Could anintracardiac defibrillatorbreak a heart? Takotsubo cardiomyopathy: rare but real complication of intra cardiac devices implantation "Case Report"

Un défibrillateur peut-il briser un cœur ? La cardiomyopathie de Takotsubo : une complication rare mais réelle de l'implantation de dispositifs intra-cardiaques

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RÉSUMÉ

Nous décrivons le cas d'un patient qui a présenté une douleur thoracique sévère avec une détérioration aiguë de la fonction ventriculaire gauche peu après l'implantation d'un stimulateur cardiaque. A l'échocardiographie, une akinésie des segments apicale et inféro-latérale du ventricule gauche, une coronarographie normale et des critères de diagnostic interTAK validés, la cardiomyopathie de Takotsubo induite par l'implantation d'un stimulateur cardiaque a été diagnostiquée.

MOTS-CLÉS

cardiomyopathie de Takotsubo, défibrillateur automatique implantable, complications

SUMMARY

We describe the case of a patient with severe chest pain and acute deterioration of left ventricular function shortly afterintra cardiac devices implantation. With the documentation of akinesia of the apical and infero lateral portions of the left ventricle, normal coronarography and valid interTAK Diagnostic criteria's, intracardiac defibrillator implantation induced Takotsubo cardiomyopathy was diagnosed.

KEYWORDS

Tako-Tsubo cardiomyopathy, intracardiac defibrillator, complications, case report

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INTRODUCTION

Post-procedural complications are varied and associated with increased patient morbidity, healthcare costs, and even mortality [1]. Takotsubo cardiomyopathy (TTC) causing acute heart failure following implantation is a very uncommon but potentially serious complication [2]. TTC is a form of transient LV systolic dysfunction that predominantly affects ageing women and is frequently but not always precipitated by an emotional or a physical stress[3]. Its diagnosis is very challenging. Our case came to comfort the very few reports that have documented the occurrence of TTC after pacemaker implantation and have proposed TTC as a possible complication of this procedure [2].

PATIENT AND OBSERVATION

A 49-year-old manwithout specific pathological antecedents, no smoking or alcohol habits, was referred to our department for intracardiac defibrillator (ICD) implantation as secondary prevention in dilated cardiomyopathy with angiographically normal coronary arteries complicated with ventricular tachycardia (VT).

On presentation, the patient was asymptomatic; hemodynamically stable with a blood pressure at 110/70mmHg anda heart rate of 50 beats per minute. He had no jugular venous distension, no pulmonary rales, no murmurs in the heart and no edema in lower extremities.

The routine blood test, liver function test, renal function test, thyroid function test, electrolytes and troponin were all normal. The electrocardiogram showed sinus rhythm and Q wave in apico lateral leads (**Figure1**).

A transthoracic echocardiography (TTE) performed five days earlier and revealed a mild depressed left ventricular function with an ejection fraction of 45% and the apex was severely hypokinetic as well as the infero-lateral and the infero-septal walls.

During the procedure, the patient felt severe chest pain and short of breath for about 15 minutes spontaneously alleviated. No fever or signs of heart failure were objected, and vital signs were still stable.

ECG showed sinus rhythm and T wave inversion in antero-septal leads (**figure2**).

Chest radiography excluded pneumothorax and showed normal leads position, however, increase in cardio thoracic index was noticed.

The troponin was 2500 ng/l and 2221 four hours apart. The TTE which was performed after the procedure demonstrated that the LVEF has decreased down to 28 % with no pericardial effusion, apical and infero lateral portions of the left ventricle were akinetic, coronary angiography documented normal coronary flow and no coronary significant stenosis.

The diagnosis of Tako-Tsubo cardiomyopathy was suspected.

The patient was treated with ACE inhibitor and oral diuretics (beta blockers had no place because the patient had a low heart rate)and was discharged two days later.

A week later the patient was seen for follow up assessment of cardiac function. Echocardiography revealed a clot at the apex of the left ventricle and the ejection fraction did not improve.

DISCUSSION

The patient reported in this paper was suspected to have either an acute coronary syndrome or a TTC since he manifested three criteria of the Intertak diagnostic score [4] (table1). He had a score of 49 (table2).

