

Stress-induced cardiomyopathy complicating severe babesiosis

Esosa Odigie-Okon¹, Emmanuel Okon², John Dodson³, Gabriel Vorobiof³

¹Department of Medicine, Yale University (Bridgeport Hospital), Bridgeport, CT, USA

²Department of Medicine, New Milford Hospital

(New York Presbyterian Healthcare System), New Milford, CT, USA

³Departments of Cardiology and Diagnostic Radiology, Yale University School of Medicine, New Haven, CT, USA

Abstract

A post-menopausal lady with severe babesiosis developed a basal-type stress-induced cardiomyopathy. Left ventricular function normalized at three months. We believe this is the first reported case of stress cardiomyopathy complicating severe babesiosis. Cardiac biomarker elevation disproportionate to the area of myocardial dysfunction, electrocardiographic changes, the patient's clinical condition, and close follow-up of left ventricular function parameters are all vital in diagnosing stress cardiomyopathy and may exclude the need for coronary angiography. There may be a possible association between severe babesiosis and stress cardiomyopathy. (Cardiol J 2011; 18, 1: 83–86)

Key words: stress-induced cardiomyopathy, tako-tsubo, babesiosis

Introduction

Babesiosis is a hemolytic protozoan illness caused most commonly by *Babesia microti* specie in the United States. It is transmitted to humans via the tick vector *Ixodes scapularis*, and less often via blood transfusion. We describe a case of stress-induced cardiomyopathy (SCM) that occurred in association with severe babesiosis. To the best of our knowledge, this is the first report of SCM complicating babesiosis.

Case and results

A 62 year-old Caucasian lady with hypertension, hypercholesterolemia, and no significant family or social history, presented initially to her PMD's office complaining of fevers up to 102.9° F, chills, nausea and headaches. Approximately seven weeks prior to this admission, she had a nine day hospital stay for surgery involving resection of a left-sided retroperitoneal liposarcoma, left kidney, adrenal

gland, ovary, hemi-diaphragm and a splenectomy. She received two units of packed red blood cells following a post-operative drop in hematocrit from 32.1% to 20.7%. Approximately three and a half weeks after her discharge, she was readmitted for nine days for management of nosocomial pneumonia and malnutrition. She was discharged home on total parenteral nutrition (TPN) via a peripherally inserted central (PICC) line. She presented ten days later. Physical examination in the office revealed a fatigued lady with laboratory investigations significant for a platelet count of 70,000/ μ L. Blood cultures were drawn, a dose of vancomycin and cefepime was given, and she was admitted to the hospital for further management. In the hospital, she was in mild distress and febrile, with a temperature of 102.7° F. Blood pressure was 100/70 mm Hg, heart rate 104 bpm and oxygen saturation was 100% on room air. Other significant findings included decreased breath sounds at the left lung base and a normal cardiovascular exam.

Address for correspondence: Esosa Odigie-Okon, MD, MSc, Bridgeport Hospital, 267 Grant St, Bridgeport, CT 06610, USA, tel.: 781 706 10 02, fax: 203 384 42 94, e-mail: sesky146@yahoo.com

