# Prevalence and characteristics of left ventricular outflow tract obstruction in Tako-Tsubo syndrome

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**Background and Objective** Tako-Tsubo syndrome is a clinical entity mimicking acute coronary syndrome (ACS). Left ventricular outflow tract (LVOT) obstruction may occur in Tako-Tsubo syndrome. The aim of this study was to determine the prevalence and features of LVOT obstruction in Tako-Tsubo syndrome in a population presenting with ACS.

**Methods** This study included consecutive patients admitted to 2 catheterization laboratories for suspected ACS. All patients underwent echocardiography, coronary arteriography, and left ventricular angiography if no significant coronary lesions were found.

**Results** Among 10,366 patients referred for coronary angiography, the study population consisted of 3,909 patients with suspected ACS. Thirty-two patients (mean age 71 ± 13 years old) presented with Tako-Tsubo syndrome, resulting in a prevalence of 0.8% in our population of ACS and 5% of patients without significant coronary lesions. Eight women (mean age 81 ± 4 years old, $P = .01$) exhibited LVOT obstruction, a prevalence of 25% among Tako-Tsubo syndrome cases. All patients with intraventricular pressure gradient had systolic anterior motion of the mitral valve and septal bulge. Prevalence of septal bulge was 100% in patients with Tako-Tsubo syndrome and LVOT obstruction versus 29% in patients without LVOT obstruction ($P = .002$). Mean degree of mitral regurgitation was 2.1 ± 0.7 in cases of LVOT obstruction versus 0.9 ± 0.7 in patients without LVOT ($P = .0003$) and significantly decreased during follow-up (1 ± 0.8, $P = .002$). Recovery of left ventricular ejection fraction was similar in patients with and without LVOT obstruction ($P = .58$).

**Conclusions** The present study demonstrates that the prevalence of LVOT obstruction in Tako-Tsubo syndrome is high, with specific characteristics as compared with patients without LVOT obstruction. Echocardiography should be systematically performed for all patients presenting with Tako-Tsubo syndrome for the detection of LVOT obstruction. (Am Heart J 2008;156:543-8.)

Tako-Tsubo syndrome, which is also called stress cardiomyopathy or transient left ventricular apical (or midventricular) ballooning syndrome, is a clinical entity mimicking an acute coronary syndrome (ACS).[1,2] It is characterized by reversible left ventricular dysfunction that is frequently precipitated by a stressful event, as demonstrated by previous studies.[2-4] In the acute period, some complications may occur, particularly cardiogenic shock.[5] Management of such patients remains difficult, with the use of inotropic agents or intraaortic balloon pump.[3]

Some cases of left ventricular outflow tract (LVOT) obstruction after Tako-Tsubo syndrome have been reported,[6-9] and their detection is of importance because the use of inotropic agents may increase the intraventricular pressure gradient and induce cardiogenic shock.[10,11] The aim of our study was to determine the prevalence and features of LVOT obstruction in Tako-Tsubo syndrome in a population presenting with ACS.

## Methods

Over a 6-year period (January 2000-September 2006), we reviewed from our databases all patients (N = 10,366) referred to 2 catheterization laboratories for coronary angiography. Data concerning the prevalence of Tako-Tsubo syndrome has been previously published.[2] According to the clinical data, electrocardiogram, and biological markers, we thus individualized patients with suspected ACS, representing the study population (n = 3,909) (Figure 1). Criteria for selection included patients aged >18 years and presenting with suspected ACS.[12] All patients systematically underwent coronary angiography and left ventricular angiography if no

