Clinical manifestations and diagnosis of stress (takotsubo) cardiomyopathy

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INTRODUCTION — Stress cardiomyopathy (also called apical ballooning syndrome, takotsubo cardiomyopathy, broken heart syndrome, and stress-induced cardiomyopathy) is a syndrome characterized by transient regional systolic dysfunction of the left ventricle (LV), mimicking myocardial infarction, but in the absence of angiographic evidence of obstructive coronary artery disease or acute plaque rupture [1-16]. In most cases of stress cardiomyopathy, the regional wall motion abnormality extends beyond the territory perfused by a single epicardial coronary artery. The term “takotsubo” is taken from the Japanese name for an octopus trap, which has a shape that is similar to the systolic apical ballooning appearance of the LV in the most common and typical form of this disorder (image 1 and movie 1); mid and apical segments of the LV are depressed, and there is hyperkinesis of the basal walls. (See 'Approach to diagnosis' below.)

This topic will review the epidemiology, pathogenesis, clinical manifestations, and diagnosis of stress cardiomyopathy. The management and prognosis of stress cardiomyopathy is discussed separately. (See "Management and prognosis of stress (takotsubo) cardiomyopathy".)

EPIDEMIOLOGY — Stress cardiomyopathy was first described in 1990 in Japan and has since been increasingly recognized around the world [1,2,6,7,9,10,16,17]. Stress cardiomyopathy occurs in approximately 1 to 2 percent of patients presenting with troponin-positive suspected acute coronary syndrome (ACS) or suspected ST-elevation myocardial infarction [17-19]. A prevalence of 1.2 percent was reported from a registry of 3265 patients with troponin-positive ACS [17]. Similarly, stress cardiomyopathy accounted for 1.7 to 2.2 cases presenting with suspected ACS or ST-elevation infarction in a systematic review [18].

The incidence of stress cardiomyopathy among individuals exposed to physical or emotional stress is not known. A prospective study of 92 patients admitted to a medical intensive care unit with a non-cardiac diagnosis and no prior history of cardiac disease found that 26 patients (28 percent) had left ventricular (LV) apical ballooning consistent with stress cardiomyopathy [20]. LV function normalized in 20 of these patients at a mean of seven days. In multivariable analysis, sepsis was the only predictor of LV apical ballooning. The high incidence of transient LV apical ballooning in this series requires validation in larger series, but it appears that this phenomenon is not uncommon in a medical intensive care unit population.

Stress cardiomyopathy is much more common in women than men and occurs predominantly in older adults. [3,4,7,15-17]. In the International Takotsubo Registry (a consortium of 26 centers in Europe and the United States) of 1750 patients with stress cardiomyopathy, 89.9 percent were women and mean age was 66.4 years [16]. Similarly, in a review of 10 small prospective series, women accounted for 80 to 100 percent of cases, with a mean age of 61 to 76 years [15].

PATHOGENESIS — The pathogenesis of this disorder is not well understood. It is not known why this disorder affects postmenopausal women disproportionately or why the left ventricular (LV) mid-cavity and apex are predominantly affected. Studies comparing LV systolic and diastolic function in patients with stress

cardiomyopathy with function in patients with acute myocardial infarction (MI) have reached differing conclusions [21,22]. Initial systolic function may be similar [21] or worse [22] with stress cardiomyopathy compared with acute MI, while diastolic function may be similar [21] or better [22] with stress cardiomyopathy.

Postulated mechanisms include catecholamine excess [8,23], coronary artery spasm, and microvascular dysfunction. Alternatively, there may be dynamic mid-cavity or LV outflow tract obstruction that may contribute to apical dysfunction. Analogous permanent (rather than transient) apical outpouchings develop in patients with hypertrophic cardiomyopathy with mid-ventricular obstruction. (See "Types and pathophysiology of obstructive hypertrophic cardiomyopathy", section on 'Midcavity obstructive HCM'.)

Role of catecholamines — A number of features of stress cardiomyopathy, including its association with physical or emotional stress [4,5,7-10,16,20,24], suggest that this disorder may be caused by diffuse catecholamine-induced microvascular spasm or dysfunction, resulting in myocardial stunning [18], or by direct catecholamine-associated myocardial toxicity [25]. In some patients with stress cardiomyopathy, the only apparent stressor is exposure to catecholamine or beta-agonist drugs in routine clinical doses [26]. (See 'History' below and "Clinical syndromes of stunned or hibernating myocardium".)

