

Systematic Review: Transient Left Ventricular Apical Ballooning: A Syndrome That Mimics ST-Segment Elevation Myocardial Infarction

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The transient left ventricular apical ballooning syndrome, also known as takotsubo cardiomyopathy, is characterized by transient wall-motion abnormalities involving the left ventricular apex and mid-ventricle in the absence of obstructive epicardial coronary disease. In this paper, we review case series that report on patients with the transient left ventricular apical ballooning syndrome to better characterize patients presenting with the syndrome.

We identified 7 case series that reported on at least 5 consecutive patients with the transient left ventricular apical ballooning syndrome. The syndrome more often affects postmenopausal

women (82% to 100%) (mean age, 62 to 75 years). Patients commonly present with ST-segment elevation in the precordial leads, chest pain, relatively minor elevation of cardiac enzyme and biomarker levels, and transient apical systolic left ventricular dysfunction despite the absence of obstructive epicardial coronary disease. An episode of emotional or physiologic stress frequently precedes presentation with the syndrome. The in-hospital mortality rate seems to be low, as does the risk for recurrence.

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The transient left ventricular apical ballooning syndrome, also known as takotsubo cardiomyopathy, is a recently described novel acute cardiac syndrome. The syndrome is characterized by peculiar, yet characteristic, transient regional systolic dysfunction involving the left ventricular apex and mid-ventricle with hyperkinesis of the basal left ventricular segments. The syndrome was initially recognized and reported in the Japanese population (1–6). Dote and colleagues (1) were one of the first to describe the syndrome, which was given the moniker *tako-tsubo-like left ventricular dysfunction*, naming it after a round-bottomed narrow-necked Japanese fishing pot used for trapping octopus (Figure 1, parts A, B, and C). More recently, the condition has been called *transient left ventricular apical ballooning syndrome* in reference to the associated left ventricular morphologic features that accompany the syndrome. After the initial recognition and description of the transient left ventricular apical ballooning syndrome in the Japanese population, subsequent recent reports have documented the syndrome in the United States (7) and Belgium (8). Despite the acute onset of transient left ventricular systolic dysfunction involving the left ventricular apex and mid-ventricle, patients with the transient left ventricular apical ballooning syndrome do not have obstructive atherosclerotic coronary disease. The cause of the syndrome is unknown.

Available case series of the transient left ventricular apical ballooning syndrome have included relatively few patients. This review presents reported case series of the syndrome in order to more precisely summarize the demographic characteristics, clinical characteristics, and outcomes of patients presenting with the syndrome. In addition, we review the available literature evaluating possible pathophysiologic mechanisms responsible for the syndrome.

METHODS

Literature Search and Identification of Relevant Studies

We identified relevant English-language articles pertaining to the transient left ventricular apical ballooning syndrome by searching the PubMed and EMBASE databases (through June 2004). The following search terms were used to identify primary articles: *left ventricular apical ballooning syndrome*, *takotsubo cardiomyopathy*, *ampulla cardiomyopathy*, and *transient left ventricular dysfunction*. We then manually searched references from the primary articles. We included only peer-reviewed reports and did not search for unpublished data. When more than 1 case series was reported from the same medical center, we included the case series reporting on the largest number of patients and excluded those with fewer patients to avoid duplicate reporting. Only case series reporting on at least 5 consecutive patients with the transient left ventricular apical ballooning syndrome were initially identified and assessed for potential inclusion in this case series review. Case reports and small case series reporting on fewer than 5 patients were not included in the review in an attempt to minimize potential reporting bias of uncharacteristic or nonrepresentative cases. We included prospective and retrospective case series. Figure 2 outlines the search and selection process.