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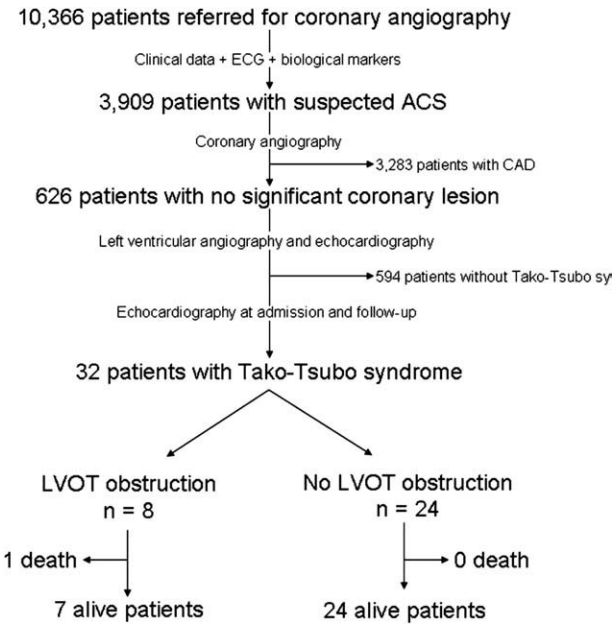
**Figure 1**




Flowchart used in the study for the diagnosis of Tako-Tsubo syndrome.

significant coronary lesions were found, <48 hours after symptom onset. Ventricular angiograms were used to calculate left ventricular ejection fraction and detect wall motion abnormalities. Patients with known renal failure, defined as a creatinine clearance <30 mL/min, had no left ventricular angiogram, and left ventricle was systematically assessed by echocardiography. We identified 2 groups of patients: patients with coronary lesions (n = 3,283) and patients with no significant coronary lesion, defined as luminal stenosis <50% (n = 626). Coronary vasospasm provocation test was performed in case of suspected Prinzmetal's angina. Among patients without coronary artery disease, we strictly defined Tako-Tsubo syndrome as (1) an acute chest pain during stressful incident associated with ST-segment abnormalities and/or increased serum troponin level, (2) transient nonsystematized systolic dysfunction, and (3) no significant coronary lesions.[2,13] Non-systematized systolic dysfunction was defined as regional wall motion abnormalities extending beyond a single epicardial coronary distribution. Treatment was left to the discretion of the physician. At admission, all patients with suspected Tako-Tsubo syndrome systematically underwent transthoracic echocardiography. Echocardiographic studies consisted of apical 2-, 3-, 4-, and 5-chamber views for (1) calculation of left ventricular ejection fraction using 2-dimensional Simpson's rules; (2) assessment of wall motion abnormalities; and (3) assessment of LVOT obstruction, using 2-dimensional second harmonic imaging, color Doppler, and continuous wave Doppler. Left ventricular outflow tract obstruction was defined as a dynamic intraventricular pressure gradient (cut-off value 20 mm Hg) detected by continuous wave Doppler using a modified Bernoulli equation.[3] We also assessed the presence of systolic anterior motion of the mitral valve and of septal bulge (localized hypertrophy of the proximal interventricular septum) using 2-dimensional imaging.[14] Mitral regurgitation was quantified by calculating regurgitant volume (or color Doppler jet area and/or vena contracta width when regurgitant volume was impossible to obtain), according to the American College of Cardiology/American Heart Association guidelines.[15] Mild mitral regurgitation was scored as 1, moderate as 2, and severe as 3. Patients with Tako-Tsubo syndrome underwent serial echocardiographic studies with the same protocol in our echocardiographic laboratory at day 7, 1 month, and 1 year after the acute phase. All echocardiographic studies were reviewed by 2 experienced physicians (N.M. and O.D.) who were blinded to clinical data. Finally, inhospital and 1-year follow-up was collected for all patients with Tako-Tsubo syndrome by phone contact with the patients or with their cardiologists.

### Statistical analysis

Continuous variables are presented as means ± SD and ranges, unless otherwise specified. Categorical data are presented as absolute values and percentages. Continuous and categorical variables were compared with use of the $\chi^2$ test,paired *t* test, unpaired *t* test, and Fisher test, as appropriate. We analyzed the data using SAS statistical software, version 8.2 (SAS Institute, Inc, Cary, NC). A *P* valueof <.05 was considered significant.

## Results

### Population characteristics

Among the 3,909 patients with suspected ACS (Figure 1), 2,755 (70%) underwent percutaneous coronary interventions, 111 (3%) were referred for coronary artery bypass graft surgery, and 1,043 (27%) had a medical treatment. Coronary angiography revealed the following distribution: 37% with single-vessel disease (1,456 patients), 24% with 2-vessel disease (956 patients), and 22% with 3-vessel disease (870 patients).

