Post-Induction Stress-Cardiomyopathy: A Case Report

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Citation

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Abstract

We present a case of a 38 year-old morbidly obese female with an unremarkable cardiac history undergoing elective diagnostic laparoscopy for removal of a left ovarian cyst. Despite modified sequence induction and successful intubation, arterial oxygen saturations continued to decline. Bilateral wheezing was heard on auscultation and shortly thereafter, pink frothy liquid filled the endotracheal tube and circuit. Upon stabilizing the patient, radiography and echocardiogram demonstrated diffuse pulmonary vascular congestion and an ejection fraction of 15%, respectively. Remarkably, despite initial elevation in cardiac enzymes and ECG changes, cardiac function completely normalized by hospital day three.

INTRODUCTION

The incidence of stress-induced cardiomyopathy in the pre- and peri-operative setting has garnered notable attention in recent years [1,2]. Stress-induced cardiomyopathy, also known as Takotsubo syndrome, or transient left ventricular dysfunction syndrome was first described by Sato and Uchida [3], whom rendered the nature of the syndrome by its distinctive wall motion abnormalities to that of resembling an octopus fishing pot with its round bottom and narrow neck. Initial case reports demonstrated findings of hypokinetic wall abnormalities or dyskinesia of the basal segments. However, several case reports since the inception of this syndrome have subsequently revealed the presence of additional characterizations, notably isolated mid-ventricular and basilar hypokinetic “non-apical variants” [4,5]. Historically, this transient left ventricular ballooning syndrome has been seen in postmenopausal women whom have been subjected to recent emotional or physical stress. Although initial signs and symptoms are often suggestive of acute coronary syndrome presenting with chest pain, ECG changes and elevated cardiac enzymes, subsequent studies reveal no angiographic evidence of intracoronary thrombus or plaques. Interestingly, though many of these patients invariably experience congestive heart failure, pulmonary edema, and cardiogenic shock necessitating mechanical support, prognosis is often favorable with eventual complete reversal and resolution of symptoms.

CASE REPORT

This is a case of a 38 year-old Caucasian female with a past medical history remarkable for seasonal allergies, morbid obesity, heavy snoring, and hypercholesterolemia who presented for an elective diagnostic laparoscopy after two months of worsening vague left upper quadrant pain. Subsequent imaging from sonogram revealed the presence of a left ovarian cyst and the patient was scheduled for elective surgery. On the day of admission, the patient expressed her fears of having not undergone surgery in the past and was quite candid in expressing that the impending prospect of surgery was causing her quite a degree of stress and anxiety in recent days. With preoperative testing revealing normal laboratory studies and ECG, the patient was prepared for surgery. In the setting of extreme obesity, a short neck with excess tissue, and Mallampati III airway, the potential of encountering a difficult airway was of particular concern. In anticipation, a bougie and video-assisted laryngoscope was present on standby.

Upon arrival into the operating suite and the patient optimally positioned, routine monitors were applied and pre-oxygenation was commenced. A modified rapid sequence induction approach was utilized with application of cricoid pressure, administration of 2 mg Midazolam, 100 mcg of Fentanyl, 200 mg of Propofol and 100 mg of Succinylcholine. Apnea ensued shortly thereafter and laryngoscopy was attempted with a Macintosh III blade with visualization of a Grade III airway. Unable to fully appreciate an optimal view of the vocal cords, an initial attempt to intubate was aborted. While preparing the bougie, a subsequent attempt at intubation was successful with the airway immediately secured. Despite the presence of a large amount of secretions necessitating suctioning, oral or gastric

contents were not observed.

Sustained end tidal carbon dioxide, bilateral breath sounds were present and the endotracheal tube was taped at 22.0 centimeters. Attributing decline in oxygen saturation to 70% in the setting of a presumed relatively low functional reserve capacity from obesity, the presence of end tidal carbon dioxide, visualization of endotracheal tube placement past the vocal cords provided assurance that oxygenation would eventually normalize. However, oxygen saturations continued to decline to a nadir of approximately 50% in spite of continual administration of 100% oxygen with tidal volumes delivered via manual breathing bag. At this point, the patient had been intubated for one to two minutes with end-tidal Sevofluorane administered and maintained at approximately 0.7-1%. With little improvement in oxygen saturations, additional anesthesia assistance was requested.