Case Series and Data Assessment

We identified 13 case series for potential inclusion in this review. Of these, 10 reported on unselected, consecutive patients with the transient left ventricular apical ballooning syndrome. Seven of these 10 case series reported on patients who met the following criteria and were subsequently included in this review. First, the patient must have had transient apical and mid-left ventricular wall-motion abnormalities resulting in the left ventricular morphologic appearance of “apical ballooning.” Second, coronary angiography data that excluded obstructive

atherothrombotic coronary artery disease as the causative mechanism for left ventricular dysfunction had to be available. Third, the study had to report on patient demographic characteristics, presenting symptoms, clinical presentation, electrocardiographic characteristics, laboratory data, cardiac catheterization data, clinical complications, and clinical outcome. Of the 3 excluded series reporting on consecutive patients, 2 were excluded because they lacked coronary angiography data for all patients (9, 10), and 1 was excluded because it reported on only 2 patients (11). In addition, data that addressed potential pathophysiologic mechanisms responsible for the transient left ventricular apical ballooning syndrome were identified by using the search strategy described earlier. These data were then reviewed and summarized. One author read and assessed all studies.

RESULTS

Table 1 summarizes the 7 case series that met the inclusion criteria for this review. Of these, 5 reports were from Japan (2–6), 1 was from the United States (7), and 1 was from Belgium (8). Three studies reported prospectively collected data, 2 studies reported retrospective data, and 2 studies did not specify the prospective or retrospective nature of the study.

Demographic Characteristics and Presenting Symptoms

All 7 case series consistently reported a sex discrepancy in patients presenting with the transient left ventricular apical ballooning syndrome: Most patients were women (range, 82% to 100%). The mean age of patients presenting with the syndrome was 62 to 75 years (overall range, 10 to 88 years). Patients often presented with chest pain at rest (33% to 71%), although dyspnea as the initial symptom was not uncommon. Isolated cases of syncope as the presenting symptom have been reported (2, 4, 5, 7).

Electrocardiographic Data

The most common finding on the admission electrocardiogram was ST-segment elevation (range, 46% to 100% of patients). All series except 1 reported ST-segment elevation in at least 81% of patients. ST-segment elevation, when present, was most often reported in the precordial leads. Concomitant ST-segment elevation in the inferior leads has been reported in a few cases, and isolated inferior or lateral ST-segment elevation seems to be an unusual electrocardiographic finding in patients presenting with the transient left ventricular apical ballooning syndrome. New left and right bundle-branch block on the presenting electrocardiogram has been reported. Almost all series patients developed evolutionary T-wave inversions that were usually present in most leads. New pathologic Q waves on the electrocardiogram were reported in 6% to 31% of patients; in some instances, the Q waves were transient. The corrected QT interval on the presenting electrocardiogram

Key Summary Points

The transient left ventricular apical ballooning syndrome is a novel cardiac syndrome. It is characterized by peculiar transient apical “ballooning” of the left ventricle, which is the result of characteristic wall-motion abnormalities in the left ventricular apex and mid-ventricle.

Despite the absence of obstructive epicardial coronary artery disease, clinical presentation in patients with the syndrome is similar to that of patients with ST-segment elevation myocardial infarction.

Postmenopausal women seem to be most at risk for developing the syndrome.

An episode of acute emotional or physiologic stress seems to often precede presentation with the syndrome.

Patients with the syndrome should be monitored and treated for left heart failure, dynamic intraventricular obstruction, arrhythmias, and mechanical complications, should they develop.

Patients with the syndrome seem to have a favorable in-hospital prognosis despite the development of acute left-sided heart failure and hemodynamic instability in many patients.

The cause of the syndrome is not yet known.

was prolonged; it ranged from a mean of 450 milliseconds to 501 milliseconds.

