Reverse Takotsubo cardiomyopathy (RTC) occurs in 1-2% of patients presenting with acute coronary syndrome-like symptoms. Clinically, it presents similarly to Takotsubo cardiomyopathy (TC). Although, cardiac catheterization findings show apical hypokinesis and basal hypokinesis, as opposed to the apical hypokinesis and basal hypokinesis seen in classical Takotsubo syndrome. The scarcity of RTC occurrence and apparent rarity of the condition may lead to the assumption that RTC is uncommon. Our case is quite rare as there are only two peer-reviewed publications describing the coexistence of these two conditions is worth exploring with future studies, including correlations between dynamic electrocardiogram changes and rising blood pressure. A close look can be made at the remarkable fluctuations of ST changes, and increased QTC is important to do as early in the clinical course as possible in order to monitor closely for the evolution of possible stress-induced cardiomyopathy.

Our case is unique in that our patient did not have any other condition or factors mediating the relationship between RTC and hypertensive emergency. Thus, this case demonstrates a possible direct association between RTC and hypertensive emergency. Further studies exploring this association could provide new insight into the pathophysiology of RTC.

Introduction

The emergence of Takotsubo Syndrome as a recognized clinical entity can be traced back to Japan in the 1990's [1]. It was described after the autopsy of the left ventricle which resembled an “octopus trap”, in the first recognized and now most common form of the condition [1]. The clinical profile of the condition has been further defined by continued research over the last 30 years, we now know that more atypical variants do exist, notably the basal (or reverse), midventricular, and focal wall motion patterns [2]. The differences in the clinical profile as well as confirming factors of such patients is of profound significance in order to ensure timely diagnosis and treatment of these variants [3]. The association of reverse Takotsubo cardiomyopathy with hypertensive emergencies, an acute event characterized by marked elevation in blood pressure, was first described in 2015 in Japan [2]. It was traced back to Japan in the 1990's [1]. It was named after the shape of the left ventricle, which resembles an octopus trap. The association of reverse Takotsubo cardiomyopathy with hypertensive emergencies, an acute event characterized by marked elevation in blood pressure, was first described in 2015 in Japan [2]. It was traced back to Japan in the 1990's [1]. It was named after the shape of the left ventricle, which resembles an octopus trap.

Case Presentation

A 60-year-old woman with a past medical history significant for hypertension, hyperlipidemia, generalized anxiety disorder, major depressive disorder, post-traumatic stress disorder, and insomnia presented to the emergency department (ED) with complaints of abrupt retrosternal chest pain that started 30 minutes prior to admission. Chest pain was pressure-like, non-radiating, and seven out of ten in severity. She denied signs of angina, shortness of breath, nausea, chest discomfort, and diaphoresis. She denied any fevers, chills, cough, recent trauma, or recent travel history. Family history was non-contributory. She endorsed having numerous life stressors, including having a young son with special needs (autism spectrum disorder), a sudden death of her husband a year ago, and the death of her father after multiple hospitalizations, which contributed to a “psychotic breakdown” in her mother and subsequently led to an inpatient hospitalization. She described her recent stressors were marked and related to her work as a New York City police detective during the COVID-19 pandemic, including intrusive memories of past work as a New York City police detective during the COVID-19 pandemic, emotional stress related to the intrusive memories of past work as a New York City police detective during the COVID-19 pandemic, and then presented with hypertensive emergency, by means of intermittent catecholamine surges [10]. Our case is unique in that our patient did not have any other condition or factors mediating the relationship between reverse Takotsubo syndrome and hypertensive emergency. Thus, our case appears to be a unique case of a possible direct association between reverse Takotsubo physiology and hypertensive emergency.

Discussion

Our case was reportable because at its core, it was a diagnostic dilemma. Typically, when we as clinicians think about stress-induced cardiomyopathy, we think of the apical subtype and we look for clues that point to this diagnosis, like electrocardiogram (EKG) changes [3]. What made this case interesting was that we expected ST elevations in the electrocardiogram, and elevated left ventricular end diastolic pressure, both of which were absent in this case [3]. It is important to consider even the less common subtypes of a disease entity, especially in cases where the relevant history and diagnostic studies are at odds.

In the International Takotsubo registry study, patients with typical Takotsubo cardiomyopathy had a higher likelihood of ST segment depression, consistent with typical reverse Takotsubo cardiomyopathy, but ST-segment depression is also a defining feature of hypertensive emergencies, due to subendocardial ischemia with a sudden increase in afterload on the heart [5]. Thus, in our patient, EKG alone was not sufficient enough to point to a unifying diagnosis. Additionally, stress-induced cardiomyopathy is usually preceded by an acute emotional or physical stressor [6]. However, the presence of any identifiable acute stressor, such as in the case of our patient (all chronic emotional stressors), it is reasonable to explore the possibility that the acute stressor might have been the cause of our patient's reverse Takotsubo cardiomyopathy or merely an associated feature that confounds the picture is impossible to claim. However, the co-existence of these two conditions is worth exploring with future studies, including correlations between dynamic electrocardiogram changes and rising blood pressure. A close look can be made at the remarkable fluctuations of ST changes, and increased QTC is important to do as early in the clinical course as possible in order to monitor closely for the evolution of possible stress-induced cardiomyopathy.

References