Takotsubo syndrome: lonely hearts

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Although the nosology of takotsubo syndrome (TTS) has shifted from an acute coronary syndrome–like condition to a heart failure phenotype, its pathology and clinical course still present many unresolved issues. This gap in our knowledge has a significant impact on the various phases of clinical practice: from a challenging differential diagnosis to uncertain therapeutic management and prognosis.

In the current issue of Polish Archives of Internal Medicine (Pol Arch Intern Med), Budnik et al provide their experience regarding 232 patients with TTS from 2 Polish cohorts. The paper confirms the “state-of-the-art” profile of patients with TTS: mostly women, with a great burden of comorbidities, and with a non-negligible rate of in-hospital complications such as thromboembolism and life-threatening arrhythmias, as demonstrated by the current literature.

Indeed, a major concern in TTS arises from evidence of a nonbenign course of the disease, contrary to what was initially thought. In recent years, such findings have prompted a search for predictors of poor prognosis. The paper by Budnik et al confirms that tools from the setting of acute coronary syndromes, such as heart rate on admission or the Global Registry of Acute Coronary Events risk score, have proven utility. However, once high-risk patients with TTS are identified, the choice of the best treatment strategy remains difficult, as their prognosis appears to be burdened not only by cardiovascular but also other mortality.

This uncertainty in outcomes mainly stems from the unknown physiopathology of TTS. How do diverse predisposing factors, such as catecholaminergic excess, genetic polymorphisms, and comorbidities, interplay to induce TTS? How do emotional and physical stresses produce the same parameters on presentation may merely be indicators of a fragile phenotype that is more prone to complications.

In conclusion, though still poorly understood, TTS is an extremely intriguing disease and its study may potentially unravel important mechanisms which act not only at its basis but also in the whole spectrum of cardiovascular disease.

REFERENCES


How to Cite


