The Happiness for Italy's Victory at the European Soccer Championships Costs a “Happy Heart Syndrome”

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ABSTRACT
Takotsubo syndrome (TTS) is usually caused by physical or emotional negative stressors. Sometimes positive emotions trigger a rare form of Takotsubo syndrome, called the “happy heart” syndrome. We discuss the management of a 52-year-old female with happy heart syndrome, the differences between these stress cardiomyopathies and the relationship with hyperthyroidism.

LEARNING POINTS
• The happy heart syndrome is less common than other stress cardiomyopathies but emergency doctors, cardiologists and all specialists in internal medicine should take into account this cardiomyopathy, which occurs in patients with previous positive emotions. It is triggered by opposite stressors to Takotsubo syndrome and sometimes it may be misdiagnosed. Beyond triggers it has some different clinical features, and the management is similar. It is a rare disease, and is therefore underdiagnosed.
• Everybody experiences positive emotions in life but only a small percentage develop stress cardiomyopathy. A susceptibility is needed to trigger these cardiomyopathies such as hyperthyroidism, which has to be promptly treated with an endocrinologist’s help.
• In clinical practice it is common to diagnose stress cardiomyopathies without following up the patients. We need to follow up these patients especially looking for concomitant conditions such as hyperthyroidism or hypersympathetic activity, which could present during follow-up.

KEYWORDS
Takotsubo syndrome, happy heart syndrome, hyperthyroidism.

CASE DESCRIPTION
A 52-year-old woman, with history of arterial hypertension and insulin-dependent diabetes mellitus, presented to our emergency department. She was complaining of an oppressive chest pain radiating to the neck associated with dyspnea, which arose during the celebrations for Italy's victory at the European soccer championship. According to ECG findings of subendocardial ischaemia and an increase in cardiac troponin level, she was admitted to our coronary care unit (CCU) for acute coronary syndrome suspicions.

On ECG, sinus tachycardia was 105/min with diffuse inverted and symmetric widespread T-waves; blood tests also showed an increase in cardiac troponin. The transthoracic echocardiography (TTE) revealed left ventricle dysfunction – ejection fraction (EF) 37% – apical and left ventricle mid segments akinesia associated with basal segments hyperkinesis with typical apical ballooning appearance. According to these findings, Takotsubo syndrome (TTS) suspicions overtook acute coronary syndrome.
An optimal medical treatment based on beta-blockers, diuretics and angiotensin-converting enzyme inhibitors was begun. Despite the low probability of coronary disease, she underwent coronary angiography, which described a normal coronary angiogram. A thyroid function test showed hyperthyroidism with negative thyroid peroxidase antibodies and anti-thyroglobulin. Thyroid scintigraphy did not show any abnormal finding. She was treated for hyperthyroidism with anti-thyroid medications. The chest pain resolved during the first day of hospitalisation and she was asymptomatic until discharge. Before discharge, TTE showed complete recovery in regional and global left ventricle function with a normal left ventricle EF (61%). After more than one month of follow-up, after the initiation of methimazole therapy, there was a complete normalisation of thyroid hormones. After correction of hyperthyroidism, the patient has been event-free for the past two years and has been asymptomatic.

**DISCUSSION**

TTS is an unusual form of acute cardiomyopathy characterised by transient left ventricle motion abnormalities, which occur in the absence of significant coronary artery disease and could cause acute heart failure. TTS is typically triggered by physical or emotional negative stressors such as grief, anger and fear, and is therefore called the “broken heart syndrome”. In contrast, TTS could be triggered by pleasant emotions leading to the happy heart syndrome. TTS may present different left ventricle echo patterns such as apical ballooning and non-apical ballooning, which includes mid-ventricular, basal and focal patterns.

Happy heart syndrome is not classified as a myocardial disease. Its prevalence in the InterTAK registry [1] is 1.1% of all cases and 4.1% of patients with emotionally triggered TTS. The GEIST registry, which enrolled 2,482 patients with TTS, reported similar incidence: 1.5% of all cases and 4.1% within emotionally triggered TTS [2].