### Tako-Tsubo syndrome

Thirty-two patients with a mean age of 71 ± 13 years (range 35-90 years) presented with Tako-Tsubo syndrome, resulting in a prevalence of 0.8% in our population of ACS and 5% of patients without significant coronary lesions. The prevalence of Tako-Tsubo syndrome was 3.1% in women versus 0.03% in men ($P < .0001$). The characteristics of these patients are listed in Table I. The mean peak of plasma creatinine kinase and of troponin I was 339 ± 263 IU/L and 6.9 ± 6.6 μg/L, respectively. Mean left ventricular ejection fraction by left ventricular angiography and by echocardiography was 44% ± 9% and 36% ± 6%, respectively. On electrocardiogram, ST-segment elevation and T-wave inversion were found in 17 patients (53%) and 12 patients (38%), respectively. Twenty-eight patients (88%) presented with a typical pattern of Tako-Tsubo syndrome with akinesia of the

mid and distal segments of all walls, with compensatory hyperkinesia of the base. Four patients (12%) presented with a partial and circular pattern of Tako-Tsubo syndrome: 1 patient with a pattern of inverse Tako-Tsubo syndrome (akinesia of the basal and mid-segments of all walls) and 3 patients with a pattern of midventricular ballooning syndrome (limited akinesia of the mid-segments of all left ventricular walls).[16]

### Left ventricular outflow tract obstruction

Eight patients exhibited significant LVOT obstruction, which corresponds to a prevalence in Tako-Tsubo syndrome of 25%. Mean age of patients with LVOT was 81 ± 4 years (range 75-86 years) and was higher than that of patients without LVOT obstruction. No patient had a familial history of hypertrophic cardiomyopathy. At admission, mean New York Heart Association functional class was significantly higher in the case of LVOT obstruction ($P$ = .002, Table II). Among patients with LVOT obstruction, mean dynamic intraventricular pressure gradient detected by echocardiography was 34 ± 16 mm Hg (range 21-60 mm Hg). All patients with a gradient had systolic anterior motion of the mitral valve associated with septal bulge (Figure 2). All patients with systolic anterior motion of the mitral valve and septal bulge also had mitral regurgitation. Mean degree of mitral regurgitation was 2.1 ± 0.7 versus 0.9 ± 0.7 in patients without LVOT ($P$ = .0003). Two patients with LVOT obstruction exhibited cardiogenic shock at admission. All patients received β-blockers, and β-blocker dosage was similar in patients with and without LVOT obstruction. No patient with LVOT obstruction received inotropic agents. At day 7, echocardiography did not reveal any residual dynamic intraventricular pressure gradient. Among patients with LVOT obstruction, mean degree of mitral regurgitation significantly decreased at day 7, when compared to baseline degree of mitral regurgitation (1 ± 0.8, $P$ = .002).

### Follow-up

Echocardiography was available at day 7 for all patients, at 1 month for all patients except 1, and at 1 year for 24 patients (75%). Left ventricular ejection fraction was significantly improved at day 7 (45% ± 7%, $P$ < .0001), and after 1 month (58% ± 8%, $P$ < .0001 as compared to day 7) and 1 year (67% ± 4%, $P$ < .0001 as compared to 1-month follow-up). Recovery was similar in patients with LVOT obstruction and in patients without LVOT ($P$ = .58). No echocardiographic transient left ventricular dysfunction was detected during follow-up. All LVOT obstruction detected at admission disappeared at day 7 and no LVOT obstruction was detected during follow-up. No other significant echocardiographic changes were observed concerning 2-dimensional and Doppler data. During a mean follow-up of 21 ± 12 months, the rate of mortality was only 3%. One patient died of acute respiratory distress syndrome. No recurrence of Tako-Tsubo syndrome was observed in our population.

