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Short and long term follow-up of patients with transient left ventricular apical ballooning

Tomasz Pawłowski, Robert J. Gil, Jarosław Rzezak, Hanna Rdzanek and Marcin Młotek

Departament of Invasive Cardiology, CSK MSWiA, Warsaw, Poland

Abstract

Background: *Transient left ventricular apical ballooning (TLVAB) was described in Japan as the syndrome characterized by balloon-like left ventricular wall abnormalities without accompanying coronary atherosclerosis. The aim of the study was to evaluate the short and long-term follow-up of our patients with TLVAB.*

Methods: *The population of 1869 patients with the preliminary diagnosis of acute coronary syndrome was screened. We collected data regarding clinical course, physical or emotional trauma, ECG abnormalities, presence of balloon-like left ventricular abnormality. During follow-up, the data on cardiac events (death, recurrence of chest pain, re-hospitalization) were collected. Serial echocardiographic scans were performed to follow the TLVAB regression.*

Results: *Thirty (1.6%) patients (only women, mean age 72 ± 12 y) showed characteristic left ventricle abnormalities. There was an evidence of significant trauma (40% physical and 60% emotional) in every patient. ECG analysis showed negative T waves in anterior leads in 93% and ST elevation in 7% of the patients. Maximal MB creatine kinase and troponin I levels were 38 ± 12 U/l and 1.12 ± 0.75 ng/dl, respectively. The clinical course during hospitalization was benign in 96% and only in one patient (4%) the pulmonary oedema occurred. During follow-up (6–24 months, mean 12 months), there were two cases of chest pain recurrence, but without clinical features of TLVAB (examined by echo or angiography). Mean time for left ventricular abnormalities regression was 3 ± 1 months.*

Conclusions: *The transient left ventricular apical ballooning is a benign syndrome mimicking acute coronary syndrome with good long term outcome.* (Folia Cardiol. 2006; 13: 511–516)

Key words: transient left ventricular apical ballooning, diagnostics, angiography, long term follow-up

Introduction

The broadening of the indications and availability of urgent coronarography in patients with acute coronary syndrome (ACS) increases number of patients (1–3%) in whom normal coronary arteries are found [1]. Since the beginning of the '90, there were some reports in literature of a new disease [2–4] named the "takotsubo" cardiomyopathy (a Japanese term for a vessel for cathing octopuss) due to it's characteristic angiographic appereance, or transient left ventricular apical ballooning (TLVAB). According to relatively small number of publications, the symptoms of this disease are: stenocardia with electrocardiographic changes and minor elevation of markers of myocardial necrosis, as well as sudden, reversible akinesis or hypokinesis of the apex and adjacent segments.

Address for correspondence: Dr med. Tomasz Pawłowski
Department of Invasive Cardiology, CSK MSWiA
Wołoska 137, 02–507 Warszawa, Poland
e-mail: shortytom@o2.pl
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The aim of this study is to present the course of hospitalization and long-term follow-up of the TLVAB patients hospitalized in our Cardiology Department.

Methods

Study population

In the period between January 2002 and December 2004, 1869 patients were admitted to our Cardiology Department during 24-hour emergency service for urgent invasive diagnostics (angiography). The indication for this procedure were typical clinical and electrocardiographic signs of acute myocardial infarction with ST elevation or acute coronary syndrome with electrocardiographic changes (ST depression, negative T) and elevation of cardiac markers.

Every patient had a angiography performed using radial or femoral artery access. Standard projections in two ortogonal projections for evaluation of every branch of coronary artery tree were performed to evaluate the angiograms. According to angiographic and clinical data the patients were qualified for next stages of treatment (coronary angioplasty, coronary arterial bypass grafting, and conservative treatment). In case of normal coronary vessels, the operator performed ventriculography of the left verticle in right anterior oblique from 30 degrees angle to show anterior and inferior wall and the apex of the left ventricle.

TLVAB diagnosis

To diagnose the TLVAB following major criteria were used: akinesis or hypokinesis of the apex and adjacent segments with hyperkinesis of basal segments in ventriculography, accompanied by absence of significant atherosclerosis changes in angiography (Fig. 1) and electrocardiographic changes of ST elevation or depression type, negative T waves (Fig. 2), minor elevation of myocardial necrosis markers (which have not fulfilled the criteria of myocardial infarction) [2]. Additional minor criteria included: age above 70, recent occurrence of physical and emotional trauma, negative history of past myocardial infarction, psychiatric disorders, female sex. Moreover, the occurrence of coronary artery disease risk factors and history of cerebrovascular accidents were obtained in each patient.