Repeat auscultation of lung fields revealed course breath sounds bilaterally, initially with the left greater than right. Furthermore, peak airway pressures were noted to have markedly increased from 17 to 50 mmHg with a concomitant decrease in EtCO2. Suspecting bronchospasm, two doses of albuterol were administered directly via the endotracheal tube with 30mcg of Epinephrine administered shortly thereafter. ECG monitoring at this time revealed sinus tachycardia with heart rates in the 130s-150s, mild ST-elevation and blood pressures between 142-159/89-100.

At this point, approximately 3-4 minutes had elapsed since intubation with little improvement in oxygen saturations. While continuing to support ventilation and vital signs, a large amount of pink, frothy liquid was observed extravasating upwards within the endotracheal tube. Differential diagnoses at the time were negative pressure pulmonary edema, aspiration, severe bronchospasm, congestive heart failure, and anaphylaxis. Diuretics were administered and the patient was placed on mechanical ventilation with application of positive end expiratory pressure. Esmolol and rocuronium 50 mg were administered for elevated heart rates and to facilitate respiratory mechanics, respectively. Shortly after an arterial line was placed, a blood gas obtained on 100% fiO2 was as follows: pH: 7.29 pCO2: 45 pO2: 93 HCO3: 22 O2 Saturation: 96%.

Due to the extenuating circumstances, the case was cancelled and the patient was transferred to the post anesthesia care unit intubated where an urgent cardiology was requested. A preliminary chest X ray (fig.1) revealed the endotracheal tube above the carina and bilateral opacities suggestive of pulmonary vascular congestion. A bedside ECHO (fig.2) performed demonstrated a severely decreased left ventricular systolic function with an estimated ejection fraction of 15%. The patient was found to have normal sized atria and ventricles, severe global hypokinesis, relatively preserved contractility of the basal inferolateral segments, and mild mitral and tricuspid regurgitation. No definite evidence of pulmonary hypertension was found. In the absence of a cardiac history, findings per cardiology were suggestive of a dilated cardiomyopathy, namely a “non-apical variant” of Takotsubo cardiomyopathy.

Figure 1

Fig. 1. Chest X ray obtained shortly after patient stabilized demonstrating bilateral opacities suggestive of pulmonary vascular congestion

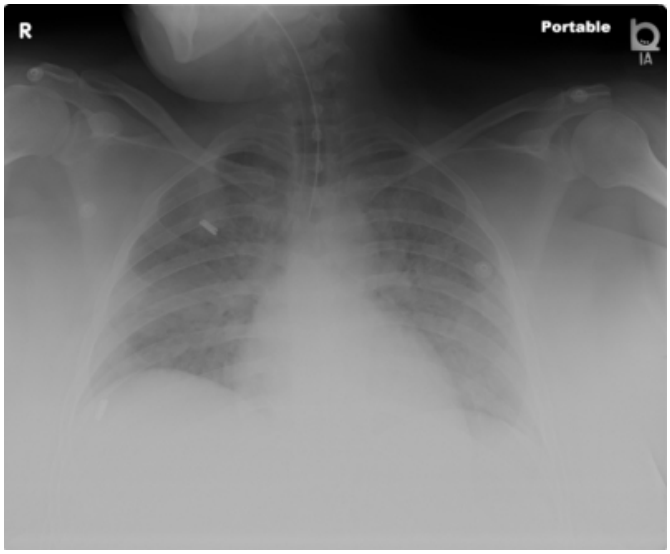
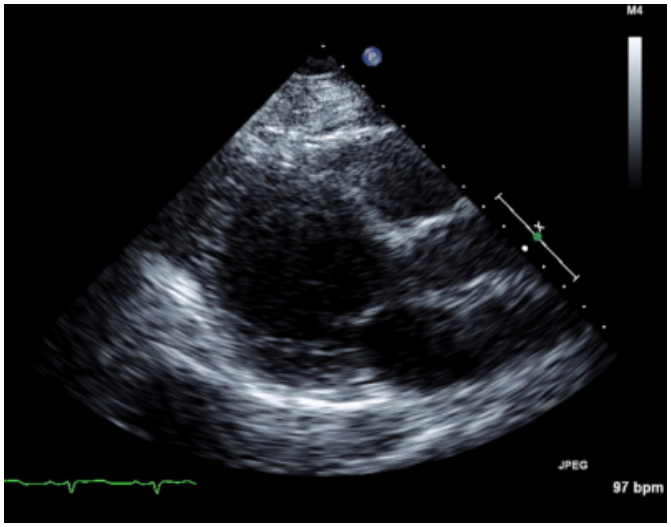


Figure 2

Fig. 2. Imaging from transthoracic echocardiography shortly after arrival to the post anesthetic care unit showing extensive akinesia with preserved contraction in the basal inferolateral portions.