Received: 30.10.2009

Accepted: 3.12.2009

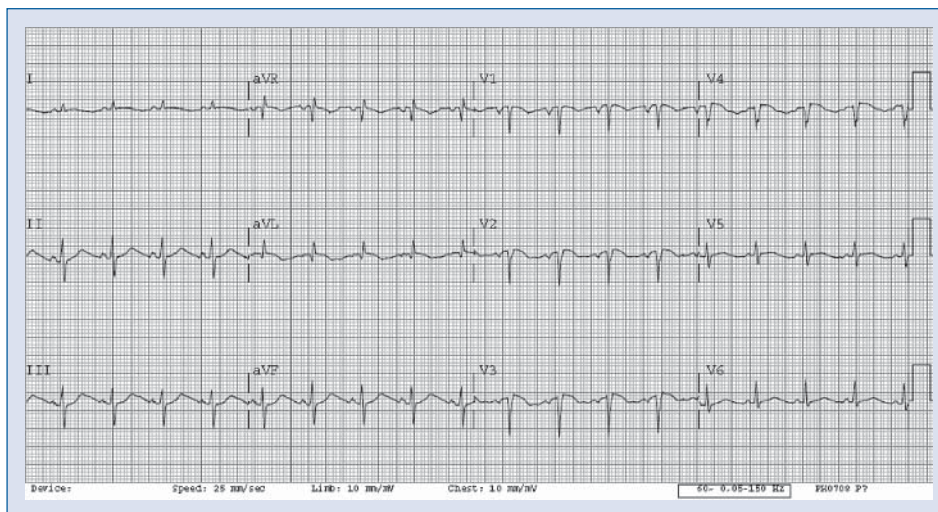


Figure 1. Electrocardiogram showing Q waves in leads aVL, V2–V4.

The patient was admitted to the medical unit, and placed on vancomycin and piperacillin-tazobactam. On day one, significant laboratory results revealed leucocytosis (WBC count 15,000/ μ L), normocytic anemia (hemoglobin 9.9 g/dL), thrombocytopenia (platelet count 83,000/ μ L), aspartate aminotransferase (AST) 119 U/L; alanine aminotransferase (ALT) 65 U/L; alkaline phosphatase 224 U/L, unconjugated hyperbilirubinemia 2.12 mg/dL and normal renal function. On day two, hemoglobin dropped to 7 g/dL, reticulocyte count was 13.5%, and lactate dehydrogenase 1770 μ /L (normal range 313–618 μ /L), suspicious for ongoing hemolysis. Coagulation work-up was negative for consumptive coagulopathy. Blood smear was positive for intra-erythrocyte parasites consistent with babesia. Babesia polymerase chain reaction was positive and the parasite load was 7%. Clindamycin and quinine were started and exchange blood transfusion was performed with seven units of packed red blood cells. Subsequent parasite load monitored over one week remained less than 1%. In addition, blood cultures grew methicillin-sensitive staphylococcus aureus and torulopsis glabrata. She was treated with oxacillin and anidulafungin.

On day four, she became progressively short of breath and hypoxic. Chest X-ray showed pulmonary edema, unresponsive to furosemide. She was intubated and transferred to the intensive care unit. An electrocardiogram revealed sinus tachycardia, ST elevations and Q-waves in leads aVL, V2–V4 (Fig. 1).

Three sets of cardiac biomarkers over the course of 24 hours showed mildly elevated tropon-

in-I levels from 0.28–0.44 ng/mL and creatinine kinase-MB fraction (CK-MB) from 2.8–4.3 ng/mL, brain natriuretic peptide was 1,860 pg/mL. An echocardiogram showed an overall decrease in left ventricular (LV) systolic function, estimated ejection fraction of 30%, a hypokinetic mid antero-septum and anterior wall, extending to and involving the entire apex, with a global hypokinesis of all other segments. Right ventricular function was normal, and trace mitral and tricuspid regurgitations were noted (Fig. 2).

A diagnosis of stress-induced cardiomyopathy was entertained, and a decision was made not to proceed with invasive coronary angiography as a true anterior wall myocardial infarction would have resulted in far greater elevations of the cardiac biomarkers than the area of myocardial injury visible on echocardiography. She was diuresed and extubated after 48 hours.

Repeat echocardiogram six days later revealed improvement of the left ventricular function to an estimated left ventricular ejection fraction of 45%, normal left ventricular cavity size, normal left ventricular wall thickness, hypokinetic mid septum and mid inferior segment, and significantly improved anterior and anteroseptal wall motion abnormalities. She was discharged on day eleven to complete a five week course of atovaquone and azithromycin for babesiosis, and chronic management with lisinopril, furosemide and aspirin. She was seen three months later in the cardiology clinic, and a follow-up echocardiogram showed a normal left ventricular systolic function without wall motion abnormalities.