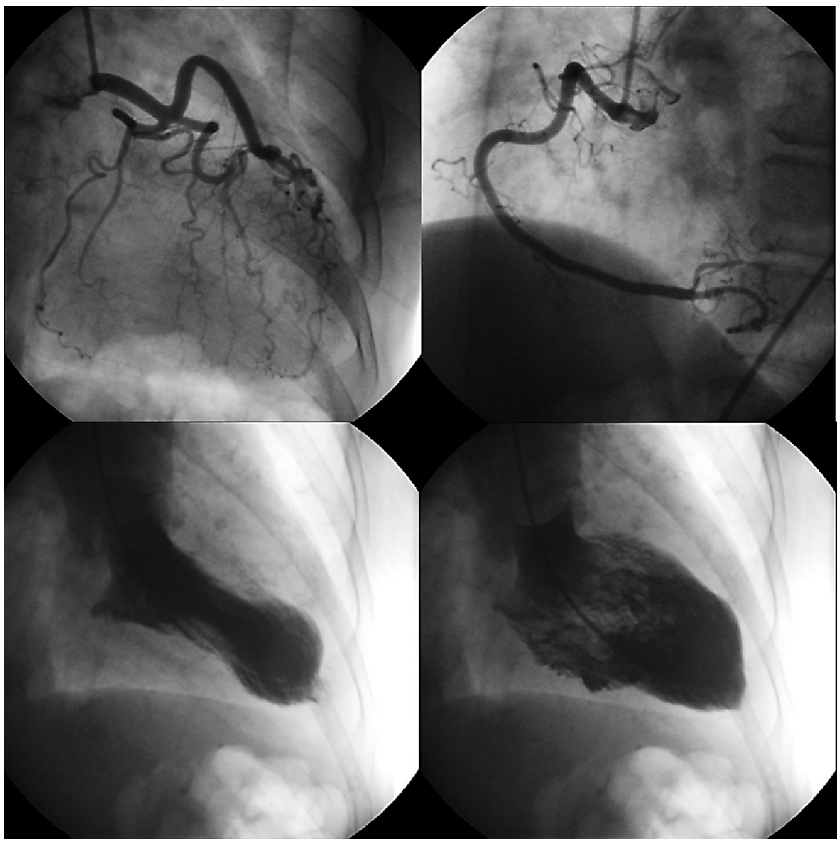


Figure 1. Angiography (no change in coronary arteries; upper row) and left ventriculography of transient left ventricular apical ballooning during end-systolic and end-diastolic phase.

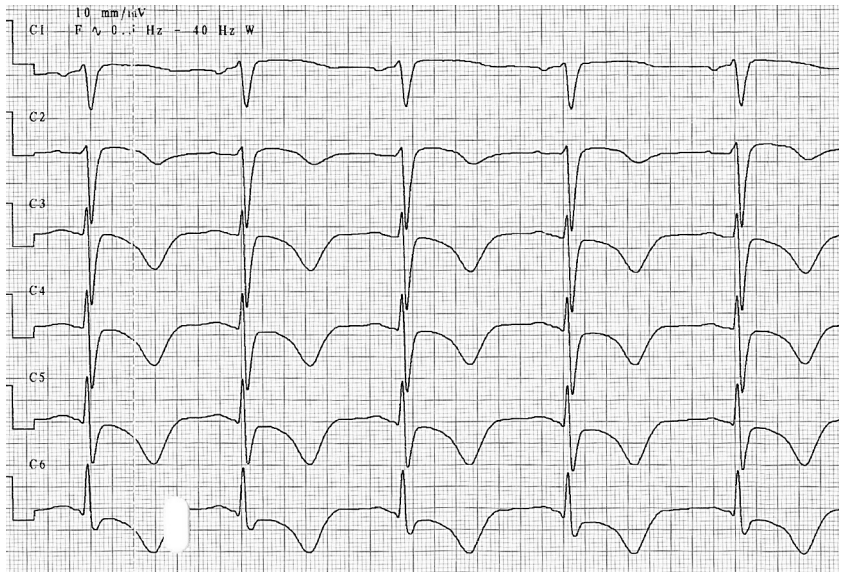


Figure 2. ECG recording of a patient with TLVAB, chest pain and positive troponin test (max 1.45 ng/dl).