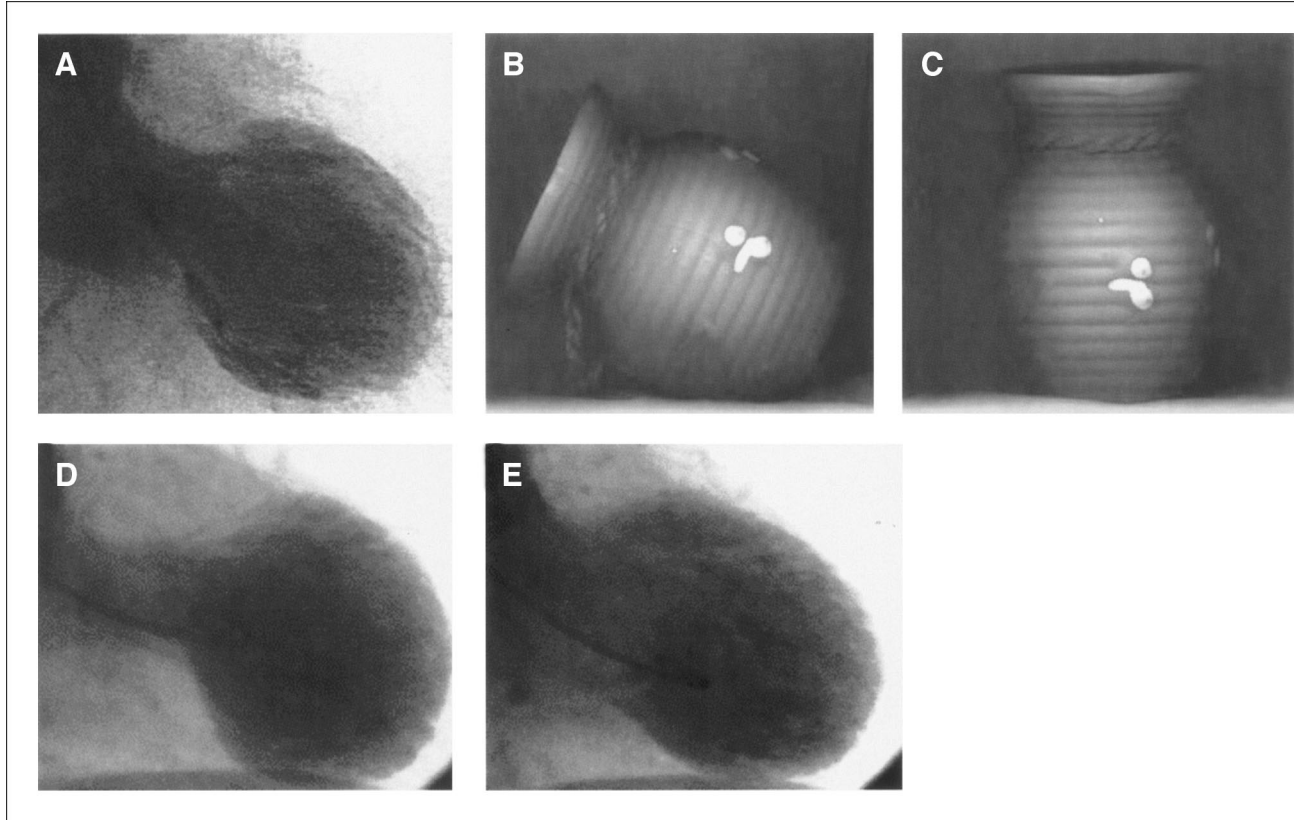
Cardiac Enzyme and Biomarker Release

Most patients had a small, rapid increase in cardiac enzyme and biomarker levels. Fifty-six percent to 100% of patients had a peak cardiac enzyme or biomarker level above the upper limit of normal. The series reporting a 56% incidence of cardiac enzyme increase assessed only creatine kinase (3), whereas the 2 series reporting a 100% incidence of biomarker release evaluated cardiac troponin (7, 8). Several series have reported that peak cardiac biomarker levels are often those drawn at the time of initial presentation; they appear to not follow the slow rise-and-fall kinetics observed with conventional myocardial infarction.

Angiographic Data and Left Ventricular Function

Patients with the transient left ventricular apical ballooning syndrome had either no angiographically detectable coronary disease (range, 25% to 100% of patients) or nonobstructive coronary disease (range, 0% to 75% of patients). None of the patients had epicardial stenosis greater than 50% of the luminal coronary artery diameter. Patients with the syndrome had an abnormal left ventricular ejection fraction at presentation (mean, 0.39 to 0.49) that improved rapidly over a period of days to weeks (mean follow-up left ventricular ejection fraction, 0.60 to 0.76). Apical and mid-ventricular regional wall-motion abnor-

Figure 1. Left ventriculograms (end-systole) of 2 patients with the transient left ventricular apical ballooning syndrome and examples of a tako-tsubo.