The patient was admitted to the CICU where she remained on ventilator support. Increasing amounts of Fentanyl and Midazolam were necessary for sedation due to continual agitation by the patient while intubated. A femoral central line was placed in the right groin due to inability to gain access via the neck secondary to excess soft tissue. Serial cardiac enzymes shortly after admission to the CICU revealed tropinins of 0.27 [NG/ML], CPK of 49 [IU/L], and myoglobin of 124 [NG/ML]. Values for tropinin, CPK, and myoglobin peaked the following day at 1.98 [NG/ML], 2353 [IU/L], and 385 [NG/ML], respectively. Comparison of the patient's pre-operative initial ECG(fig.3) and ECG taken within after the event (fig.4) demonstrated new QTc prolongation and T wave inversions in I and aVL. A Pro-BNP was relatively unremarkable with a value of 86.

Figure 3

Fig. 3. Preoperative ECG demonstrating normal sinus rhythm, axis, and intervals.

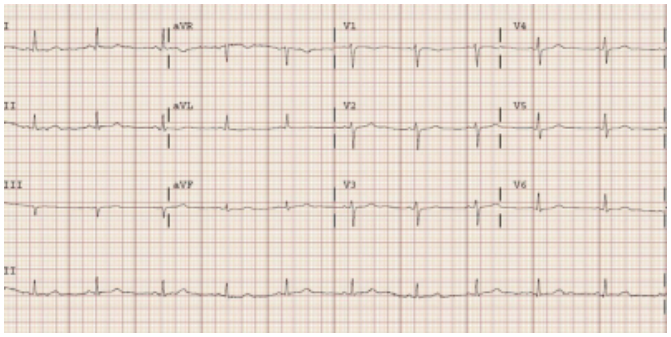
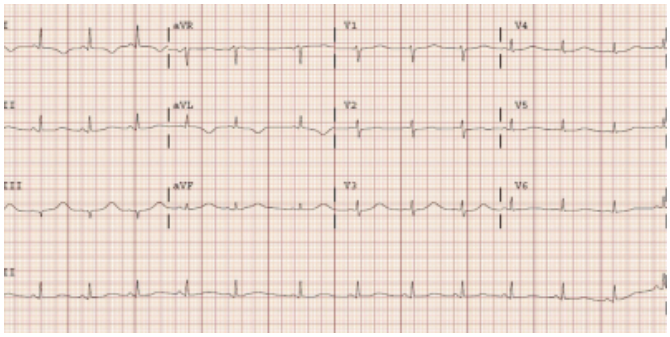


Figure 4

Fig. 4. ECG obtained in the acute stage demonstrating prolongation of the QTc interval, T-wave inversions in the leads I and aVL.



Over the next 24 hours upon admission to the CICU, hemodynamic instability involved pressor support to maintain a mean arterial pressure above 60 mmHg and oxygen saturations ranging from 72 to 96% on 100% fiO2. A subsequent chest x ray demonstrated the presence of pulmonary vasculature congestion suggestive of cardiogenic pulmonary edema without evidence of infiltrate or a chemical pneumonitis. On hospital day 2, bilateral lower extremity duplex scans were performed to exclude the presence of deep vein thromboses that may have served as a nidus for the presence of pulmonary emboli.

Over the next few days, the patient displayed elevated body temperatures with a max of 103° F and a persistent leukocytosis peaking at 32,500 [K/UL]. Despite repeated negative blood cultures, the patient was placed on broad spectrum antibiotics with a concomitant downward trend and eventual resolution of leukocytosis. Serial imaging (fig.5) demonstrated observable improvement in pulmonary congestion by hospital day 3. A repeat echocardiogram (fig.6) performed on hospital day 3 revealed a normal left ventricular systolic function with an ejection fraction of 60%, no evidence of left ventricular hypertrophy, and normal chamber sizes.

Figure 5

Fig. 5. Chest X Ray obtained approximately one week later post extubation demonstrating normal heart size and resolution of perihilar opacities.

