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Takotsubo syndrome due to peri-anesthesia anaphylaxis, bronchospasm per se, albuterol, or epinephrine? Dissecting the triggers



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The pathophysiology of takotsubo syndrome (TTS), is unknown, and as we are striving to unravel the causes of this mysterious disease, we explore in details the physical and/or emotional triggers, which appear to precede the inception of the illness. Perusing the daily expanding literature of TTS [1] one is left with the impression that in many reported patient cases there are many plausible triggers (some apparent, and other unsuspected) which could have started either singly or in combination, the disease process. Depending on the subspecialty of the authors, emphasis is placed on particular trigger(s), which could have been the culprit of the TTS phenotype, at the exclusion of other possible and plausible offenders. Thus anesthesiologist emphasize anesthetic agents, surgeons the surgical procedure performed, pulmonologist the underlying pathology or drugs administered for its management, and other physicians a large array of associated comorbidities and/or emotional upheavals or trivial everyday life's concerns. A case in point is a recently published paper about a 44-year old woman who suffered a mid-cavitary variant of TTS, associated with pulmonary congestion and a left ventricular ejection fraction (LVEF) of 25%, in the course of anesthesia for an elective abdominal panniculectomy, which procedure was aborted [2]. The patient developed post-tracheal intubation severe bronchospasm, non-responsive to nebulized albuterol and intravenous epinephrine (EPI), and continuous infusion of EPI [2]. The authors focused on the need for allergy skin testing for neuromuscular blocking agents, and specifically rocuronium, the suspected drug for the anaphylactic reaction of their patient, which caused the severe bronchospasm,

not responding to a β_2 -agonist; other possible offenders were propofol and fentanyl, but these 2 drugs are less common causes of anaphylaxis, as the authors remarked [2].

There should be little doubt that the patient had TTS, in spite the ambivalence resulting from the absence of prior echocardiographic (ECHO) evaluation (the patient was asymptomatic, and "she had very good exercise tolerance and could walk and run many miles" [2] making it unlikely that she suffered from an underlying cardiomyopathy), and the absence of post-discharge ECHO follow-up testing, proving the restoration of LVEF to normalcy.

The authors did not speculate about the pathophysiology of TTS, but implicitly attributed it to the anaphylactic response to rocuronium [2]; however TTS could have been due to the physical stress of the severe persisting bronchospasm per se, the β_2 -agonist albuterol, used before and after the emergence of bronchospasm [1], and the administered EPI (50 μ g one time dose, and subsequently intermittent boluses of 50–100 μ g intravenously, along with continuous intravenous infusion of EPI [3], diphenhydramine, or any conceivable combination of the above [1]. There is ample literature on the association of TTS with respiratory distress, intubation, or use of albuterol, diphenhydramine [1], or EPI [3]. Reading carefully the details of this case report [2], reveals the importance of dissecting the possible triggers, which could have mediated a given episode of TTS in a patient.

Conflicts of interest

None.

References

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