A. Note the characteristic appearance with “apical ballooning.” B and C. A tako-tsubo; note the similarities in appearance with the morphologic appearance of the left ventricle during systole. D and E. Left ventriculograms of another patient with the syndrome were obtained by cardiac catheterization during systole (D) and diastole (E). The photograph taken during systole demonstrates the characteristic left ventricular “apical ballooning” seen with the syndrome. Coronary angiography revealed no evidence of coronary artery disease. An echocardiogram obtained 30 days after presentation showed complete resolution of the left ventricular wall-motion abnormalities. Part A is adapted from and parts B and C are reprinted from *American Heart Journal*, volume 143, Kurisu S, Sato H, Kawagoe T, Ishihara M, Shimatani Y, Nishioka K, et al., Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction, pages 448-455, 2002, with permission from Elsevier.

malities completely resolved in most patients and followed a time course similar to that of the associated improvement in left ventricular ejection fraction. Four studies reported endomyocardial biopsy results in the acute phase of the syndrome ($n = 18$), with no evidence of myocarditis (2, 4–6).

Evaluations of Endothelial Function and Coronary Microcirculation

Either spontaneous or provokable multivessel epicardial spasm was present in a few patients; the incidence of provokable multivessel epicardial spasm ranged from 0% to 43% in different series. Coronary flow reserve, which assesses coronary microvascular function, has been reported in a few patients, and results were conflicting (6, 12). Frame counts from the Thrombolysis in Myocardial Infarction (TIMI) study, a method of quantitating the time required for injected intracoronary contrast to reach predefined distal landmarks, were reported in 2 series. Both series report abnormal TIMI frame counts in all 3 major

epicardial coronary arteries during the acute phase of the syndrome; this finding suggests widespread coronary microvascular dysfunction (5, 7).

Clinical Complications and Outcome

Table 2 summarizes reported clinical complications of the transient left ventricular apical ballooning syndrome. The overall prognosis of patients presenting with the syndrome seems to be favorable; reported in-hospital mortality rates range from 0% to 8%. The largest series, which included 88 patients, reported an in-hospital mortality rate of 1%. Pulmonary edema or significant left-sided heart failure during the acute phase was reported in 3% to 46% of patients, and some patients required insertion of an intra-aortic balloon pump. A transient, dynamic intraventricular pressure gradient due to obstruction in the left ventricular cavity can develop as a result of dyskinetic apical and mid-ventricular segments with hyperdynamic function of the basal segments. This complication was documented

in 13% to 18% of patients and can be accompanied by mitral regurgitation due to systolic anteromotion of the mitral valve leaflets and chordal apparatus. Although ventricular tachycardia and ventricular fibrillation have been reported at the time of presentation and as delayed complications of the syndrome, they seem to be infrequent. Isolated cases of left ventricular mural thrombus formation have been reported (13). One case of left ventricular free-wall rupture occurred 3 days after presentation with the syndrome (14).

Preceding Stressors, Incidence, and Recurrence of the Syndrome

Most reported patients with the transient left ventricular apical ballooning syndrome presented immediately after an episode of acute emotional (range, 14% to 38% of patients) or physiologic (range, 17% to 77%) stress (such as acute medical illness or surgery). Few data have evaluated the frequency of the syndrome. In 1 series, the syndrome represented an estimated 2.2% of ST-segment elevation acute coronary syndromes presenting to a referral hospital during 2002 and 2003 (7). A separate series reported that the syndrome was ultimately diagnosed in 1.5% of patients who had presented with an apparent Q-wave acute coronary syndrome (4). Recurrence of the syn-

drome seems to be rare and has been documented in 0% to 8% of series patients.

DISCUSSION

The transient left ventricular apical ballooning syndrome is a unique disorder that has only recently been generally appreciated. In most reported cases, the syndrome mimics ST-segment elevation acute myocardial infarction because patients present with chest pain or dyspnea, have electrocardiographic ST-segment elevation, and have elevated cardiac biomarker levels. Unlike most patients presenting with ST-segment elevation acute myocardial infarction, patients with the transient left ventricular apical ballooning syndrome present with these changes in the absence of obstructive epicardial coronary atherosclerosis. The clinical characteristics of the syndrome are consistent across series and include the acute onset of ischemic-like chest pain or dyspnea, characteristic transient apical and mid-ventricular regional wall-motion abnormalities, minor elevations of cardiac enzyme and biomarker levels, and electrocardiographic ST-segment changes with QT interval prolongation and evolutionary T-wave inversions.

