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TAKOTSUBO SYNDROME: A THEORETICAL REVIEW OVER A CLINICAL CASE

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The clinical case serves as an introductory note to the description of the Takotsubo Syndrome.

The Takotsubo Syndrome has gained recent notoriety. It is estimated that this diagnosis is made in 2% of acute coronary syndromes. In up to 75% of the cases, a psychological stressor can be found, usually traumatic, and 25-40% of the patients meet the diagnostic criteria for depressive or anxious pathologies. It presents as an acute coronary syndrome, with pre-cordial pain and dyspnea, and in 90% of the cases there is elevated Troponin I, in average 0.49 ng/mL. There is mild ST-wave elevation and T-wave invertion, mostly on V2 and V3. The ventriculography presents the typical image, with apical ballooning and hypokinesia, that reverse in 20 days, average.

The ratio of ß1 and ß2 receptors in the human heart is 4:1, and norepineprhin acts mostly in the former. It is released by the sympathetic terminals in the heart, which are 40% more dense in the basal region. Thus, the catecholaminergic action at the apex is mostly performed by circulating epinephrine, via ß2 receptors. The phenomena that occurs at supra-physiologic levels of epinephrine is called “stimulus trafficking” and results in a net change of intracellular signaling from Gs protein to Gi protein. The effect exists to protect against apoptosis in such conditions, but results in negative inotropism, most evident at the apex, where ß2 receptors are most prevalent.

At last, this poster points some pathophysiologic factors in common between anxious pathologies and Takotsubo syndrome.

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