with abdominal mass whereas the immunodeficiency-associated form often involves the lymph nodes.

It is often accompanied by oncologic emergencies secondary to compression by the tumor bulk, metabolic disturbances, or treatment-related hematological toxicities. Despite its aggressive nature, response to combination chemotherapy is prompt, although associated with high risk for the development of tumor lysis syndrome.

One of the rare metabolic complications of Burkitt's lymphoma is lactic acidosis. Its underlying pathogenesis is complex and believed to be due to imbalance of lactic acid production by the tumor itself and clearance rather than hypoxia and hypoperfusion [1]. It is a surrogate of poor prognosis as described in few case reports.

2. Case Report

A 33-year-old female with no past medical history presented to an outside facility for 2 months history of severe weight loss, malaise and dysphagia.

She underwent an esophagogastroduodenoscopy which demonstrated a large necrotic ulcerative fungating mass in the cardia, body and lesser curvature of the stomach, of which biopsies were taken. The diagnosis of B-cell lymphoma with features intermediate between diffuse large B-cell lymphoma and Burkitt's lymphoma was made on pathology. C-myc gene rearrangement was positive on fluorescence in situ hybridization (FISH). On immunohistochemistry CD10, CD20 and leukocyte common antigen (LCA) were positive and CD3, CD5 and cyclin D1 were negative.

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#Corresponding author.

Upon examination, she appeared cachectic. Pulse was 142/min and regular, blood pressure 104/78 mmHg, respiratory rate 20/min, temperature 96.0 F, oxygen saturation 97% on room air.

Physical exam was remarkable for oral thrush and massive left abdominal mass on palpation. No lymphadenopathy was noticed.

Her laboratory studies showed the following pertinent values (reference range): sodium 125 mmol/L (136 - 145 mmol/L), potassium 4.4 mmol/L (3.5 - 5.1 mmol/L), chloride 91 mmol/L (98 - 107 mmol/L), bicarbonate 17 mmol/L (21 - 32 mmol/L), anion gap 17 mmol/L (5 - 15 mmol/L), glucose 64 mg/dl (70 - 110 mg/dL), creatinine 0.6 mg/dL (0.6 - 1.3 mg/dL), uric acid 2 mg/dL (2.6 - 6 mg/dL) , bilirubin 1.6 mg/dL (0.2 - 1 mg/dL), aspartate aminotransferase (AST) 119 U/L (15 - 37 U/L), alanine aminotransferase (ALT) 32 U/L (12 - 78 U/L), lactate 11.1 mmol/L (0.7 - 2.1 mmol/L), lactate dehydrogenase (LDH) 1884 U/L (84 - 246 U/L), HIV serology positive confirmed by western blot with 349494 RNA copies per ml. Hepatitis B and C serology negative, white blood cell count (WBC) 15.7 K/microl (3.4 - 9.2 K/microl), Hemoglobin 11.4 g/dL (11.3 - 15.4 g/dL), platelet 136 K/microl (142 - 405 K/microl), CD4 count of 83/mm^3, partial thromboplastin time (PTT) 33 sec (24.7 - 35.5 sec) and international normalized ratio (INR) 1.38. Electrocardiogram was concordant with atrial tachycardia.

She was immediately started on intravenous fluid, bicarbonate, allopurinol for tumor lysis syndrome prophylaxis and dexamethasone 40 mg intravenous daily for 3 days. Emtricitabine/tenofovir/efavirenz was commenced for HIV and metoprolol for tachycardia, respectively.

Further work-up by echocardiogram showed a 3 × 5 cm large right atrial mass protruding to the tricuspid valve with maintained normal ejection fraction. Positron emission tomography/computed tomography scan (PET/CT scan) indicated extensive intensely fluorodeoxyglucose (FDG) avid widespread malignant disease involving the right atrium, distal esophagus and stomach, several bowel loops, left adrenal gland, right kidney, bone marrow and possibly the intraspinal canal. Bone marrow biopsy confirmed lymphoma involvement, and spinal fluid analysis revealed presence of lymphoma cells while MRI of the brain was unremarkable.