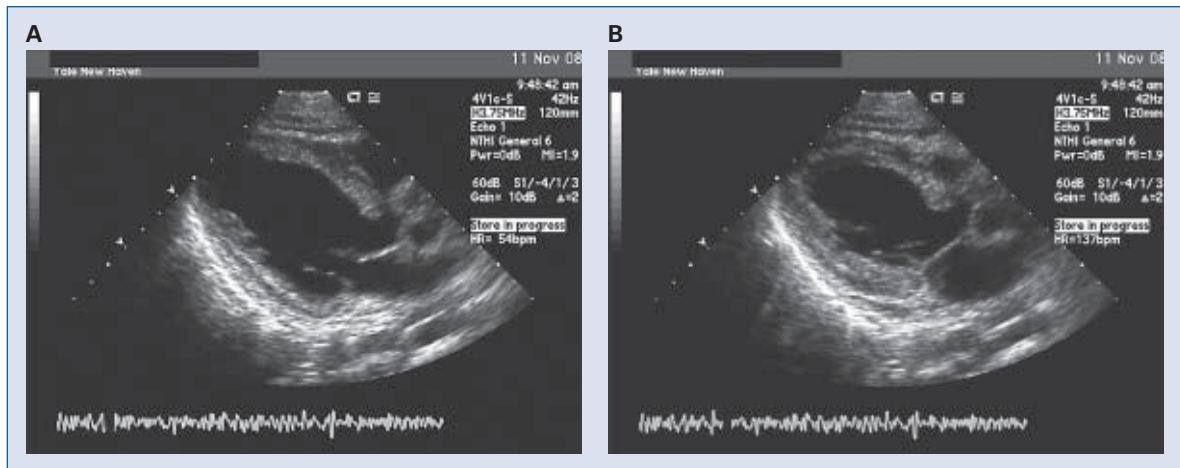


Figure 2. A. Parasternal long axis still shot in diastole; B. Parasternal long axis still shot in systole.

Discussion

Prompted by the rarity in Japan of a peculiar pattern of contrast left ventriculogram, consisting of hypokinesis or akinesis from the mid-portion to the apical area and hyperkinesis of the basal area, combined with an end-systolic left-ventriculogram similar to a ‘tako-tsubo’ (a contraption used for trapping octopuses in Japan), the term ‘tako-tsubo left ventricular dysfunction’ was coined [1]. Over time, recognition of similar pathogenetic mechanisms has given rise to the term ‘stress-induced cardiomyopathy’.

SCM is a syndrome of unclear etiology, characterized by a transient contractile dysfunction of the mid-LV segments with or without apical involvement, extending beyond a single epicardial vascular distribution; absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; new electrocardiographic abnormalities (either ST-segment elevation, T-wave inversion, or both) or modest elevation in cardiac troponin; and the absence of a pheochromocytoma or myocarditis [2]. Variants of this syndrome are increasingly recognized including a mid-ventricular and basal ballooning pattern of LV dysfunction. Various mechanisms of SCM have been proposed, including neurogenic myocardial stunning, excessive catecholamines, microvascular dysfunction and myocarditis [3, 4]. In babesiosis, infected erythrocytes cause vascular stasis, leading to microvascular obstruction, tissue anoxia and ischemia [5]. Animal models have specifically described cases of cardiomegaly, myocarditis, and multifocal coagulation cardiac necrosis [6, 7]. We hypothesize that there may be an association of SCM in cases of severe babesiosis. Research into the pathophysiology is warranted.

The incidence of SCM is uncertain, perhaps due to the complexities involved in its presentation and the requirement for a diagnosis to be made after excluding an acute myocardial infarction. Nonetheless, evidence suggests it is more common in women than men [8] and also seems to have a high incidence in medical intensive care units [9, 10]. Observed complications have ranged from pulmonary edema (as in our patient) to cardiogenic shock and death [11]. Treatment of this illness is largely supportive and based on the overall clinical condition of the patient. Empiric therapy favors the use of standard medications for left ventricular systolic dysfunction including angiotensin-converting enzyme inhibitors, beta-blockers and diuretics [12]. The appropriate duration of therapy remains unknown.