Reports of the transient left ventricular apical balloon-

Figure 2. Study identification.

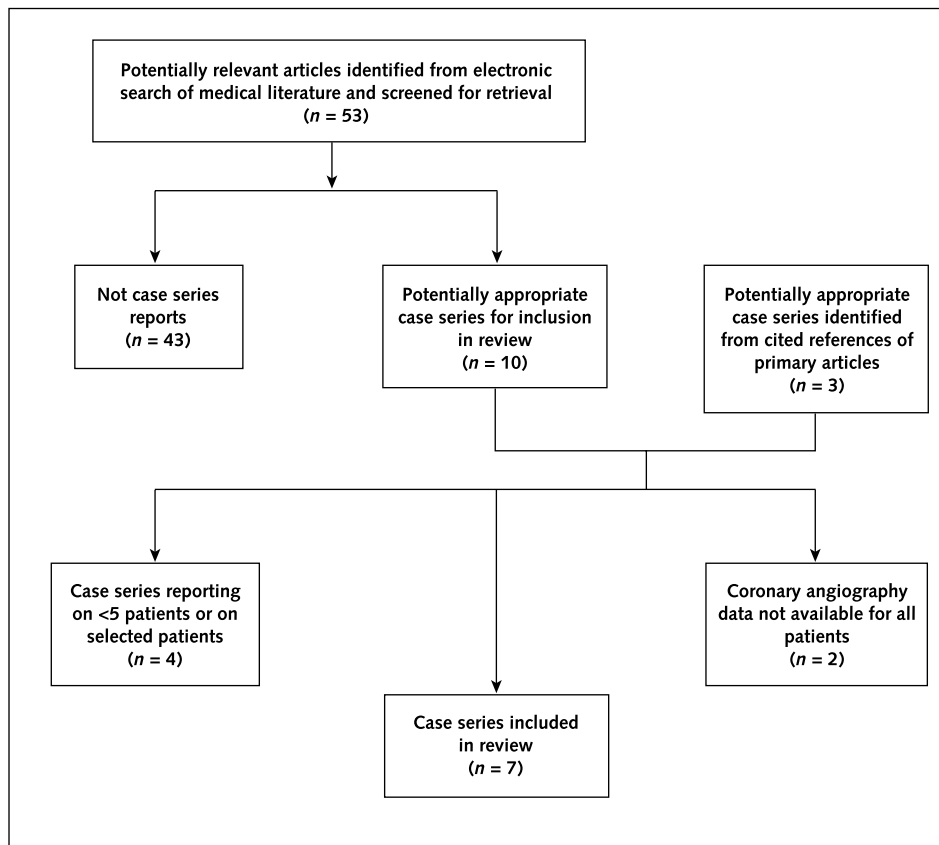


Table 1. Demographic and Clinical Characteristics of Consecutive Patients Given a Diagnosis of the Transient Left Ventricular Apical Ballooning Syndrome*