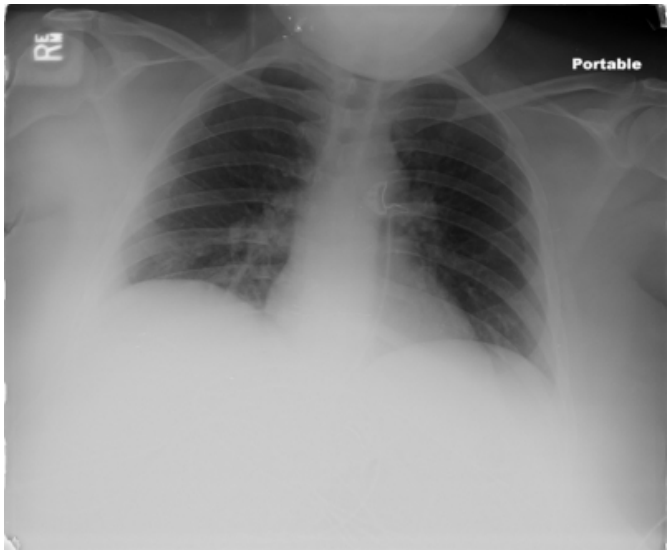
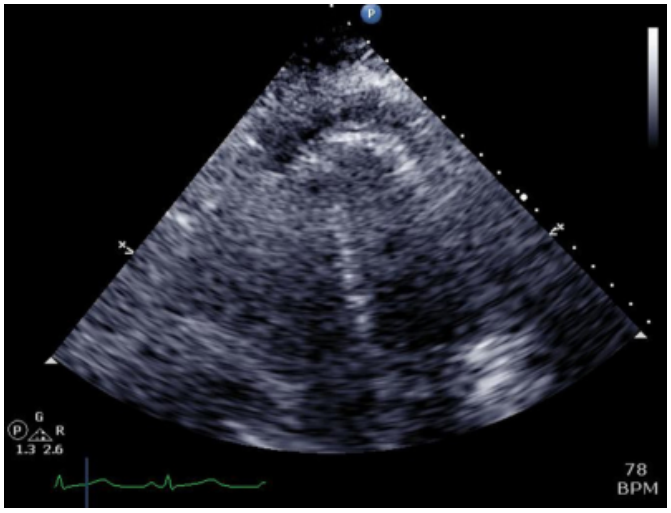


Figure 6

Fig. 6. Despite poor image penetration secondary to patient body habitus, transthoracic echocardiogram on hospital day three demonstrates normal cardiac chambers sizes and ejection fraction.



By hospital day 5, the patient no longer required pressor support as hemodynamics continued to stabilize. Pulmonary function continued to improve as reflected by lower fiO2 requirements, decreased pulmonary vascular congestion, and narrowing of previously widened A-a gradient. Arterial blood gas on day 6 revealed a pH of 7.49 pCO2: 34 pO2: 115 HCO3: 26 and oxygen saturation of 99% on 35% fiO2, however despite this improvement, the patient was unable to tolerate a spontaneous breathing trial. A spontaneous breathing trial attempted the following morning was met with favorable result as she was successfully extubated later that afternoon.

The patient's remaining tenure at the hospital was relatively unremarkable. Although remaining febrile post extubation on broad spectrum antibiotics, cultures failed to reveal growth of organisms. Furthermore, rheumatologic studies failed to demonstrate any evidence of an underlying autoimmune process that may have contributed to the patient's acute decompensation. The patient's remaining hospital course was relatively uneventful with discharge on hospital day 11 on Coreg 3.125 mg b.i.d and Enalapril 2.5 mg daily with plans to follow up as an outpatient with cardiology and gynecology regarding cardiac status and future management of her ovarian cyst, respectively. Post op diagnosis per cardiology was deemed respiratory failure secondary to Takotsubo's cardiomyopathy