Support for a possible pathogenic role for catecholamines comes from studies in which plasma catecholamines were measured at presentation [8,27-29]. Combining the results from these series, plasma norepinephrine levels were elevated in 26 of 35 patients (74 percent) [18]. Elevated catecholamine levels and reversible LV ballooning have also been observed in a rat model of immobilization-induced stress [30].

The magnitude of catecholamine excess associated with this disorder was illustrated in a report that measured plasma catecholamine levels in 13 patients with stress cardiomyopathy and seven patients with a Killip class III MI (table 1) [8]. Plasma catecholamines were significantly higher in the patients with stress cardiomyopathy as compared with those with MI: epinephrine (1264 versus 376 pg/mL) and norepinephrine (2284 versus 1100 pg/mL). However, elevation in blood catecholamine levels is not uniformly present and some studies have reported normal levels [31].

Further support for the catecholamine hypothesis is provided by observations of a similar reversible cardiomyopathy with global or focal dysfunction in patients with pheochromocytoma (see "Clinical presentation and diagnosis of pheochromocytoma") [32], and in the setting of acute brain injury, which has also been postulated to be related to catecholamine excess [33]. (See "Cardiac complications of stroke", section on 'Neurogenic cardiac damage'.)

The following observations support the hypothesis of catecholamine-induced myocardial effects:

- Limited available endomyocardial biopsy data [5,8,25] are consistent with histologic signs of catecholamine toxicity [34,35]. Findings have ranged from no evidence of myocarditis [36] to interstitial fibrosis with or without slight cellular infiltration [5] to mononuclear infiltrates with contraction band necrosis [8]. In a series of eight patients, acute biopsies obtained during the period of LV dysfunction revealed intracellular accumulation of glycogen, many vacuoles, disorganized cytoskeletal and contractile structure, contraction bands, and increased extracellular matrix proteins [25]. These alterations resolved nearly completely after functional recovery.

- In a mouse model, it has been demonstrated that a high level of epinephrine is negatively inotropic due to a switch from beta-2 adrenoreceptor-mediated Gs protein signaling, which is positively inotropic, to Gi protein signaling, which is negatively inotropic [37]. It is speculated that the greater effect at the apical myocardium may be due to a higher density of beta-adrenoreceptors at this location [38].

Role of coronary artery disease or dysfunction — Although the clinical presentation simulates that of an acute MI, coronary arteriography typically shows no obstructive lesions [4,7], and only a minority of patients display coronary spasm with acetylcholine provocation [4].

The following observations support the hypothesis of coronary vascular dysfunction, which may be catecholamine-induced:

- The occasional finding of multifocal coronary vasospasm on coronary angiography [4,7,36].
- Transient myocardial perfusion abnormalities that resolve with improvement in the myopathy [27].
- The presence of abnormal TIMI frame counts on angiography [10]. The TIMI frame count is the number of cine frames required for dye to first reach standardized distal coronary landmarks. (See "Fibrinolytic (thrombolytic) agents in acute ST elevation myocardial infarction: Markers of efficacy", section on 'TIMI frame count'.)

A potential role for plaque rupture and thrombosis with spontaneous thrombolysis has not been established and the results of intravascular ultrasound (IVUS) studies are mixed. Although one IVUS study found evidence of mid left anterior descending (LAD) coronary artery plaque rupture in five of five patients diagnosed with stress cardiomyopathy [39], other IVUS series found no evidence of culprit lesions in the LAD [40,41].

Some investigators have hypothesized that stress cardiomyopathy is not a distinct clinical entity, but rather a manifestation of aborted anterior MI in patients with a long "wrap-around" LAD [39]. Transient occlusion in such a vessel, with subsequent spontaneous thrombus lysis, could produce apical stunning and wall-motion abnormalities that would improve over follow-up. However, in one series, the prevalence of "wrap-around" LAD in stress cardiomyopathy was found to be low (27 percent) and comparable to that in patients diagnosed with anterior ST elevation MI [42].

Predisposing factors — Limited data are available on predisposing factors for stress cardiomyopathy. There have been reports of familial cases, raising the possibility of a genetic predisposition [43-45]. Small studies of patients with stress cardiomyopathy have found genetic heterogeneity and suggest a possible polygenic basis [46-49].