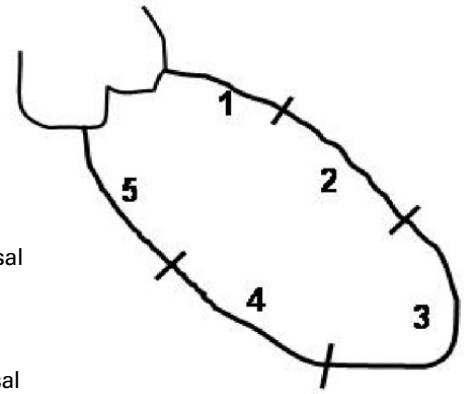
ECG and imaging methodology

The electrocardiograms were analysed for relevant ST wave changes (1 mm depression or elevation), negative T wave occurrence. During hospital stay, the clinical course of the disease was observed, as well as occurrence of such events as death, acute circulatory failure (cardiogenic shock, pulmonary oedema), recurrent anginal symptoms, relevant supraventricular and ventricular arrhytmias.

During long-term follow-up, the data was analysed for occurrence of death, recurrence of stenocardial complaints, and re-hospitalizations.

The images acquired during left ventricular ventriculography were analysed for the extent of contraction abnormalities using the AHA classification in own modification (Fig. 3) for the evaluation of the segments of the left ventricle [5] and evaluation of contractility to classify them as hyper-, normo-, hypo- and akinesis of a segment. According to those findings, each segment was described with numerical value in accordance to observed contractility (hyperkinesis — 0 pt, normokinesis — 1 pt, hypokinesis — 2 pt, akinesis — 3 pt). From the sum of those points divided by the number of segments, the wall motion index was calculated (WMI).

A commercially avaliable CMS-QCA (MEDIS Co) programme was used for planimetric evaluation of end-diastolic volume (EDV) and end-systolic volume (ESV) and ejection fraction. The measurments were conducted during acute phase and during control angiography.



1. Anterobasal
2. Anterior
3. Apical
4. Inferior
5. Infero-basal

Figure 3. Left ventricle segments.

Echocardiography was performed in every patient and angiography with ventriculography in a group of patients to evaluate the regression of contractility abnormalities of the left ventricle. This examination was performed at least 6 weeks after the occurence of TLVAB.

Results

In the examined group of patients, 30 of them (2.2%) met the criteria of TLVAB, among whom there were only women in mean age of 72 ± 12 years of age. The demographic data and risk factors of coronary artery disease found are shown in Table 1. History of coronary heart disease was reported in 26.7% of patients. Table 2 shows percentage of patients who meet individual criteria of TLVAB diagnosis.

Table 1. Baseline characteristics of the patients.

Mean age (year)	72 ± 12
Female sex	30 (100%)
Hypertension	21 (70.0%)
Diabetes	4 (13.3%)
Smoking	11 (36.7%)
Obesity	16 (53.3%)
History of coronary artery disease	8 (26.7%)
History for psychiatric disorders	12 (40.0%)

Table 2. Criteria of transient left ventricular apical ballooning.

Changes in ventriculography	100%
Changes in ECG	100%
Elevated myocardial necrosis markers	93.3%
Age over 70 years	86.7%
Physical or emotional trauma	100%
Negative history for prior myocardial infarction	100%
Psychiatric disorders	40%
Female sex	100%

Every patient in the studied group had a history of physical or emotional trauma within 4 weeks prior to hospitalization. In 40% of patients, it was a physical trauma, and in 60% it was related to a highly emotional experience. Detailed data is shown in Table 3.

The analysis of electrocardiograms revealed that in 2 (6.7%) patients there was ST elevation present, in the first in anterior region, and in the second on the lateral wall. In 24 (80%) patients deep T waves in ECG in precordial leads were observed (Fig. 2), and in 4 (13.3%) patients there were ST depression-type changes (in 2 of them those were found on the anterolateral wall, and in 2 the following on the inferior wall).

Table 3. Risk factors for triggering transient left ventricular apical ballooning in studied patients.

Pleurocentesis	1 (3.3%)
Excessive physical exercise	5 (16.7%)
Pacemaker implantation	1 (3.3%)
Peripheral arterial angiography	1 (3.3%)
Incident of asthma/dyspnea	2 (6.6%)
External trauma	2 (6.6%)
Death of a relative	11 (36.7%)
Accident/theft	5 (16.7%)
Acute phase of a physical disorder	2 (6.6%)

Table 4. Hemodynamic parameters during acute phase of transient left ventricular apical ballooning and in follow-up ventriculography.

Acute phase (n = 29)	
End-diastolic volume index [ml/m^2]	83 ± 16
End-systolic volume index [ml/m^2]	42 ± 19
Ejection fraction	52 ± 10%
Wall motion index	1.32 ± 0.2
Long-term follow up (n=14)	
End-diastolic volume index [ml/m^2]	65 ± 12*
End-systolic volume index [ml/m^2]	29 ± 13*
Ejection fraction	69 ± 12%*
Wall motion index	1.08 ± 0.1

*$p < 0.05$ *pre vs. post*

The maximal values of myocardial necrosis markers were: 38 ± 12 U/l for CK-MB, 286 ± 67 U/l for CPK and 1.12 ± 0.75 ng/dl for troponin I.