DISCUSSION

Stress-induced cardiomyopathy, a now not-so-recently described syndrome has gained substantial notoriety in recent years as described in numerous case reports since it was first described in the early 90's by Sato et al. Often mistaken as a myocardial infarction [6,7], patients typically present with chest pain, pulmonary edema [8], prolongation of the QT interval, ST-segment elevation, T-wave inversions and elevated cardiac enzymes [9,10]. A study by Wittstein and colleagues examined 19 patients at the Johns Hopkins University whom developed left ventricular dysfunction shortly after a sudden emotional stress (e.g. death of a loved one, fear of procedure, surprise reunion) manifested by changes in ECG, echocardiogram, and angiography suggestive of stress-induced cardiomyopathy [11]. Thirteen of the 19 patients evaluated were found to have supraphysiologic levels of plasma catecholamines and stress-related neuropeptides. Furthermore, plasma levels were noted to be several times those of patients with myocardial infarction, remaining markedly elevated up to a week after the onset of symptoms. Previously seen as more prevalent in post-menopausal women, the phenomenon has now been reported in a wide variety of age groups [12,13,14]. Imaging characteristics of transient left ventricular apical ballooning syndrome (TLVAB) often demonstrate contractile abnormalities of the apex and mid-portion of the left ventricle with a relative sparing of the basal segments [15]. Why these regional differences occur with such unique distinction have been hypothesized to be secondary to an abnormal responsiveness in the myocardium influenced by both blood flow and possibly at the level of catecholamine

receptors [16], multivessel transient vasospasm [17], and rupture of atheromatous plaques in the left anterior descending artery [18]. Moriya and colleagues theorized that apical ballooning may be due to a greater density of adrenoreceptors in the apex compared to basal regions within the left ventricle [19]. Given our patient's notable anxiety regarding her upcoming surgery, it is plausible that in the setting of possible supra-physiologic levels of catecholamines, the conglomeration of sympathetic stimulation from prolonged laryngoscopy and subsequent treatment with epinephrine resulted in acute decompensation in cardiac function. However, the exact etiology of the events that transpired in this particular case bring up several questions and considerations that may ultimately provide useful information to the Anesthesiologist with regards to how an anesthetic plan is dictated. Many instances of intraoperative pulmonary edema encountered by the anesthesiologist involve negative pressure pulmonary edema, myocardial ischemia, severe bronchospasm, and fluid overload secondary to aggressive fluid administration.

Initial concerns for the patient's hypoxia raised the possibility that aspiration had occurred due to loss of airway reflexes, obesity, potential for full stomach, and prolonged attempts at laryngoscopy. However, despite copius secretions during laryngoscopy, at no time was there observed gastric contents within the oropharynx. Furthermore, subsequent chest radiographs demonstrated a global event suggestive of vascular congestion which unlike aspiration events, often may resolve within a few days.

Negative pressure pulmonary edema, typically seen in young, healthy athletic adults whom attempt to inspire against an upper airway obstruction [20,21,22] was another consideration. Though our patient did not fall within the characteristic population, her history of snoring and possible upper airway obstruction in the setting of inadequate paralysis could theoretically result in a similar scenario. Lorch and Sahn reviewed several cases of pulmonary edema secondary to upper airway obstruction masking as laryngospasm [21]. Patients harboring risk factors for upper airway obstruction such as short, thick necks and inability to visualize vocal cords whom are also unresponsive to the effects of succinylcholine may indeed have an underlying hypopharyngeal airway obstructive etiology to explain an acute episode of pulmonary edema [21,22]. It is believed that after relief of an upper airway obstruction, the abrupt decrease in airway pressure may result in increased venous return and therefore larger pulmonary blood volumes resulting in acute pulmonary edema [23,24].

Laryngospasm or upper airway obstruction-induced negative pressure pulmonary edema leading to cardiac dysfunction also appears plausible. The effect of negative intrathoracic pressure in augmenting left ventricular afterload by virtue of a more negative transmyocardial pressure for a given pressure head was demonstrated by Buda et al via radiopaque intramyocardial markers during Muller and Valsalva maneuvers [21,25] In the setting of absent airflow and alveolar oxygenation, CNS mediated alpha adrenergic discharge occurs resulting in subsequent peripheral vasoconstriction and centrally shunted blood volumes [21,26]. The increased pulmonary blood volume results in transudation of fluid across the pulmonary membranes via leaky capillary membranes. Fortunately, treatment of negative pressure pulmonary edema revolves around supportive care and generally tends to resolve rather quickly with little to no sequelae.