Patients with psychiatric and/or neurologic disorders may be predisposed to develop stress cardiomyopathy [16,50]. In the International Takotsubo Registry study, 55.8 percent of patients with stress cardiomyopathy had an acute, former, or chronic psychiatric (such as affective or anxiety disorder) or neurologic disorder (such as seizure or headache disorder) as compared with 25.7 percent of patients with ACS [16].

CLINICAL MANIFESTATIONS — The clinical presentation of stress cardiomyopathy is similar to that of an acute coronary syndrome (ST-elevation myocardial infarction [MI], non-ST elevation MI, or unstable angina) [3,4,7,16].

History — The onset of stress cardiomyopathy is frequently but not always triggered by intense emotional or physical stress (eg, death of relatives, particularly if unexpected, domestic abuse, arguments, catastrophic medical diagnoses, devastating financial or gambling losses, natural disasters, or acute medical illness) [4,5,7-10,24].

Among the 1759 patients in the International Takotsubo Registry study, 36 percent had a physical trigger (such as acute respiratory failure, post-surgical/fracture, central nervous system condition, or infection), 27.7 reported an emotional trigger (such as grief/loss, panic/fear/anxiety, interpersonal conflict, anger/frustration, financial or employment problem), 7.8 percent had both physical and emotional triggers, and 28.5 percent had no evident trigger [16].

Similarly, a systematic review including 19 studies with a total of 1109 patients found that emotional stressors were present in 39 percent of patients and physical stressors in 35 percent [51].

In addition, patients with psychiatric or neurologic disorders may be predisposed to develop stress cardiomyopathy. (See 'Pathogenesis' above.)

Symptoms and signs — The most common presenting symptom is acute substernal chest pain, but some patients present with dyspnea or syncope. In the International Takotsubo Registry study, the most common symptoms were chest pain, dyspnea, and syncope (75.9, 46.9, and 7.7 percent, respectively) [16].

Some patients develop symptoms and signs of heart failure, tachyarrhythmias (including ventricular tachycardia and ventricular fibrillation), bradyarrhythmias, sudden cardiac arrest, or significant mitral

regurgitation [3,4,7,9,52]. Approximately 10 percent of patients with stress cardiomyopathy develop symptoms and signs of cardiogenic shock (such as hypotension, abnormal mental status, cold extremities, oliguria, or respiratory distress) [16].

Left ventricular outflow tract obstruction, induced by left ventricular basal hyperkinesis produces a late peaking systolic murmur, similar to that heard in patients with hypertrophic cardiomyopathy and can contribute to the development of shock and cause severe mitral regurgitation [53]. (See "Auscultation of cardiac murmurs in adults", section on 'Subvalvular outflow obstruction' and "Management and prognosis of stress (takotsubo) cardiomyopathy", section on 'With left ventricular outflow tract obstruction'.)

Symptoms and signs of transient ischemic attack or stroke may develop (likely due to embolization from apical thrombus) [26,54].

Test results

Electrocardiogram — Electrocardiographic abnormalities are common in patients with stress cardiomyopathy.

- ST segment elevation is frequent (eg, in 43.7 percent of patients in the International Takotsubo Registry study [16]). ST segment elevation occurs most commonly in the anterior precordial leads and often is similar to that seen with an acute ST-elevation MI [55].
- ST depression is a less common finding (eg, occurring in 7.7 percent [16]) among patients with stress cardiomyopathy
- Other findings include QT interval prolongation, T wave inversion, abnormal Q waves, and non-specific abnormalities [4,8,16,56].

Cardiac biomarkers — Serum cardiac troponin levels are elevated in most patients with stress cardiomyopathy (eg, median initial troponin 7.7 times the upper limit of normal with interquartile range 2.2 to 24 in the International Takotsubo Registry study [16]), while creatine kinase levels are generally normal or mildly elevated (eg, median creatine kinase 0.85 times the upper limit of normal with interquartile range of 0.52 to 1.48) [18]. The normal to mild elevation in creatine kinase contrasts with the substantial (approximately 10 percent) risk of severe hemodynamic compromise.

Natriuretic peptides — Levels of brain natriuretic peptide (BNP) or N-terminal pro-BNP are elevated in most patients with stress cardiomyopathy [16,57]. As an example, BNP levels were elevated in 82.9 percent of patients with stress cardiomyopathy in the International Takotsubo Registry study, with median level 6.12 times the upper limit of normal (interquartile range 2.12 to 15.70) [16]. BNP levels in a matched cohort of patients with stress cardiomyopathy exceeded those seen in matched cohort of patients with acute coronary syndrome (median 5.89 versus 2.91 times the upper limit of normal).