## Discussion

Left ventricular outflow tract obstruction may occur in Tako-Tsubo syndrome.[6-9] At the time of this study, its prevalence was uncertain because of a scarcity of data.[5,10,17] In the present report, LVOT obstruction was detected in 25% of patients with Tako-Tsubo syndrome and we found that the characteristics of this population are different. Patients were older and all presented septal hypertrophy associated with systolic anterior motion of the mitral valve.

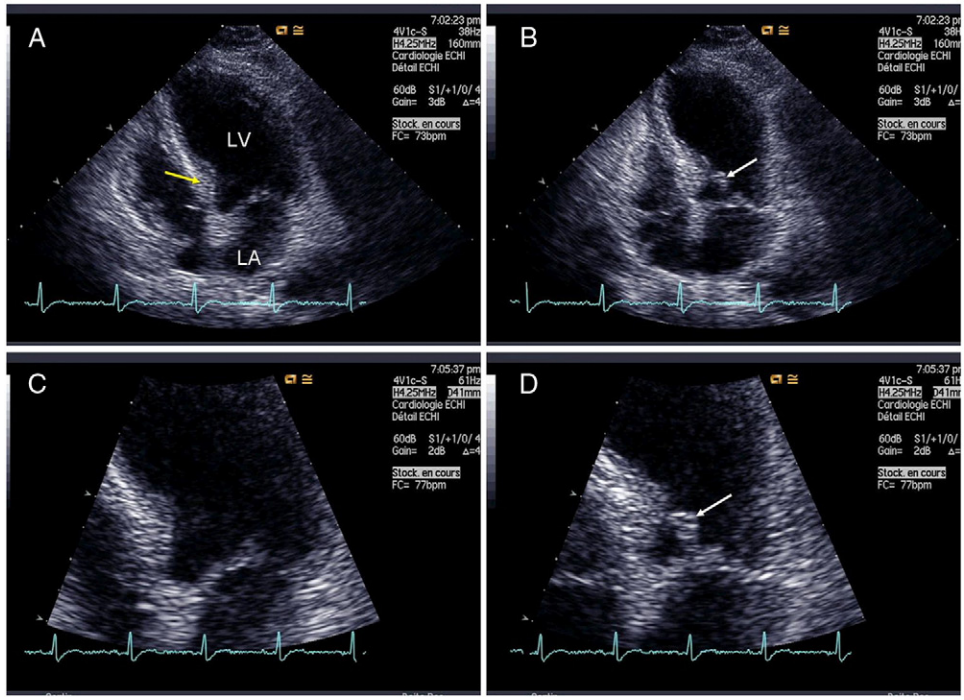
Tako-Tsubo syndrome is most often suspected in patients with a clinical presentation of ACS, typically associated with an emotional stress event.[2,3,18-20] It is diagnosed when coronary angiography reveals no significant coronary lesion and left ventricular angiography detects a typical or partial and circular pattern of Tako-Tsubo syndrome.[2] Conventional transthoracic echocardiography alone may be deficient in making this diagnosis. However, echocardiography in Tako-Tsubo syndrome is of great interest for the detection of LVOT obstruction. Few previous studies have reported the prevalence of LVOT obstruction in Tako-Tsubo syndrome (<20%)[5,10,17,21] and were not focused on this specific topic. In our study, echocardiography was performed at admission of the patients (ie, during the acute phase) and detected all patients with LVOT obstruction. The characteristics of patients with LVOT obstruction differed from those of patients with Tako-Tsubo syndrome but without LVOT obstruction. In our study, patients with LVOT obstruction were older and all presented a septal bulge, associated with systolic anterior motion of the mitral valve and mitral regurgitation.[14] This morphological pattern of the interventricular septum is mostly present in elderly patients and seems to be an important factor of LVOT obstruction in Tako-Tsubo syndrome, mimicking a pattern of hypertrophic cardiomyopathy obstruction.[22-24] As a French reference center of hypertrophic cardiomyopathy, we systematically reviewed all echocardiographic examinations of patients with Tako-Tsubo syndrome and we found that LVOT obstruction was related to the typical pattern of septal bulge in elderly patients. We concluded that echocardiographic pattern in patients with Tako-Tsubo syndrome and LVOT obstruction was definitively not in favor of hypertrophic cardiomyopathy, even when pressure gradient and systolic anterior motion of the mitral valve were initially found. Furthermore, the pathogenesis of Tako-Tsubo syndrome remains debated. Several hypotheses have been proposed. One of them is that LVOT obstruction could be a major factor in the development of Tako-Tsubo syndrome.[11] However, this hypothesis is still debated.[10]