There were no relevant arrhythmias noted during ECG monitoring, only in 3 patients there were episodes of atrial fibrillation, which have resolved on typical treatment (amiodarone, beta-blockers).

In only 1 patient (4%) the occurrence of TLVAB was associated with signs of left ventricular cardiac failure, the remaining patients had no relevant clinical incidents.

Every patient had normal coronary arteries confirmed in angiographic images.

Table 4 shows data acquired from quantitative analysis of the left ventriculography. As compared to the period of disease, ejection fraction and left ventricular volume index have improved significantly ($p < 0.05$). An example of angiography and ventriculography is shown in Figure 1.

In the long-term follow-up (mean period of observation 12 months, range 6-24 months) no relevant clinical events were observed, except for periods of stenocardial complaints recurrence in two patients (after 8 and 11 months) — however, those were not related to new electrocardiographic changes, and no recurrent contractile disorders of TLVAB type were found in ventriculographic examination.

Echocardiographic evaluation, performed in every patient during the long-term follow-up (mean of 12 months), showed normal contractility of left ventricle in all patients.

Discussion

Acute coronary syndromes are a major problem in modern cardiology, being the main cause of death in industrial countries. Angiography is the basic diagnostic method. However, finding normal coronary arteries [6], despite evident clinical signs of myocardial ischaemia, requires further diagnostic steps to determine causes of this condition.

It has been a common belief for many years, that strong emotions are associated with chest pains in the chest, occurence of myocardial infarction, or sudden death. Terms such as "broken heart", "scared to death" or "heart torn with sorrow" express the problem of the relation between emotions and circulatory system. Reports from the past years revealed that in patients with TLVAB neurohormone concentration in peripheral blood serum is significantly elevated. Wittstein et al. [7] proved that in a population of 19 patients with TLVAB the epinephrine, noradrenaline and dopamine as well as their metabolites are significantly higher than in patients with "classical" myocardial infarction, complicated by heart failure. Both authors and our own observation show that a relevant factor triggering TLVAB can be direct emotional stress or physical trauma. There is an interesting observation that in two of our patients the occurrence of the syndrome was related to acute phase of schizophrenia.

Every author [2–4, 7, 8] stressed out that the contraction disorders of the left ventricle is transient and resolves without a trace. This fact has been confirmed in this paper. Most authors considers TLVAB a form of stunned myocardium, however it's mechanism is not fully explained. There is postulated the influence of catecholamines on local myocardial receptors, causing contraction of microcirculation vessels and therefore worsening the heart blood flow [8]. Another hypothesis suggests a direct damage to cardiomyocytes caused by overdose of catecholamines, through overloading of the endoplasmatic reticulum with calcium [9]. This is probably the reason why myocardial necrosis markers are only slightly elevated.

The localization of contractile disorders is not coincidential. The distribution of terminal parts of sympathetic system and local noradrenaline concentrations are significantly greater near the apex of the left chamber than in the rest of the muscle [10, 11]. A disturbance in their function seems to be manifested with the characteristic image of the left chamber.

The clinical course of TLVAB was benign in the presented population, with one exception of occurrence of pulmonary oedema. Also in the long-term follow-up no serious clinical incidents were found. However, reports in the literature prove that in some patients TLVAB may have a very dramatic and even life-threatening course. For example in the work of Desmet et al. [4] out of 13 patients as many as 5 required therapy with aortic counterpulsation because of shock, and in the largest population presented in literature until now [2] acute heart failure occurred in 36,6% of patients, 9% of patients presented dangerous arrhythmias (VT/VF), and in the long-term follow-up two deaths occurred.

Every author agrees that the main criteria of TLVAB diagnosis should be a characteristic contractility abnormalities in ventriculography. Other changes are less specific and may be related to "true" coronary arterial disease. Although most of the described disturbances were observed in our patients, based on them it is difficult to give standards of TLVAB diagnostics. Our observation show that echocardiographic evaluation is associated with a certain disadvantage because of inability to visualise the whole left ventricle, as it is possible in ventriculography, that is why it seems reasonable that when TLVAB is being suspected to be referred for invasive diagnostics. Perhaps in the future, measurements of neurohormones and cateholamine metabolites will improve diagnosing this syndrome.

Conclusions

Transient left ventricular apical ballooning is a new disease, which should be considered in differential diagnosis of acute coronary syndromes. Further investigation is required to improve criteria of diagnosis of this syndrome.

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