Available online at www.sciencedirect.com

ScienceDirect

Journal of Acute Medicine 4 (2014) 99–102

www.e-jacme.com

Case Report

Lazarus phenomenon in a patient with Duchenne muscular dystrophy and dilated cardiomyopathy

Müge Adanalı [a], Tolga Sinan Güvenç [b,*], Murat Yıldırım Kale [c], Sibel Onaç [a], Ülfettin Hüseyinoğlu [a], Yavuz Karabağ [b], Bahattin Balcı [b]

[a] *Department of Anesthesiology and Reanimation, Kafkas University School of Medicine, Kars, Turkey*
[b] *Department of Cardiology, Kafkas University School of Medicine, Kars, Turkey*
[c] *Department of Neurology, Kafkas University School of Medicine, Kars, Turkey*

Received 11 February 2014; accepted 3 March 2014
Available online 25 June 2014

Abstract

Lazarus phenomenon (LP) is the spontaneous return of circulation after cessation of resuscitation. We herein report the case of a 21-year-old man with Duchenne muscular dystrophy and dilated cardiomyopathy who was hospitalized in the intensive care unit because of respiratory distress. Three days after the initial admission, the patient experienced asystole and did not respond to resuscitation for 30 minutes. Ten minutes after cessation of resuscitation, normal sinus rhythm with a palpable pulse was noted, and the patient was reintubated. Sixty days after admission, the patient was discharged from hospital with complete neurological recovery. Intensive care physicians should be aware of the LP, although it is rare, and a watchful waiting period is advisable after cessation of resuscitation.

Keywords: dilated cardiomyopathy; Duchenne muscular dystrophy; Lazarus phenomenon; spontaneous return of circulation

1. Introduction

Lazarus phenomenon (LP) is the term applied to spontaneous return of circulation after the cessation of cardiopulmonary resuscitation (CPR). The term was first coined by Bray in 1983 because of its resemblance to the Biblical story of Lazarus.[1] The frequency of the condition is not known, and it is considered quite rare because of the scarcity of reported cases.

The exact mechanism underlying LP is not known, although hyperventilation, hyperkalemia, late effects of inotropic drugs, and myocardial ischemia are possible explanations.[2] Because of the presumed infrequency of this condition, there are no studies that have specifically researched LP in the scientific literature.

In this article, we report a 21-year-old man with Duchenne muscular dystrophy and dilated cardiomyopathy who experienced in-hospital cardiac arrest and did not respond to cardiac resuscitation for 30 minutes. Ten minutes after cessation of resuscitation, spontaneous respiration and circulation were observed, with a complete neurological recovery by discharge. To the best of our knowledge, this is the first reported case of LP in a patient with dilated cardiomyopathy.

2. Case report

A 21-year-old man with known Duchenne muscular dystrophy was admitted to emergency department with shortness of breath and peripheral cyanosis for 2 days. His only known medical condition was Duchenne dystrophy and he was not using any medications. On physical examination, his blood pressure was 100/70 mmHg, pulse rate was 120 beats/minute, and body temperature was 35.9°C. Auscultation revealed

* Corresponding author. Department of Cardiology, Kafkas University School of Medicine, Kafkas University Campus, Paşaçayırı, Kars, Turkey.
E-mail address: tsguvenc@gmail.com (T.S. Güvenç).

http://dx.doi.org/10.1016/j.jacme.2014.03.003

globally reduced pulmonary sounds, with a 1/6 systolic murmur at the apex of heart. His electrocardiogram (ECG) showed sinus rhythm with mild tachycardia and prominent R waves in the right precordial leads (Fig. 1). He was cachectic, with severe muscle wasting in all extremities (Fig. 2). Arterial blood gas analysis showed severe, uncompensated respiratory acidosis with the following measurements: pH, 6.9; partial pressure of O_2 (pO_2), 74 mmHg; partial pressure of CO_2 (pCO_2), 105 mmHg; and HCO_3, 22.5 mmol/L. He was diagnosed with respiratory failure and possible pneumonia, transferred to the intensive care unit (ICU), and intubated to avoid a respiratory collapse. His initial biochemical analysis showed hypokalemia (2.68 mEq/L) with high liver transaminase levels (alanine aminotransferase, 292 IU/L; aspartate aminotransferase, 109 IU/L). His initial medical treatment included the following medications: cefazolin [3 × 1 g, intravenously (i.v.)], salbutamol [6 × 2.5 mg intratracheal (i.t.)], *N*-acetylcysteine (10 mL; 10% sol.; 6 × 1 i.t.), famotidine (50 mg 3 × 1 i.v.). Potassium chloride was given through a central line to correct hypokalemia, but the rate of infusion was kept slow to avoid cardiac arrest. The initial echocardiographic assessment showed mild left ventricular dilatation, moderate systolic dysfunction with an ejection fraction of 35%, and right ventricular dilatation with normal arterial pressure (Fig. 3). Three days after hospitalization, short bursts of polymorphic tachycardia were observed on telemetry, which rapidly deteriorated to sinus bradycardia and asystole. He was resuscitated for 30 minutes. During resuscitation, ventilation was maintained with the mechanical ventilator in the intermittent mandatory ventilation mode. A total of 11 mg of adrenaline was given during resuscitation. Resuscitative efforts were halted after 30 minutes as no electrical activity was seen on the ECG. Mechanical ventilation was stopped and the endotracheal tube was removed. Five minutes after the cessation of CPR, an ECG showed asystole without any electrical activity (Fig. 1). However, the monitor electrodes were not removed from the patient, and 10 minutes after cessation of resuscitation a nurse noticed sinus rhythm on the monitor. As his pulse was palpable, he was intubated and mechanical ventilation was reinstituted. His blood pressure was 109/90 mmHg, and an ECG obtained a few hours after resuscitation showed findings similar to the initial ones (Fig. 1). Blood gas analysis obtained after stabilization of the patient showed mild acidosis with pH, 7.35; pCO_2, 40 mmHg; pO_2, 125 mmHg; and HCO_3, 22 mmol/L. Biochemical analyses obtained within 24 hours of recovery revealed persistently elevated transaminase levels and normal potassium levels (K, 4.01 mEq/L), while his renal function tests and urinary output were normal (creatinine, 0.14 mg/dL; blood urea nitrogen, 15 mg/dL; calculated creatinine clearance, 295.14 mL/minute). The patient regained consciousness 6 hours after resuscitation, and he was capable of understanding questions within 8 hours. A repeat echocardiogram showed findings similar to the initial one, with an ejection fraction of 25–30% and mild mitral regurgitation (Fig. 3). At follow up, his spontaneous breathing was inadequate for extubation, and a tracheostomy was performed on the 33rd hospital day (Fig. 2). During his hospitalization in the ICU,

