Prognostic impact of QT intervals in takotsubo cardiomyopathy: still a long way to trap the octopus

"*Takotsubo*" cardiomyopathy (TTC), also called "*apical ballooning*" *syndrome* or *broken heart syndrome* is a heterogenous clinical disorder, first described in 1990 by Sato et al. (1), named "*takotsubo*" because the typical apical dyskinesia of the left ventricle resemble the ceramic pot with a round bottom and narrow neck, used by Japanese fishermen to trap octopuses.

The clinical presentation is much more variable than initially described. Typically, TTC is a syndrome, more common in postmenopausal women, usually triggered by intense emotional and/or physical stress, characterized by a reversible dyskinesia more often involving left ventricular apical segments, changes of ST segment, myocardial enzymatic release, without significant epicardial coronary artery stenoses, that can mimic an acute myocardial infarction (2). However, many cases are described in the absence of acute stress (3) or without an antecedent event (4). Although the prognosis is usually good with complete recovery of ventricular function, prolongation of the QT interval is very frequent and the combination of TTC with another condition associated with the QT prolongation (drugs, hypokalemia, hypomagnesemia, hypocalcemia) (5-7) could adversely affect clinical outcome. Furthermore, TTC is a cause of acquired long QT syndrome and could be a trigger able to unmask latent or unapparent congenital long QT syndrome (8).

The issue of the arrhythmic risk has been neglected in contemporary papers on the TTC. In first important reviews we never read the word "*arrhythmias*". It's likely that the publications about the link between TTC and ventricular arrhythmias, based mainly on case reports, may underestimate the true arrhythmic risk. The possibility that TTC may present as sudden cardiac death further enhance this underestimation (7). Bonello et al. (9), showed that the fatal ventricular arrhythmias were observed in 15% of TTC patients. In this issue of the Anatolian Journal of Cardiology, Song et al. (10) provide an interesting contribution to this too long forgotten topic, examining a wide range of clinical, laboratory, electrocardiographic and echocardiographic parameters in TTC patients with and without QT prolongation. They found that patients with QT prolongation have a worse hemodynamic impairment even if the prognosis is still good. It can be hypothesized that the link between the two conditions can be catecholamine-mediated cardiotoxicity and subsequent intramyocyte calcium overload (11). The intracellular myocyte calcium overload may be responsible for myocyte dysfunction and for QT prolongation and cardiac arrhythmias. The good prognosis, would be in relation to the complete recovery of left ventricular systolic function, typical of this disease. However, little is known about the prevalence of sudden death before admission in patients with TTC and abnormally prolonged QT intervals. Recently, Pant et al. (12) showed that patients with TTC and arrhythmias had significantly longer length of stay, increased cost of hospitalization, and mortality.

There is still a lack of guidelines on the treatment of TTC. However, since it is widely accepted that an increased concentration of catecholamines has an important pathogenetic role in TTC, the use of beta-blockers would theoretically reasonable, despite the absence of large randomized trials. Conversely, it is more doubtful the usefulness of chronic treatment with beta-blockers because the inconsistent results of different study (2, 13). The prevention and treatment of life-threatening arrhythmias should be based on elimination of all the causes that increase the risk of long QT (drugs and electrolyte imbalances). In the case of torsade des pointes, a temporary pacemaker should be implanted and magnesium sulphate administered (7). Therefore, prospective randomized studies designed to assess therapeutic options in patients with TTC are warranted.

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