Radionuclide myocardial perfusion imaging — Radionuclide myocardial perfusion imaging is generally not indicated in patients presenting with suspected stress cardiomyopathy since most have high-risk features for acute coronary syndrome (including elevated cardiac troponin levels) and will require coronary angiography. Patients with suspected non-ST elevation acute coronary syndrome with low- or intermediate-risk features may undergo radionuclide myocardial perfusion imaging as discussed separately. Transient perfusion abnormalities in the left ventricular apex have been documented in patients with stress cardiomyopathy [3,5]. (See "Acute rest radionuclide myocardial perfusion imaging for the evaluation of suspected non-ST elevation acute coronary syndrome" and "Noninvasive imaging and stress testing in patients with suspected acute coronary syndrome".)

DIAGNOSIS

Approach to diagnosis — The diagnosis of stress cardiomyopathy should be suspected in adults (particularly postmenopausal women) who present with a suspected acute coronary syndrome (with symptoms such as chest pain or dyspnea in combination with electrocardiographic changes and/or cardiac troponin elevation), particularly when the clinical manifestations and electrocardiographic abnormalities are

out of proportion to the degree of elevation in cardiac biomarkers [11]. A physical or emotional trigger is often but not always present. (See 'History' above.)

We use the following proposed Mayo Clinic diagnostic criteria, all four of which are required for the diagnosis [3,14]:

- Transient left ventricular systolic (LV) dysfunction (hypokinesis, akinesis, or dyskinesis). The wall motion abnormalities are typically regional and extend beyond a single epicardial coronary distribution; rare exceptions are the focal (within one coronary distribution) and the global type.
- Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture. If coronary disease is found, the diagnosis of stress cardiomyopathy can still be made if the wall motion abnormalities are not in the distribution of the coronary disease. This exception is made since some patients with stress cardiomyopathy have concurrent coronary disease (15.3 percent in the International Takotsubo registry [16]).
- New electrocardiographic abnormalities (either ST-segment elevation and/or T wave inversion) **or** modest elevation in cardiac troponin.
- Absence of pheochromocytoma or myocarditis.

Thus, a diagnosis of stress cardiomyopathy generally requires coronary angiography, serial assessment of LV systolic function (initial assessment generally by ventriculography or echocardiography with subsequent assessment generally by echocardiography), an electrocardiogram, and cardiac troponin level. Differentiation from pheochromocytoma and myocarditis is discussed below. (See 'Differential diagnosis' below.)

An important issue is how the possible diagnosis of stress cardiomyopathy should influence the evaluation of a suspected acute coronary syndrome (ST elevation myocardial infarction [MI], non-ST elevation MI, or unstable angina). We suggest the following approach:

- Patients presenting with ST elevation who can undergo urgent cardiac catheterization for the purpose of primary percutaneous coronary intervention (PCI) should proceed with angiography in the usual manner. If the patient has stress cardiomyopathy, angiographic findings will suggest the diagnosis by showing no critical coronary disease and the presence of apical ballooning (or mid wall hypokinesis) on left ventricular angiography. (See "Primary percutaneous coronary intervention in acute ST elevation myocardial infarction: Determinants of outcome".)
- Patients with ST elevation meeting criteria for reperfusion therapy presenting in a setting without availability of urgent angiography and PCI are usually treated with fibrinolytic therapy. In such cases, suspicion of the diagnosis of stress cardiomyopathy is not a sufficient reason to withhold fibrinolytic therapy since the majority of patients with acute ST elevation will have a critical coronary lesion. (See "Fibrinolysis for acute ST elevation myocardial infarction: Initiation of therapy".)

  In this later scenario, the diagnosis of stress cardiomyopathy may later be suggested by such clinical features as the absence of critical stenoses on coronary angiography, modest cardiac enzyme elevations, and recovery of LV function. However, none of these features is diagnostic, as they may also reflect successful early fibrinolysis.
- Patients who present without ST elevation will usually fit into a "high-risk" non-ST elevation MI profile (positive troponin, elderly, significant LV dysfunction). Early (less than 48 hours) cardiac catheterization will be performed in most such patients, which should help differentiate an acute coronary syndrome from stress cardiomyopathy. (See "Coronary angiography and revascularization for unstable angina or non-ST elevation acute myocardial infarction".)