Ghadri et al.'s work [3] aimed to analyse the prevalence and characteristics of TTS in patients after pleasant rather than unpleasant events. They state that both positive and negative life events could trigger TTS; nevertheless, negative life events trigger TTS in 95.9% of the cases and only 4.1% are caused by positive stressors. In our series of 100 patients hospitalised from 2007 to 2022 in our CCU, only one patient presented a happy heart syndrome, with a prevalence in our general population of 1%, as reported in the literature. Patients’ characteristics such as sex, mean age and socio-economic status appear to be similar between the two groups as well as symptoms and electrocardiogram results at presentation [3]. According to the GEIST registry, patients with happy heart syndrome were instead more frequently male (18.9% versus 5.0%; p<0.001) [2].

Concerning the Takotsubo echo pattern, patients with happy heart syndrome presented an apical ballooning pattern in 65% of cases. The mid-ventricular pattern was noted in 35.0%. None of the patients presented with basal or focal TTS type. Patients with TTS triggered by negative stressors had apical ballooning in 79.8% and mid-ventricular pattern in 16.3%. The basal TTS type was present in 1.9% and the focal type in 1.9%. Higher prevalence of mid-ventricular involvement was noted in happy heart syndrome, which was significantly different [3](Table 1).

Although the exact cause of TTS remains unknown, several pathogenetic mechanisms have been suggested. Catecholamines seem to play a central role and thyroid hormones interact with a sympathetic nervous system; therefore, hyperthyroidism is associated with hypersensitivity to catecholamines. It is known that emotional distress may cause cardiovascular effects such as life-threatening arrhythmias, TTS and even sudden cardiac death due to sympathetic hyperstimulation and/or parasympathetic withdrawal. The role of happiness and positive emotions is not well known nor described. We barely know the pathophysiology of this cardiomyopathy caused by negative stressors, and we assume that the mechanisms are similar but triggered by different emotions. The molecular pathways and functional connections in the central nervous system involved in the systemic effects are still barely understood.
Happy heart syndrome seems to be caused by pleasant emotional life events such as a birthday party, a son’s wedding, meeting after 50 years with friends from high school, becoming a grandmother and so many others [3]. Nevertheless, although everybody is exposed to stressors in daily life, only a few people develop TTS, and most of these individuals were exposed to stressful events before without an episode of TTS. This fact points to an individual susceptibility or vulnerability and further implies that a certain stress level is required to trigger the occurrence of TTS [2].

Our patient presented a clinical scenario of unknown hyperthyroidism in concomitance with a pleasant and unexpected event such as the Italian victory in the European football championship. The role of the enhancement of the thyroid hormones on the effects of catecholamines is controversial; the metabolic and cardiovascular effects of hyperthyroidism are largely known. Positive emotions tend to be associated with heart rate decreases compared to heart rate increases associated with negative emotion, lower blood pressure [4], and increases in the high-frequency (vagal) component of heart rate variability [5]. These findings are consistent with greater parasympathetic activity counteracting sympathetic effects of positive emotion. This lower adrenergic stimulation during positive emotions compared to stressful events could explain the lower incidence of TTS following deep happiness and probably, as in our case, the happy heart syndrome occurs when two factors are simultaneously present such as a strong positive emotion and an increase in thyroid hormones, which could enhance the effect of catecholamines on the heart.

CONCLUSION
Happy heart syndrome is a rare form of emotionally triggered Takotsubo syndrome (TTS). Joyful triggers account for <5% of emotionally triggered TTS and <2% of all cases. Patients with happy heart syndrome are more frequently male and exhibit a higher prevalence of atypical, non-apical ballooning compared to patients with broken heart syndrome. Long-term mortality and in-hospital complications seem similar in patients with positive and negative emotional events. We need to consider the linkage between hyperthyroidism and Takotsubo syndrome.

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