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Review Article

Stress (Tako-tsubo) cardiomyopathy: Review

absence of obstructive epicardial coronary disease [1,2]. On left ventriculography, echocardiography or cardiac MRI, these functional abnormalities typically resemble a flask with a short, narrow neck and wide, rounded body. The shape of the ventricle at end systole resembles the Japanese fisherman's octopus pot—the tako-tsubo—from which the syndrome derives its original name. The hypercontractile basal myocardium can generate left ventricular outflow tract obstruction in the presence of apical and midwall hypokinesis. The final element of the syndrome is that left ventricular function and apical wall motion return to normal within days or weeks of the acute insult, in a similar manner to traditional myocardial stunning, providing no further acute cardiac events occur [3].

Although the basic cause of this condition is unresolved, the frequent association with stress has focused attention on the autonomic nervous system. It has been suggested that when powerful hormones such as adrenaline are released in excess, the heart muscle can be damaged in patients with takotsubo. In fact, events have been reported in patients after accidental overdose of adrenaline or associated with adrenaline-producing tumors (pheochromocytoma) [4]. Proposed mechanisms for catecholamine-mediated stunning in stress cardiomyopathy include epicardial spasm, microvascular dysfunction, hyperdynamic contractility with midventricular or outflow tract obstruction, and direct effects of catecholamines on cardiomyocytes [5]. The possibility of myocardial injury due to microvascular spasm has also been suggested [6], Ako and coworkers [7], by the use of an intracoronary Doppler wire technique, demonstrated microcirculation impairments in instances of transient LV hypocontraction. High levels of circulating epinephrine trigger a switch in intracellular signal trafficking in ventricular cardiomyocytes, from Gs protein to Gi protein signaling via the β_2 -adrenoceptor. Although this switch to β_2 -adrenoceptor-Gi protein signaling protects against the proapoptotic effects of intense activation of β_1 adrenoceptors, it is also negatively inotropic. This effect is greatest at the apical myocardium, in which the β -adrenoceptor density is greatest [5].

Presentation

The clinical presentation of stress cardiomyopathy is similar to that of an acute Myocardial Infarction (MI). The

Introduction

Stress cardiomyopathy, also referred to as Takotsubo cardiomyopathy, is an increasingly recognized clinical syndrome characterized by acute reversible apical ventricular dysfunction. Stress cardiomyopathy, also called apical ballooning syndrome, broken heart syndrome, takotsubo cardiomyopathy, and stress-induced cardiomyopathy, is generally characterized by transient systolic dysfunction of the apical and/or mid segments of the left ventricle that mimics myocardial infarction, but in the absence of obstructive coronary artery disease. In Japanese, "tako-tsubo" means "fishing pot for trapping octopus," and the left ventricle of a patient diagnosed with this condition resembles that shape. Takotsubo cardiomyopathy is not rare, and heightened awareness of this unique cardiomyopathy likely will lead to a higher reported incidence. Diagnosis of takotsubo cardiomyopathy has important implications for clinical management at presentation and afterward. The long-term prognosis is generally favorable; however, a small subset has potentially life-threatening complications during the initial presentation.

Etiopathogenesis

Stress cardiomyopathy can occur following a variety of emotional stressors such as grief, fear, extreme anger, and surprise. On the other hand numerous physical stressors such as stroke, seizure or acute asthma can also trigger the condition. Common presenting signs of this syndrome are chest pain, ST segment elevation in the precordial leads, mild elevation of cardiac enzyme and biomarker levels, and transient apical systolic left ventricular dysfunction in the



most common presenting symptom is acute substernal chest pain, but some patients present with dyspnea, syncope, shock, or electrocardiographic abnormalities. Acute complications of stress cardiomyopathy can include heart failure, tachyarrhythmias (including ventricular tachycardia and ventricular fibrillation), bradyarrhythmias, mitral regurgitation and cardiogenic shock [1,2,8,9]. Takotsubo cardiomyopathy should be considered in the differential diagnosis of acute myocardial infarction for a post-menopausal woman who presents with symptoms of myocardial ischemia after acute emotional or physical stress. For such patients, emergent transfer to a center with primary angioplasty capabilities may lead to the diagnosis of takotsubo cardiomyopathy, thereby avoiding the administration of fibrinolytic therapy and the potential, subsequent complications. However, such transfers should only be considered when transfer and angiography can be performed within recommended door-to-balloon guidelines [10]. Furthermore, Scott WS [11], revealed that the reoccurrence of takotsubo cardiomyopathy was around 10%.

Management

The management of stress cardiomyopathy mainly consists of supportive and symptomatic treatment [12]. We should be able to exclude any significant coronary artery disease [13]. Initially patients are managed as for myocardial infarction, including urgent coronary angiography with a view to primary coronary intervention. As is the case for patients with coronary artery disease, all patients should be started on aspirin, low molecular weight heparins, and Angiotensin-Converting Enzyme (ACE) inhibitors; β -blockers and diuretics can be started if needed. Recent study [14], suggested that the using angiotensin-converting-enzyme inhibitors or angiotensinreceptor blockers improved the 1-year prognosis significantly.

Beyond the standard supportive care for congestive heart failure with diuretics and vasodilators, the treatment of stress cardiomyopathy largely remains empirical. With good initial medical support, patients with stress cardiomyopathy show good clinical and echocardiographic improvement. They have an excellent prognosis.

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