The events in this patient’s illness occurred between September and November, outside the typical tick-bite summer months which peak in July. However, our patient’s recent hospital stay made it unlikely that her babesiosis infection was the result of a tick-bite. As such, circumstantial evidence points to the post-operative blood transfusion as the source of her babesiosis. In contrast to the 1–6 week incubation period following a tick bite, post-transfusion disease typically has a 6–9 week incubation period [13]. In the hospital setting, where blood products have seemingly undergone rigorous screening processes, physicians are prone to forget the inadequacy of screening blood products for babesiosis [14, 15]. A broad index of suspicion should be the ‘rule’ in patients with a recent history of blood transfusion presenting with a febrile illness and hemolytic anemia.

In conclusion, this is the first known case demonstrating that SCM may be a complication of se-

vere babesiosis with high parasitemia counts. Clues to the diagnosis are: cardiac biomarker elevation out of proportion to the area of myocardial dysfunction; typical electrocardiographic findings; the patient's clinical context; and close follow-up of global and regional LV function.

Acknowledgements

The authors declare no conflicts of interest.

References

1. Kurisu S, Sato H, Kawagoe T et al. Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: A novel cardiac syndrome mimicking acute myocardial infarction. *Am Heart J*, 2002; 143:448-455.
2. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): A mimic of acute myocardial infarction. *Am Heart J*, 2008; 155: 408-417.
3. Kurowski V, Kaiser A, von Hof K et al. Apical and midventricular transient left ventricular dysfunction syndrome (tako-tsubo cardiomyopathy): Frequency, mechanisms, and prognosis. *Chest*, 2007; 132: 809-816.
4. Bybee KA, Prasad A. Stress-related cardiomyopathy syndromes. *Circulation*, 2008; 118: 397-409.
5. Dao AH, Eberhard ML. Pathology of acute fatal babesiosis in hamsters experimentally infected with the WA-1 strain of *Babesia*. *Lab Invest*, 1996; 74: 853.
6. Oz HS, Hughes WT. Acute fulminating babesiosis in hamsters infected with *Babesia microti*. *Int J Parasitol*, 1996; 26: 667-670.
7. Wozniak EJ, Lowenstine LJ, Hemmer R, Robinson T, Conrad PA. Comparative pathogenesis of human WA1 and *Babesia microti* isolates in a Syrian hamster model. *Lab Anim Sci*, 1996; 46: 507-515.
8. Wittstein IS, Thiemann DR, Lima JAC et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med*, 2005; 352: 539.
9. Park JH, Kang SJ, Song JK et al. Left ventricular apical ballooning due to severe physical stress in patients admitted to the medical ICU. *Chest*, 2005; 128: 296-302.
10. Dec GW. Recognizing the apical ballooning syndrome in the intensive care unit. *Intensive Care Med*, 2006, 32: 962-964.
11. Donahue D, Movahed MR. Clinical characteristics, demographics and prognosis of transient left ventricular apical ballooning syndrome. *Heart Fail Rev*, 2005; 10: 311-316.
12. Bybee KA, Prasad A, Barsness GW et al. Clinical characteristics and thrombolysis in myocardial infarction frame counts in women with transient left ventricular apical ballooning syndrome. *Am J Cardiol*, 2004; 94: 343-346.
13. Villar BF, White DJ, Benach JL. Human babesiosis. *Prog Clin Parasitol*, 1991; 2: 129.
14. Leiby DA, Chung AP, Gill JE et al. Demonstrable parasitemia among Connecticut blood donors with antibodies to *Babesia microti*. *Transfusion*, 2005; 45: 1804-1810.
15. McQuiston JH, Childs JE, Chamberland ME, Tabor E. Transmission of tick-borne agents of disease by blood transfusion: A review of known and potential risks in the United States. *Transfusion*, 2000; 40: 274-284.