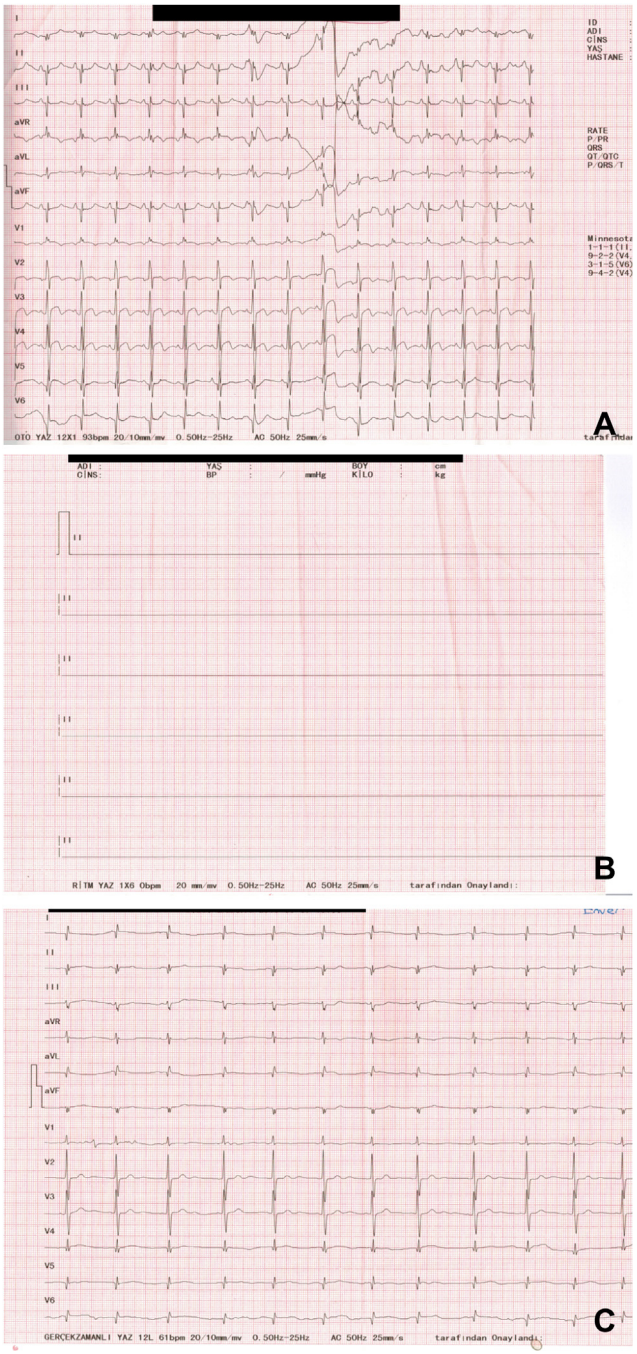

Fig. 1. Electrocardiograms (ECGs) of the patient (A) before, (B) during, and (C) after cardiac arrest. ECGs taken before and after the event show right ventricular enlargement and nonspecific ST–T segment alterations.

pseudomonas pneumonia and candidemia were diagnosed, and treated. On the 60th day of hospitalization, the patient was discharged with a home-type ventilator.

3. Discussion

As far as we know, this case is the first demonstration of LP in a patient with an underlying dilated cardiomyopathy. After asystolic cardiac arrest, patients with advanced heart disease are less responsive to CPR and have poorer outcomes compared