Patient received 12 mg of methotrexate intrathecally time one. Subsequently and after written informed consent was obtained from the patient, dose adjusted R-EPOCH multiagent chemotherapy (rituximab, etoposide, vincristine, cyclophosphamide, and doxorubicin) was initiated. Three days later, her blood counts dropped significantly with platelet of 67 K/microl, WBC of 0.79 K/microl and Hemoglobin of 10.2 g/dL. Her creatinine, phosphorus, potassium and uric acid remained normal, and her lactic acidosis improved with a lactate of 2.6 mmol/L. Clinically, she acutely deteriorated over the next 24 hours developing fever and severe hypotension, and was transferred to the intensive care unit. She was started on intravenous vancomycin and piperacillin/tazobactam and aggressive volume substitution. Microbiological work-up revealed positive cultures for E. coli in blood and urine, consistent with E. coli sepsis. Her clinical state further deteriorated over the next few hours, leading to acute respiratory failure, altered mental status and shock. Despite the short duration of events, in consideration of the poor overall prognosis, the family decided on do not resuscitate/Do not intubate (DNR/DNI), and the patient succumbed to gram negative sepsis 7 days after admission.

3. Discussion

We presented a patient with HIV-related Burkitt-like lymphoma with atrial tachycardia due to lymphomatous cardiac involvement, severe systemic extent of disease and metabolically lactic acidosis and hypoglycemia in the absence of hypoxia or hypoperfusion. Although the patient presented with tachycardia and elevated white count, the negativity of blood cultures upon admission along with the improvement of the lactic acidosis after treatment with chemotherapy are arguments that favor lymphoma as a cause of the lactic acidosis rather than an underlying sepsis. Our case underscores few particular observations. Although rarely described, lactic acidosis without evidence for hypoxia or hypoperfusion can point towards an underlying hematological malignancy and prompt diagnosis and early treatment of underlying etiology potentially improves outcome. The underlying pathophysiology of lactic acidosis in these patients is not yet fully understood. Several mechanisms have been postulated including 1) overproduction of lactic acid by tumor cells through an upregulation of enzymes involved in aerobic glycolysis, through a highly proliferative tumor in mismatch with needed tumor blood supply leading to tumor cell hypoxia and increased anaerobic glycollysis [1-5], and it was also suggested, that lactic acidosis is simply the result of bulk of disease rather than an increase of lactic acid at a cellular level [6]; 2) Impaired hepatic and renal clearance of lactic acid [1,5]; 3) Microvascular embolization of tumor cells causing a state of tissue hypoperfusion and therefore leading to anaerobic glycolysis [5,7]; 4) Thiamine deficiency leading to a reduction in the pyruvate dehydrogenase activity and consequently to accumulation of pyruvate which will be converted to lactate [8].

The treatment of malignancy-associated lactic acidosis is mainly empirical and based on few case reports and case series. Acidosis is often responsive to aggressive chemotherapy [1,5,9,10], although this does not seem to affect the clinical outcome as illustrated in our patient and previous reported cased described in **Table 1**. Che-

Table 1. Table summarizing the characteristics and outcomes of reported cases of Burkitt and Burkitt-like lymphoma associated with lactic acidosis.

Case	Age	Sex	Presenting sign	HIV status	Glycemia	Lactate peak	Lactic acidosis resolution	Treatment received	Outcome	Comment
Block J. B. *et al.* [2] (1966)	27	F	Ascites, pleural effusion	NR	Normal	3.55	Yes	Nitrogen mustard, vincristine, prednisone, ibenzmethyzin	Died in 3 months	Increased lactate in pleural fluid
Rouzet P. *et al.* [19] (1991)	8	M	Small bowel obstruction	NR	Normal	139	Yes	LMB-84	Alive after 2.5 years	LA responded quickly to thiamine
Bergin C. *et al.* [17] (1993)	26	M	Constitutional symptoms	positive	Mildly low	12.2	Slight improvement (decreased to 7.9)	Combination chemotherapy (not specified)	Died in 7 days	
Revesz T. *et al.* [16] (1995)	8	M	Seizure, diabetes insipidus	NR	Normal	24	Improved (decreased to 6.2)	Etoposide, steroid, XRT	Died in 4 months after relapse	Kidney involvement
Megarbane B. *et al.* [18] (2000)	77	M	Fever of unknown origin, SIRS	NR	Normal	27	Yes	CVP, intrathecal chemo	Died from sepsis	
Glasheen J. *et al.* [14] (2005)	74	M	Pleural effusion, right sided edema	NR	Low	15.8	NR	None	Died in 13 days	
Lopez-Rodriguez M. *et al.* [15] (2007)	33	M	Back pain	Positive	Low	8.41	NR	None	Died in 7 days	
Rastogi M. *et al.* [21] (2008)	11	M	Fever, edema, rash	Positive	Intractable hypoglycemia	15.2	Decreased to 9.6	None	Death in 33 days	Received glucagon for hypoglycemia
Kulkarni K. *et al.* [20] (2010)	12	M	Abdominal pain, weakness	Negative	Normal	21	Yes	MCP-842	Recovery (unspecified follow-up)	Elevated triglyceridemia
Present report	33	F	Constitutional symptoms, dysphagia, atrial tachycardia	Positive	Low	11.1	Improved to 2.6	R-EPOCH	Died in 7 days	