Characteristic	Tsuchihashi et al. (3) (n = 88)	Kurusu et al. (5) (n = 30)	Abe et al. (6) (n = 17)	Bybee et al. (7) (n = 16)	Desmet et al. (8) (n = 13)	Kawai et al. (2) (n = 9)	Akashi et al. (4) (n = 7)
Country	Japan	Japan	Japan	United States	Belgium	Japan	Japan
Series type	Retrospective	Retrospective	Prospective	Prospective	Unspecified	Unspecified	Prospective
Age, y†	67 ± 13	70 ± 8	74	71 ± 12	62	66	75
Women, %	86	93	82	100	92	89	86
Chest pain, %	67	67	53	69	62	33	71
ST-segment elevation, %	90	100	82	81	46	100	86
ST-segment elevation in precordial leads, %	85	97	–	81	38	100	86
Elevation of cardiac enzyme levels, %	56	–	–	100	100	–	86
Pathologic Q waves, %	27	–	6	31	31	–	–
Mean QTc, mst	–	–	500	501 ± 55	450	–	–
Initial average LVEF†	0.41 ± 0.11	0.49 ± 0.12	–	0.40	–	–	0.40 ± 0.08
Follow-up LVEF†	0.64 ± 0.10	0.69 ± 0.12	–	0.60	–	0.76	0.63 ± 0.07
Heart failure or pulmonary edema, %	22	3	–	44	46	–	29
IABP, %	8	0	–	6	46	–	14
Coronary stenosis >50%, %	0	0	0	0	0	0	0
Angiographically normal coronary arteries, %	–	83	–	25	38	100	100
Spontaneous multivessel spasm, %	0	10	0	0	0	11	0
Provocable multivessel spasm, n/n (%)	5/48 (10)	6/14 (43)	2/7 (29)	–	–	–	0/4 (0)
Transient dynamic intraventricular gradient, %	18	–	–	13	15	–	–
Preceding emotional stressor, %	20	17	18	38	23	22	14
Preceding physiologic stressor, %	43	17	77	44	46	33	71
In-hospital mortality, %	1	0	0	0	8	0	0
Documented recurrence, n/n (%)	2/72 (2.7)	0	0	1/16 (6)	1/13 (8)	0	0

* IABP = intra-aortic balloon pump; LVEF = left ventricular ejection fraction; QTc = corrected QT interval.
 † Values with a plus/minus sign are the mean ± SD.

ing syndrome consistently demonstrate that most patients presenting with the syndrome are postmenopausal women who most frequently have symptom onset after an episode of acute emotional or physiologic stress. The depressed left ventricular systolic function and characteristic left ventricular apical and mid-ventricular regional wall-motion abnormalities are transient features and generally resolve within days to weeks after initial presentation. The overall prognosis seems to be favorable, although isolated cases of death have been reported. The most common reported clinical complication is left heart failure, which may require aggressive diuresis, inotropic drugs, and hemody-

amic support. In addition, it is not uncommon for patients to develop a hemodynamically significant dynamic left ventricular intracavitary obstruction resulting from dyskinetic apical and mid-ventricular segments with concomitant hyperdynamic basal segments. The identification of a dynamic left ventricular intracavitary pressure gradient has implications for therapy; hemodynamic impairments in this situation require significantly different management from that needed for hypotension due to pure pump failure.

Optimal management of the transient left ventricular apical ballooning syndrome in the acute setting is difficult to determine given the limited available data. Although 1 report documents more pronounced ST-segment elevation in leads V4 to V6 compared with leads V1 to V3 in patients presenting with the syndrome (15), there is still no reliable way to distinguish apical ballooning syndrome-associated ST-segment elevation on the presenting electrocardiogram from ST-segment elevation caused by plaque rupture and acute coronary thrombosis. It is expected that most patients with the transient left ventricular apical ballooning syndrome will undergo emergency diagnostic coronary arteriography. In addition, the syndrome should be suspected in patients with the characteristic left ventricular wall-motion abnormalities in the absence of obvious plaque rupture and coronary thrombosis. After diagnostic arteriography, an appropriate approach to the syndrome seems to involve supportive adjunctive medical management with β -blockers, angiotensin-converting enzyme inhibitors (in patients without an intracavitary gradient), as-

Table 2. Reported Complications Associated with the Transient Left Ventricular Apical Ballooning Syndrome

Left heart failure with and without pulmonary edema
Cardiogenic shock
Dynamic intraventricular obstruction with left ventricular intracavitary pressure gradient generation
Mitral regurgitation resulting from chordal tethering as well as systolic anterior motion of the mitral valve apparatus
Ventricular arrhythmias
Left ventricular mural thrombus formation
Left ventricular free-wall rupture
Death