**Table I.** Characteristics of 32 patients with Tako-Tsubo syndrome

| Patient no. | Age (y)/sex | Precipitating factor | Presenting symptoms | NYHA Status | ECG | | Peak of | | Invasive LVEF (%) | Wall motion abnormalities | | | LVOT Obstruction |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | ST Elevation | T inversion | CK (UI/L) | Troponin I (μg/L) | | Apical | Median | Basal | |
| 1 | 35/M | Fear of surgery | Chest pain | I | 0 | + | 290 | 23 | 46 | 0 | + | + | 0 |
| 2 | 45/F | Acute asthma | Palpitations | I | + | 0 | 1230 | 16 | 33 | + | + | 0 | 0 |
| 3 | 49/F | Professional conflict | Chest pain | I | + | 0 | 1049 | 28.44 | 27 | + | + | 0 | 0 |
| 4 | 55/F | Robbery | Chest pain | I | + | 0 | 264 | 6.2 | 40 | + | + | 0 | 0 |
| 5 | 59/F | Altercation | Chest pain | I | 0 | + | 422 | 4.64 | 45 | + | + | 0 | 0 |
| 6 | 60/F | Car accident | Chest pain | I | 0 | + | 336 | 2.19 | 50 | + | + | 0 | 0 |
| 7 | 60/F | Divorce | Chest pain | I | 0 | 0 | 211 | 2.61 | 47 | + | + | 0 | 0 |
| 8 | 64/F | Annoyance | Chest pain | I | 0 | + | 100 | 1.07 | 56 | + | + | 0 | 0 |
| 9 | 65/F | – | Chest pain | I | + | 0 | 34 | 1.32 | 64 | + | + | 0 | 0 |
| 10 | 65/F | Familial difficulties | Chest pain | I | 0 | + | 246 | 1.68 | 47 | + | + | 0 | 0 |
| 11 | 69/F | Altercation | Chest pain | I | 0 | + | 255 | 0.67 | 55 | 0 | + | 0 | 0 |
| 12 | 69/F | Tragic news | Chest pain | I | 0 | + | 139 | 2.49 | 31 | + | + | 0 | 0 |
| 13 | 70/F | Car accident | Chest pain | II | 0 | + | 367 | 5.3 | 44 | + | + | 0 | 0 |
| 14 | 71/F | Parent disease | Chest pain | I | 0 | + | 334 | 3.51 | 55 | + | + | 0 | 0 |
| 15 | 72/F | Escalator failure | Chest pain | II | + | 0 | 190 | 8.6 | 44 | + | + | 0 | 0 |
| 16 | 75/F | Fear of death | Chest pain + dyspnea | III | + | 0 | 332 | 7.8 | 40 | + | + | 0 | + |
| 17 | 75/F | Tragic news | Chest pain | I | + | 0 | 441 | 8.1 | 40 | + | + | 0 | 0 |
| 18 | 77/F | Severe epilepsy | - | I | + | 0 | 387 | 5.3 | 50 | 0 | + | 0 | 0 |
| 19 | 77/F | Professional conflict | Chest pain | I | 0 | 0 | 253 | 8.4 | 39 | 0 | + | 0 | 0 |
| 20 | 78/F | Fire | Chest pain | I | + | 0 | 413 | 6.75 | 50 | + | + | 0 | 0 |
| 21 | 78/F | Tragic news | Chest pain | I | + | 0 | 154 | 4.95 | 57 | + | + | 0 | + |
| 22 | 79/F | Chronic stress | Chest pain | I | + | 0 | 270 | 7.97 | 32 | + | + | 0 | + |
| 23 | 80/F | Altercation | General malaise | II | 0 | + | 171 | 3.34 | 41 | + | + | 0 | + |
| 24 | 80/F | Familial difficulties | Chest pain | I | 0 | + | 276 | 7.08 | 53 | + | + | 0 | 0 |
| 25 | 81/F | Tragic news | Chest pain | I | + | 0 | 637 | 5.13 | 45 | + | + | 0 | 0 |
| 26 | 84/F | Victim of assault | Chest pain | II | 0 | + | 101 | 5.5 | 45 | + | + | 0 | + |
| 27 | 84/F | – | Chest pain | I | + | 0 | 150 | 4.18 | 51 | + | + | 0 | + |
| 28 | 84/F | Familial difficulties | Chest pain | I | + | 0 | 458 | 9.08 | 37 | + | + | 0 | + |
| 29 | 84/F | Car accident | Chest pain | I | + | 0 | 180 | 0.78 | 50 | + | + | 0 | 0 |
| 30 | 86/F | Major stress | Chest pain | III | + | 0 | 795 | 21.3 | 25 | + | + | 0 | + |
| 31 | 87/F | Tragic news | Chest pain | I | 0 | 0 | 128 | 3.6 | 44 | + | + | 0 | 0 |
| 32 | 90/F | Physical trauma | Chest pain | I | + | 0 | 238 | 2.47 | 45 | + | + | 0 | 0 |