Exclusion of coronary artery disease — Coronary angiography typically demonstrates either normal vessels or mild to moderate coronary atherosclerosis. Obstructive coronary artery disease is seen in some patients with stress cardiomyopathy which likely reflects the prevalence of coronary artery disease in the

population at risk for stress cardiomyopathy [19,42]. In the International Takotsubo Registry study, 15.3 percent of patients with stress cardiomyopathy had concurrent coronary artery disease detected by coronary angiography [16].

Identification of wall motion abnormalities — LV dysfunction is identified by echocardiography or left ventriculography, which reveals regional wall motion abnormalities (hypokinesis, akinesis, or dyskinesis) in one of the characteristic patterns [3-5,7,9,16,17,27]:

- Apical type - In the typical form of this disorder, there is systolic apical ballooning of the LV (image 1 and movie 1), reflecting depressed mid and apical segments, and there is hyperkinesis of the basal walls. This type was present in 81.7 percent of patients in the International Takotsubo Registry study [16].
- Less common (atypical) variants:
    - Mid-ventricular type - In the second most common type, ventricular hypokinesis is restricted to the mid-ventricle with relative sparing of the apex [17,58]. This type was present in 14.6 percent of patients in the International Takotsubo Registry study.
    - Basal type - Hypokinesis of the base with sparing of the mid-ventricle and apex (reverse or inverted Takotsubo). This type was present in 2.2 percent of patients in the International Takotsubo Registry study.
    - Focal type - A rare focal variant is characterized by dysfunction of an isolated segment (most commonly the anterolateral segment) of the LV [16]. This type was present in 1.5 percent of patients in the International Takotsubo Registry study.
    - Global type – Rarely, patients have global hypokinesis [59].

Most patients with stress cardiomyopathy have reduced overall LV systolic function [4,7,8,18] (eg, mean LV ejection fraction [LVEF] 41 percent [16]). Although reports of stress cardiomyopathy have focused on transient dysfunction of the LV, there is evidence that about one-third of cases involve both right and left ventricles [58,60-62]. (See 'Cardiovascular magnetic resonance' below.)

Additional tests to exclude other conditions — Additional testing is suggested when the diagnosis of stress cardiomyopathy is uncertain.

Cardiovascular magnetic resonance — Cardiovascular magnetic resonance (CMR) may be helpful in the diagnosis and evaluation of stress cardiomyopathy, particularly when the echocardiogram is technically suboptimal and/or there is coexistent coronary artery disease. CMR may assist in the differential diagnosis, delineate the full extent of ventricular abnormalities, and identify associated complications.

The following are key CMR features of stress cardiomyopathy:

- Late gadolinium enhancement (LGE) on CMR imaging is generally absent in stress cardiomyopathy in contrast to MI in which intense (ie, >5 standard deviations above the mean signal intensity of remote myocardium) subendocardial or transmural LGE is seen [5,7,11,46,47,58,63]. LGE is also useful in differentiating stress cardiomyopathy from myocarditis, which is characterized by patchy late gadolinium enhancement. However, when a low threshold for LGE is used (eg, three standard deviations above the mean signal intensity of remote myocardium), LGE is occasionally detected in stress cardiomyopathy [58]. (See "Clinical utility of cardiovascular magnetic resonance imaging", section on 'Late gadolinium enhancement'.)
- CMR evidence of myocardial edema is commonly seen in stress cardiomyopathy. However, myocardial edema is also seen in acute MI and myocarditis. In one series, 81 percent of patients had evidence of focal myocardial edema on CMR and these regions corresponded to areas of wall motion abnormality [58].
- CMR may also enable identification of thrombus in the left or right ventricle, which may not be detected

by echocardiography [26].

The frequency and significance of right ventricular involvement was illustrated in a series of 34 patients with stress cardiomyopathy who underwent CMR imaging [60]. Nine patients (26 percent) had right ventricular wall motion abnormalities. Patients with right ventricular dysfunction had lower LVEF compared with patients with normal right ventricular function (40 versus 48 percent) and were also more likely to have pleural effusions. At a mean of one year after presentation, follow-up CMR imaging showed improvement or resolution of right ventricular dysfunction in eight of nine patients.