CVP: cyclophosphamide, vincristine and prednisone; **LMP-84:** intensive combination chemotherapy protocol containing vincristine, cyclophosphamide, prednisone, doxorubicin, high dose methotrexate and cytarabine; **MCP-842:** ifosfamide-based, moderately intensive short-duration combination chemotherapy protocol; **NR:** not reported; **R-EPOCH:** chemotherapy protocol containing rituximab, etoposide, vincristine, cyclophosphamide, doxorubicin and prednisone; **XRT:** radiation therapy.

motherapy seems to exert its effect by cytoreduction of tumor cells leading to decreased production of lactic acid and in cases of liver involvement to improved lactic acid clearance. Renal dialysis can be useful in some cases [1,5,9,10]. Resolution of lactic acidosis with thiamine repletion has been described previously in lymphoma [8]; however it did not have any effect on lactic acid level in Burkitt's lymphoma.

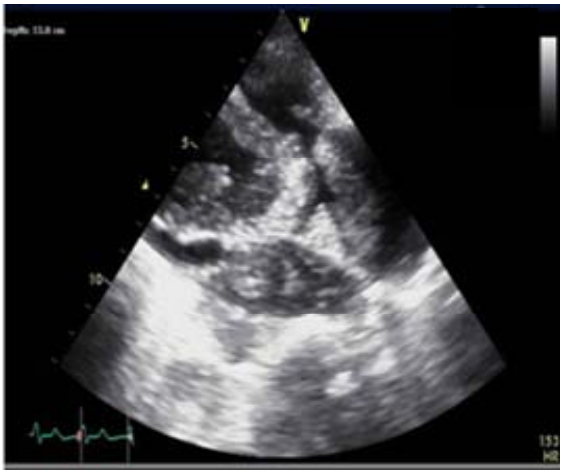
Amid the uncertainty in the optimal management of this condition, a better recognition of the different mechanisms involved in lactic acidosis of hematological malignancies is needed. For instance, anecdotal reports have found elevated IGF1 and TNFalpha levels in a lymphoma patient with lactic acidosis [1]. Although no causality link could be established between an increase in insulin growth factor 1 (IGF-1) and development of lactic acidosis, this is an interesting observation since IGF-1 induces the expression of hexokinase which results in high rates of glycolysis and pyruvate production [3,4]. On the other hand, tumor necrosis factor alpha (TNF alpha) leads to reduced activity of pyruvate dehydrogenase and subsequently increased conversion of pyruvate into lactate [11]. Accordingly, it can be postulated that the IGF1 pathway can work in concert with TNF alpha to increase lactic acid production. Importantly, IGF1 signaling is implicated in many types of cancers [12]. Several IGF targeting agents are in early clinical trials and have shown promising results [13]. It will be important to test whether lactic acidosis in lymphoma is a reflection of an underlying overactivated IGF-1 pathway that can drive tumor cell proliferation, and to determine whether IGF-1, TNF and maybe other candidate pathways are valuable therapeutic targets that could change the disease's outcome.