*CK*, Creatinine kinase; *ECG*, electrocardiogram; *LVEF*, left ventricular ejection fraction; *NYHA*, New York Heart Association; *M*, male; *F*, female.

**Figure 2**




Echocardiographic pattern of LVOT obstruction in diastole (A and C) and systole (B and D) in an apical 4-chamber view. The white arrows indicate the systolic anterior motion of the mitral valve and the yellow arrow indicates the septal bulge. *LA*, Left atrium; *LV*, left ventricle.

**Table II.** Characteristics of patients with and without LVOT obstruction

| Variable | Patients with LVOT obstruction (n = 8) | Patients without LVOT obstruction (n = 24) | *P* Value |
|---|---|---|---|
| Age (y) | 81 ± 4 | 68 ± 13 | .01 |
| History of hypertension | 6 (75%) | 11 (46%) | .22 |
| New York Heart Association functional class | 1.8 ± 0.9 | 1.1 ± 0.1 | .002 |
| Peak of creatinine kinase (UI/L) | 304 ± 203 | 350 ± 276 | .67 |
| Peak of troponin I (μg/L) | 8 ± 5.7 | 6.5 ± 6.9 | .57 |
| Left ventricular ejection fraction (%) | 41 ± 10 | 46 ± 8 | .19 |
| Septal bulge | 8 (100%) | 7 (29%) | .002 |
| Degree of mitral regurgitation | 2.1 ± 0.7 | 0.9 ± 0.7 | .0003 |

Conventional echocardiography is also valuable for management of patients with LVOT obstruction. In fact, the use of inotropic agents, particularly in patients with shock, may increase the gradient and worsen cardiogenic shock.[3,25] Echocardiography should thus be systematically performed at admission in patients with Tako-Tsubo syndrome, to guide treatment. The detection of LVOT obstruction requires the use of β-blockers, even in patients with cardiogenic shock,[8] and inotropic agents should be avoided. This therapeutic strategy is only possible if echocardiography is systematically performed.

The limitation of this study is that we reviewed in our databases all cases of suspected ACS referred to 2 catheterization laboratories over a long period. The incidence of LVOT obstruction in Tako-Tsubo syndrome could be underestimated in the general population, because this study did not include patients with an initial cardiac arrest, reflecting a severe pattern of Tako-Tsubo syndrome. However, this eventuality remains extremely rare and represents a very small number of patients around the world.

## Conclusions

The present study demonstrates that the prevalence of LVOT obstruction in Tako-Tsubo syndrome is high, with specific characteristics as compared with patients without LVOT obstruction. Echocardiography should be systematically performed for all patients presenting with Tako-Tsubo syndrome for the detection of a dynamic intraventricular pressure gradient.

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