Case Report

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Stress Induced Cardiomyopathy from Acute Inflammatory Demyelinating Polyneuropathy

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Abstract

We report a case of a 70-year-old female with progressive lower extremity weakness and heaviness accompanied with chest pain. Her EKG did not show any ischemic changes, and her Troponin T was elevated to 0.42 ng/ml. A 2D echocardiogram showed a depressed left ventricular ejection fraction to 30% with severely hypokinetic anterior septum and left ventricular apex. She underwent neurophysiology testing with findings consistent with acute inflammatory demyelinating polyneuropathy, or Guillain-Barre Syndrome, which was treated with a 7-day course of intravenous immunoglobulin therapy to which she showed significant improvement in strength in her lower extremities. An echocardiogram was repeated 4 months later showing an improved left ventricular ejection fraction of 50% and no left ventricular wall motion abnormalities. Takotsubo cardiomyopathy is a rare complication of Guillain-Barre Syndrome and literature review revealed less than 10 cases have been reported. Due to the significant autonomic dysfunction seen in Guillain-Barre Syndrome, it could lead to arrhythmias, blood pressure changes, acute coronary syndrome and myocarditis,

Takotsubo cardiomyopathy can be difficult to distinguish. Dysregulation of autonomic tone with excessive sympathetic activation in Guillain-Barre Syndrome with elevated catecholamine levels is one hypothesis. The treatment of Takotsubo cardiomyopathy is supportive with beta-blockers and angiotensin-converting enzyme inhibitors are recommended until left ventricle ejection fraction improvement. Takotsubo cardiomyopathy is a rare complication during the acute phase of Guillain-Barre syndrome and must be distinguished from autonomic dysfunction as both diagnoses have different approaches to treatment.

Keywords: Takotsubo cardiomyopathy; Guillain-barre syndrome; Left ventricular dysfunction

Case Report

Guillain-Barre Syndrome is a relatively uncommon condition in which the body's immune system attacks part of the peripheral nervous system [1]. It can affect the nerves that control muscle movement, pain, temperature sensation and can be potentially fatal if it affects the respiratory muscles [2]. Very rarely has it been associated with Takotsubo cardiomyopathy, a transient cardiac syndrome that mimics acute coronary syndrome. Here, we present a case of a 70-year-old female who developed Takotsubo cardiomyopathy in association with Guillain-Barre Syndrome.

A 70-year-old female with a history of post-polio syndrome presented with progressive, worsening lower extremity weakness and heaviness accompanied with chest pain. On presentation she was hemodynamically stable, her EKG did not show any ischemic changes, and her Troponin T was elevated to 0.42 ng/ml. A 2D echocardiogram showed a depressed left ventricular ejection fraction to 30% with severely hypokinetic anterior septum and left ventricular apex. She underwent a cardiac catheterization, which did not demonstrate significant coronary artery disease and confirmed the hypokinetic left ventricular walls. She was started on carvedilol and enalapril. Simultaneously, an acute stroke was ruled out with a computed tomography of the head. She underwent neurophysiology testing with findings consistent with acute inflammatory demyelinating polyneuropathy, or Guillain-Barre Syndrome, which was treated with a 7 day course of intravenous immunoglobulin therapy to which she showed significant improvement in strength in her lower extremities. An echocardiogram was repeated 4 months later showing an improved left ventricular ejection fraction of 50% and no left ventricular wall motion abnormalities.

Takotsubo cardiomyopathy is a rare complication of Guillain-Barre Syndrome and literature review revealed less than 10 cases have been reported. Guillain-Barre Syndrome is an autoimmune process that affects the peripheral nervous system causing autonomic dysfunction which may involve the heart [3]. Due to the significant autonomic dysfunction seen in Guillain-Barre Syndrome, it could lead to arrhythmias, blood

pressure changes, acute coronary syndrome and myocarditis, Takotsubo cardiomyopathy can be difficult to distinguish. The criterion to diagnose Takotsubo cardiomyopathy include a transient hypokinesis, akinesis, or dyskinesis of the left ventricle wall with or without apical involvement in the absence of obstructive coronary artery disease, and is often related to a stressful trigger [4-5]. The pathogenesis in relation to Guillain-Barre Syndrome is also not well understood. Dysregulation of autonomic tone with excessive sympathetic activation in Guillain-Barre Syndrome with elevated catecholamine levels is one hypothesis [5]. The pathophysiology of Takotsubo cardiomyopathy seems to be from sympathetic excitation of brain triggering catecholamine release causing hyperdynamic basal contraction, and apical systolic dysfunction [4-6]. In some instances, intravenous immunoglobulin has been associated with the development of Takotsubo cardiomyopathy [6]. The treatment of Takotsubo cardiomyopathy is supportive with beta-blockers and angiotensin-converting enzyme inhibitors is recommended until left ventricle ejection fraction improvement. Takotsubo cardiomyopathy is a rare complication during the acute phase of Guillain-Barre syndrome and must be distinguished from autonomic dysfunction as both diagnoses have different approaches to treatment.

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