Table1. International Takotsubo Diagnostic Criteria (Inter TAK Diagnostic Criteria)

Criteria	Points
Female	25
Emotional trigger	24
Physical trigger	13
Absence of ST segment depression	12
Psychiatric disorders	11
Neurologic disorders	9
QT_c prolongation	6
Diagnosis (cutoff Value [Range 0-100])	
≥50≤31	
Takotsubo (Specificity 95%)	Acute coronary syndrome (specificity 95%)

Table2. InterTAK diagnostic score

1. Patients with transient[a]left ventricular dysfunction (hypokinesia, akinesia, or dyskinesia) presenting as apical ballooning or other left ventricular segmental (midventricular, basal, or focal) wall motion abnormalities. Right ventricular involvement can be present. Besides these regional wall motion patterns, transitions between all types can exist. The regional wall motion abnormality usually extends beyond a single epicardial vascular distribution. however, rare cases can exist where the regional wall motion abnormality is present in the subtended myocardial territory of a single coronary artery (focal TTS)[b]
2. An emotional, physical, or combined trigger can precede the tako tsubo syndrome effect, but this is not obligatory.
3. Neurologic desorders (e.g., subarachnoid haemorrhage, stroke/transient ischaemicattack, or seizures) as well as pheochromocytoma may serve as triggers for takotsubosyndrome.
4. New ECG abnormalities are present (ST-segment elevation, ST-segment depression,T-wave inversion, and QTc prolongation); however, rare cases exist without any ECGchanges.
5. Levels of cardiac biomarkers (troponin and creatine kinase) are moderately elevated in most cases; significant elevation of brain natriuretic peptide is common.
6. Significant coronary artery disease is not a contradiction in takotsubo syndrome.
7. Patients have no evidence of infectious myocarditis[b]
8. Postmenopausal women are predominantly affected.

[a] Ventricular contraction abnormalities may remain for a prolonged period of time or documentation of recovery may not be possible. For example, death before evidence of recovery is captured.

[b] Cardiac magnetic resonance imaging is recommended to exclude infectious myocarditis and diagnosis confirmation of Takotsubo syndrome.

By exclusion of other diagnosis, we retained the diagnosis of pacemaker induced TTC as the cause of this acute deterioration of the LVEF.

TTC was first described by Japanese in 1990. The cardiomyopathy has got this name because the outline of the left ventricle looks like the octopus Trap. TTC is usually induced by physical triggers, emotional triggers, both and neither of them sometimes. The patients of TTC usually present the symptoms just like acute myocardial infarction or heart failure. Coronary angiography and left ventriculography can make the diagnosis and differential diagnosis [5].Eleven cases of TTC have been reported in a narrative literature review and nearly exclusively affect older womenand it is rarely seen as an adverse event in a man after invasive procedure for ICD implantation [6].

It has been demonstrated that physical stress accounts for 36% of the trigger factors while the emotional stress accounts for 27.7%. Approximately 28.5% patients had no evident triggers whereas 7.8% patients had both the triggers. Furthermore, male patients are more likely to have physical triggers than women [7].

In our patient, the anxiety due to ICD implantation, comparable to a small traumatic surgery, caused TTC typically characterized by chest pain, ECG modifications, slight cardiac enzymes increase and typical left ventricular wall motion abnormalities. TTC has been classified to four types, among them apical type is the most prevalent [8].Our patient was categorized to apical type even though the extent of akinetic area were larger than the typical one, which can be related to the patient underlying cardiomyopathy. It is important to note that while the patient has a history of probable embolic ischemic cardiomyopathy, the mismatch between the typical extended territory of LV dysfunction, the noisy clinical presentation and the slight cardiac enzymes increase cannot be attributed to transient coronary artery embolism.

There are no randomized clinical trials on the specific treatment of TTC [8].TTC is generally a transient disorder that is managed with supportive therapy with the use of common diuretics and vasodilators. Further medication commonly used to treat TTC include beta-blockers and angiotensin converting enzyme inhibitor drugs that promote heart muscle recovery. Intraventricular thrombus is a known complication of TTC especially in patients with severe apical ballooning and reduced LVEF which occurs in approximately 2%-5% of patients[8], as it was described with our patient. Some experts have recommended that prophylactic anticoagulation should be considered to prevent apical thrombus formation, following embolic events in all patients with TTC until LVEF recovery [9,10].

Short- and long-term prognosis of TTC depends on the trigger: patients with an emotional trigger have a better

prognosis whereas those with physical stress have the worst, but the overwhelming majority of TTC cases resolve spontaneously and improvement of the systolic cardiac function is noticed within few weeks[11].

CONCLUSION

Though it is rare, TTC should be suspected after pacemaker implantation in patients with acute heart failure shortly after the procedure. As this case underlines, the prevalence of this syndrome as a serious complication of intra cardiac devices implantation is more common than it used to be. The diagnosis of TTC is challenging because its clinical presentation often mirrors an acute coronary syndrome. The new international diagnostic criteria (InterTAK diagnostic criteria) came to improve identification and stratification of TTC. It is crucial to establish a long-term schedule for observation of these patients to help confirm the diagnosis.

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