pirin, and intravenous diuretics, as needed. Hypotension is managed on the basis of its cause; as a result, patients who develop hypotension must be evaluated for a dynamic intraventricular pressure gradient in the left ventricular cavity and left ventricular outflow tract. Echocardiography or left heart catheterization can be used for this evaluation. Dynamic intraventricular obstruction in patients with the syndrome is managed by administration of β -blockers to increase diastolic ventricular filling time and left ventricular end-diastolic volume (16), administration of phenylephrine to increase afterload with subsequent reduction of the intraventricular gradient (as has been described with acute myocardial infarction) (17), and administration of fluid resuscitation if pulmonary congestion is not present. However, β -blockers and phenylephrine would not be recommended for the treatment of dynamic intraventricular obstruction in patients with documented epicardial coronary vasospasm; in this situation, a nondihydropyridine calcium-channel blocker, such as diltiazem or verapamil, could be considered. Short-term anticoagulation should be considered in most patients to prevent left ventricular mural thrombus formation, especially in those with significant left ventricular systolic dysfunction. This therapy should be continued until left ventricular function has improved. As is done for patients with conventional myocardial infarction, patients should be monitored in the hospital for atrial and ventricular arrhythmias, heart failure, and mechanical complications.

Universally accepted diagnostic criteria for the transient left ventricular apical ballooning syndrome are not yet available. However, because the consistent and unique clinical presentation of the syndrome lends itself to a potentially simple diagnostic algorithm, we have proposed such an algorithm (Table 3). The proposed diagnostic criteria could be used when there is no evidence of other obvious causes that might present with similar apical and mid-ventricular wall-motion abnormalities. Obvious causes include recent significant head trauma, intracranial or subarachnoid bleeding, pheochromocytoma, obstructive epicardial coronary artery disease, myocarditis, or hypertrophic cardiomyopathy.

The cause of the transient left ventricular apical ballooning syndrome is still unknown. Several mechanisms have been proposed, including multivessel epicardial spasm, myocardial dysfunction mediated through catecholamine-induced damage, microvascular coronary spasm or dysfunction, and neurogenically mediated myocardial stunning. Inducible coronary vasospasm in at least 1 coronary artery has been reported in a substantial percentage of patients tested (3, 5). However, multivessel coronary vasospasm, which would probably be required to produce the diffuse wall-motion abnormalities seen with the syndrome, has been present in 13 of 73 patients tested across series. Transient multivessel epicardial spasm may be responsible for some cases of the syndrome; however, it is probably not the cause in patients who have persistent ST-segment ele-

Table 3. Proposed Mayo Criteria for the Clinical Diagnosis of the Transient Left Ventricular Apical Ballooning Syndrome*

1. Transient akinesis or dyskinesia of the left ventricular apical and mid-ventricular segments with regional wall-motion abnormalities extending beyond a single epicardial vascular distribution
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture
3. New electrocardiographic abnormalities (either ST-segment elevation or T-wave inversion)
4. Absence of
 - Recent significant head trauma
 - Intracranial bleeding
 - Pheochromocytoma
 - Obstructive epicardial coronary artery disease
 - Myocarditis
 - Hypertrophic cardiomyopathy

* All 4 criteria must be met.

vation at the time of coronary angiography and who have no identifiable epicardial spasm or stenosis.

Coronary microvascular function has been shown to be diffusely abnormal when assessed immediately after presentation by using invasive measurements of coronary flow reserve and TIMI frame count techniques (5, 7, 12, 18–22). In addition, injection of nicorandil into the coronary arteries during the acute phase of the syndrome acutely reduces the extent of ST-segment elevation, further suggesting that microvascular spasm may be the causative mechanism of the syndrome (23). It is unclear whether coronary microvascular dysfunction is the primary mechanism involved in the pathogenesis of the syndrome or whether it is simply an associated secondary phenomenon.

Reversible perfusion abnormalities in the left ventricular apex in patients presenting with the syndrome have been documented (6, 20, 24, 25). In addition, patients with the syndrome exhibit a pronounced abnormality in apical myocardial fatty acid metabolism that is out of proportion to apical perfusion abnormalities (24). This impairment of regional myocardial fatty acid metabolism demonstrates that myocardial “stunning” appears to be present in the distribution of the wall-motion abnormalities seen in the syndrome.