Lactic acidosis associated with Burkitt-like lymphoma portends an extremely poor prognosis. Among the 10

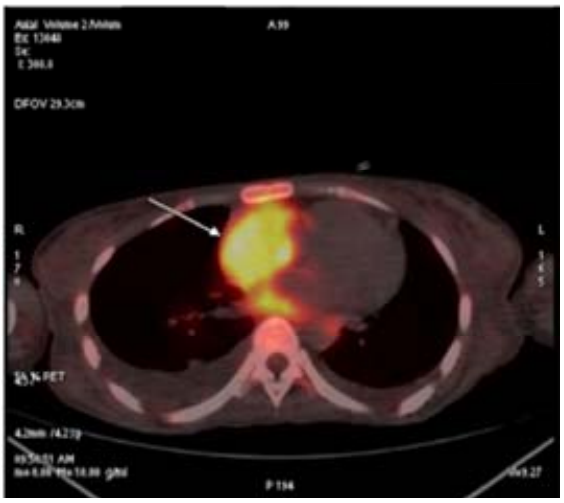
reported cases including our case, 8 patients had a fatal outcome, with death occurring in a matter of days to weeks from the onset of lactic acidosis. In only one case, the patient was alive after 2.5 years of diagnosis and in another case the outcome was not well specified (**Table 1**) [2,14-21].

Using FDG-PET/CT, generally FDG uptake is strong in myocardium and brain. In our patient, the pattern of FDG metabolic distribution demonstrated less uptake in these tissues than usually observed, whereas significant FDG uptake was seen in the lymphoma spread throughout the body. This observation eludes to an indirect visual insight into one potential mechanism of hypoglycemia in lymphoma, as it suggests an increase in glucose uptake by tumor cells as a possible contributor of systemic hypoglycemia.

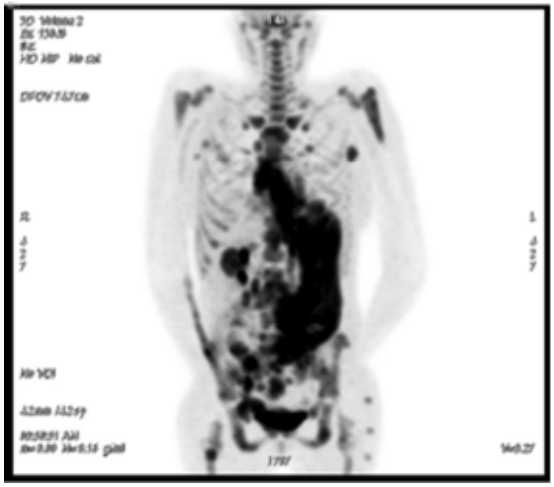
Our case illustrates the complementarities of multimodality imaging in assessing cardiac involvement by Burkitt-like lymphoma. The sensitivity and specificity of FDG PET/CT scan for detection of extranodal Burkitt-like lymphoma are 100% and 94% respectively, and few



(a)



(b)



(c)

Figure 1. (a) echocardiogram demonstrating a right ventricular Inflow track showing the right atrial mass which extends to the tricuspid valve. (b) PET/CT scan showing an intensively FDG avid lesion in the right atrium corresponding to the lesion seen on echocardiogram. (c) PET scan indicating diffusely intense FDG uptake consistent with extensive disease.

reports have shown its utility to detect intra-cardiac and myocardial involvement [22,23]. In our case, FDG-PET/CT provided imaging confirmation of echocardiographic findings of a right atrial mass, and in addition its metabolic activity provided crucial clues on the lymphomatous origin versus non-metabolically active lesion such as thrombus (**Figure 1**).

Intra-cardiac tumors presenting as arrhythmia were reported in left atrial myxoma, and intracardiac follicular B cell lymphoma, the latter mostly reported as atrial flutter [24-28]. To our knowledge this is the first reported case of intracardiac metastatic Burkitt-like lymphoma presenting as atrial tachycardia, which emphasizes a meticulous work-up in patients with malignancies for cardiac involvement in the presence of diagnostic or clinical abnormalities.

4. Conclusion

Lactic acidosis associated with Burkitt-like lymphoma is a rare and life threatening medical condition. It results from the imbalance between increase lactic acid production and decreased lactic acid clearance. Determination of lactic acid level in patients with aggressive lymphoma and elevated anion gap is warranted for early detection and treatment of this condition. Treatment of the underlying hematologic malignancy by chemotherapy can lead in some cases to resolution of lactic acidosis but the final outcome is almost invariably fatal. A better understanding of lactic acidosis pathophysiology in the setting of cancer will hopefully translate into improved

therapeutic modalities.

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