History and physical exam continues to be a mainstay of any anesthetic plan by providing essential information to the anesthesiologist in dictating the continual but evolving management of care during any procedure. In our patient, bilateral wheezing was noted to be present within minutes after intubation. The numerous etiologies of bronchospasm may confound the anesthetic management in a patient with no history of reactive airway disease. Furthermore, the rapid succession of the multiple medications often utilized for induction make it exquisitely difficult for the Anesthesiologist to identify any potential allergens that may trigger anaphylaxis. A review of the literature implicates induction agents, paralytics, opioids, local anesthetics, and antibiotics in instigating anaphylaxis, bronchospasm and subsequent pulmonary edema [27-31]. Though allergic reactions and anaphylaxis may present with cardiovascular collapse, skin erythema, swelling, and bronchospasm, these attributes are not mutually inclusive and often obscure attempts in arriving at a definitive diagnosis of the causative agent. Hypotension secondary to anaphylaxis can very well be masked shortly after induction as cardiovascular fluctuations are often expected to occur. It is entirely plausible that our patient experienced a severe allergic reaction to one of the medications in our induction regimen resulting in severe bronchospasm and subsequent pulmonary edema. A survey of anaphylaxis during anesthesia demonstrated that cardiovascular symptoms (73.6%), cutaneous symptoms (69.6%, and bronchospasm (44.2%) were the most common clinical features [32].

The management of bronchospasm secondary to anaphylaxis as opposed to addressing an underlying primary cardiac etiology may confound the anesthesiologists' plan of treatment in an emergent situation. With the increasing incidence of stress-induced cardiomyopathy being reported in recent years, it is not unreasonable to hypothesize that an already catecholamine-sensitized myocardium treated with epinephrine can result in an acute decompensation in cardiac function. Administration of epinephrine in the setting of catecholamine excess certainly provides further evidence to the hypothesis that an underlying autonomic nervous system imbalance and overt sympathetic hypersensitivity may exacerbate this particular type of cardiomyopathy. A case of iatrogenic Takotsubo's cardiomyopathy was described by Laínez and colleagues after treating hypotension mimicking as anaphylaxis with high dose adrenalin and noradrenalin [33]. Mast cell proteases such as serum tryptase are often elevated in cases of anaphylaxis, signaling an immune-mediated mechanism [34-37]. Though allergy testing in the form of assessing serum tryptase levels as an indicator of histamine release may aid in the subsequent management of suspected medication or latex allergy, the small window provided by transient elevated serum tryptase levels and its inability to distinguish between anaphylactic and anaphylactoid [38] reactions may limit its usefulness. In addition, skin-testing for potential allergens may often provide useful information on potential allergic triggers as positive tests have a high predictive value in the setting of a history of anaphylaxis [39]. Certainly, information gleaned from such studies may also aid the Anesthesiologists in dictating his or her management plan should a patient require additional surgical procedures at a later time.

Finally, it has been widely observed that the body's physiological response to laryngoscopy and tracheal intubation can manifest as a surge of sympathetic activity, often observed as hypertension and tachycardia [40]. With the advent of various devices in recent years to facilitate ventilation and oxygenation, the anesthesiologist may often be at odds with which particular modality to use in a given situation. A study comparing the hemodynamic and catecholamine stress response to insertion of the Combitube®, laryngeal mask airway (LMA) and tracheal intubation found that insertion of the Combitube® resulted in significantly higher and longer lasting increases in blood pressure, heart rate, and plasma catecholamine concentrations compared with insertion of the LMA or endotracheal tube [41].

Though the existence of several case reports describing the phenomenon of stress-induced cardiomyopathy, the underlying pathophysiology continues to be perplexing and multifactorial in nature. The valid point by Hessell and London on why Takotsubo's cardiomyopathy tends to occur after a similar degree of emotional or physiologic stress or doses of exogenous catecholamines that normally do not affect most patients [42] appears to be the crux of this perplexing syndrome. For now, it would seem logical that mitigating the effect of catecholamine surge through the use of prophylactic beta blockade, centrally acting 2 agonists and preemptive anxiolysis administration prior to surgery may serve to reduce the incidence of stress induced cardiomyopathy for certain patients. However, thus far there has not been a consensus on the optimal approach to the acute or long-term management of these individuals. Thus, Hessel and London propose a movement to encompass groups such as the Society of Cardiovascular Anesthesiologists, researchers, and institutions in establishing a peri-operative Takotsubo's registry to delve deeper into the how and why questions that only collaborative efforts may be able to answer. Global access to such registries may provide further grounds to focus clinical investigations on the possible etiologic, pathophysiologic, and predisposing genetic predispositions that continue to elude us.

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