Catecholamines probably play a role in the syndrome. Some researchers have suggested that the syndrome could be a result of catecholamine-associated stunning of the myocardium, which is provoked by emotional or physiologic stress (26). Patients presenting with the syndrome appear to have abnormalities of cardiac sympathetic innervation with evidence of sympathetic hyperactivity at the cardiac apex (27, 28). The distribution of apical wall-motion abnormalities in the syndrome is similar to the distribution reported with catecholamine-induced cardiomyopathy (29). Of note, the wall-motion abnormalities in the syndrome are not typical of those found with subarachnoid hemorrhage or intracranial hemorrhage in which the apex is generally spared and the basal left ventricular segments

are affected (30, 31). However, isolated reports describe transient apical systolic dysfunction associated with subarachnoid hemorrhage (26, 32–34). Measurements of circulating catecholamine levels in patients presenting with the syndrome have shown inconsistent results (4, 5).

Myocarditis could present with characteristics similar to those seen with the transient left ventricular apical ballooning syndrome. However, several investigators could not demonstrate biopsy evidence of myocarditis in patients with apical ballooning.

Patients with the transient left ventricular apical ballooning syndrome seem to often present in the setting of an acute mental stress, a time of enhanced sympathetic outflow. This association could be linked to mental stress-induced transient coronary endothelial dysfunction (35, 36). It is unclear why the apex of the heart is affected and the basal segments are spared. However, this may be partly explained by increased adrenergic receptor density in cardiac apical segments or increased apical myocardial responsiveness to adrenergic stimulation (37). Transient apical and mid-ventricular wall-motion abnormalities have been induced in rats through physical immobilization, a model of emotional stress (38). In this model, the wall-motion abnormalities could not be reproduced after pretreatment with the α - and β -adrenoreceptor antagonist amosulalol hydrochloride. Of note, estradiol supplementation has been reported to attenuate emotional stress-induced changes in left ventricular function in ovariectomized female rats (39).

The cause of the transient left ventricular apical ballooning syndrome is unknown. However, speculatively, it may represent a catecholamine-mediated myocardial stunning that results from a combination of myocardial ischemia related to diffuse microvascular dysfunction and, in some cases, multivessel epicardial spasm and metabolic injury. The explanation for the strong female predominance with the syndrome is also unclear. However, the explanation may be related to postmenopausal alterations of endothelial function in response to reduced estrogen levels (40) and microcirculatory vasomotor reactivity in response to catecholamine-mediated stimuli (39).

Our review has several limitations, which are mainly a consequence of the relative lack of published data on patients presenting with the transient left ventricular apical ballooning syndrome. Although we included only case series that reported on consecutive patients presenting with the syndrome, these limited reports may not precisely represent the entire spectrum of the syndrome. In addition, there are no generally accepted criteria for the consistent diagnosis of the syndrome, which could lead to heterogeneity in the patients given the diagnosis in the reported case series of the syndrome. Therefore, we have limited the case series presented in this review to those reporting on consecutive patients who presented as having a suspected acute coronary syndrome with transient apical and mid-ventricular wall-motion abnormalities in the documented

absence of obstructive epicardial coronary disease. This review is further limited by the lack of comprehensive demographic, clinical, and outcome data in all series. Although we thoroughly searched for English-language articles pertaining to the transient left ventricular apical ballooning syndrome, our search may have missed pertinent literature.

The transient left ventricular apical ballooning syndrome is a recently described cardiac syndrome that appears to commonly mimic acute ST-segment elevation myocardial infarction. Case series of the syndrome have demonstrated that patients commonly present with ischemic-like chest pain or dyspnea, electrocardiographic changes resembling acute myocardial injury, minor elevation of cardiac enzyme and biomarker levels, and transient “ballooning” of the left ventricular apex and mid-ventricle. Although the cause of the syndrome is unknown, the syndrome should be considered in the differential diagnosis in patients presenting with an apparent acute coronary syndrome in the absence of obstructive atherosclerosis. Despite the absence of obstructive epicardial coronary disease, most patients presenting with the syndrome meet the American College of Cardiology/European Society of Cardiology criteria for the diagnosis of myocardial infarction (41). It may be useful to consider the transient left ventricular apical ballooning syndrome as a unique type of acute coronary or myocardial syndrome. Additional evaluation of the syndrome is needed. The evaluation should include more precise assessment of the true incidence of the syndrome, identification of risk factors for developing the syndrome, potential preventive measures, risk stratification, as well as further elucidation of the underlying pathophysiologic mechanisms responsible for the syndrome.

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