Positron emission tomography — Although data are limited, positron emission tomography may be helpful in confirming the diagnosis of stress cardiomyopathy. Small studies of patients with stress cardiomyopathy undergoing myocardial positron emission tomography have identified a discrepancy between normal perfusion and reduced glucose utilization in dysfunction regions, known as an “inverse flow metabolism mismatch” [64]

DIFFERENTIAL DIAGNOSIS — As noted above, the clinical presentation of stress cardiomyopathy is similar to that of acute coronary syndrome (with or without ST elevation). These conditions are differentiated by angiography, which demonstrates critical disease in the coronary artery supplying the dysfunctional ventricular territory in patients with acute coronary syndrome; such critical coronary artery disease or evidence of acute plaque rupture is lacking in patients with stress cardiomyopathy. While some patients with stress cardiomyopathy have concurrent significant coronary artery disease, the extent and location of such disease does not match the territory of the observed wall motion abnormalities.

A number of syndromes other than stress cardiomyopathy have been associated with ST segment changes in the absence of significant coronary artery disease, including acute coronary syndrome related to cocaine abuse and myocarditis. As noted above, similar reversible cardiomyopathy with global or focal dysfunction has been observed in patients with pheochromocytoma and in the setting of acute brain injury [32,33].

- Cocaine-related acute coronary syndrome is suggested by history of exposure, which can be confirmed if needed by toxicology assays for the drug or its metabolites. (See "Evaluation and management of the cardiovascular complications of cocaine abuse", section on 'Evaluation of cocaine-induced chest pain'.)
- Myocarditis may present with regional wall motion abnormalities and troponin elevation similar to stress cardiomyopathy, although the pattern of wall motion abnormality generally differs and recovery of function is generally less rapid than with stress cardiomyopathy. Cardiovascular magnetic resonance imaging may be helpful as a means of excluding myocardial inflammation and scar in patients with stress cardiomyopathy [65]. (See "Clinical manifestations and diagnosis of myocarditis in adults", section on 'Diagnosis' and 'Cardiovascular magnetic resonance' above.)
- Pheochromocytoma is suspected in patients with symptoms such as headache, sweating, and tachycardia with or without hypertension. (See "Clinical presentation and diagnosis of pheochromocytoma", section on 'Approach to initial evaluation'.)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topic (see "Patient information: Stress cardiomyopathy (The Basics)")

SUMMARY AND RECOMMENDATIONS

- Stress cardiomyopathy is a syndrome characterized by transient regional left ventricular dysfunction in the absence of significant coronary artery disease. (See 'Introduction' above and 'Approach to diagnosis' above.)
- Postulated pathogenic mechanisms include catecholamine excess, microvascular dysfunction, and multivessel coronary artery spasm. (See 'Pathogenesis' above.)
- The diagnosis of stress cardiomyopathy should be suspected in adults who present with a suspected acute coronary syndrome (with symptoms such as chest pain or dyspnea in combination with electrocardiographic changes and/or cardiac troponin elevation), particularly when the clinical manifestations and electrocardiographic abnormalities are out of proportion to the degree of elevation in cardiac biomarkers. A physical or emotional trigger is often but not always present. (See 'Clinical manifestations' above and 'Approach to diagnosis' above.)
- Diagnostic criteria include presence of transient regional wall motion abnormalities (typically not in a single coronary distribution), absence of angiographic evidence of obstructive coronary disease or acute plaque rupture, presence of new electrocardiographic abnormalities or modest troponin elevation, **and** absence of pheochromocytoma or myocarditis. (See 'Diagnosis' above.)
- In patients who present with a clinical picture consistent with acute coronary syndrome (ACS, such as ST elevation myocardial infarction, non-ST elevation myocardial infarction, or unstable angina), clinical suspicion of possible stress cardiomyopathy should not alter evaluation and management of these ACS conditions. The significant majority of these cases are due to occlusion of a coronary artery and revascularization therapy should not be delayed. (See 'Approach to diagnosis' above.)
- Wall motion abnormalities in patients with stress cardiomyopathy are typically detected by echocardiography or left ventriculography. Patterns of left ventricular wall motion abnormality in patients with stress cardiomyopathy include the apical type (which occurs in the most cases), and atypical variants including mid-ventricular, basal, focal (limited to an isolated segment), and global types. (See 'Identification of wall motion abnormalities' above.)
- The differential diagnosis of stress cardiomyopathy includes ACS, cocaine-related ACS, myocarditis, and pheochromocytoma. (See 'Differential diagnosis' above.)

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