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The Case of a Broken Heart

It is a standard night in your emergency department when the paramedics wheel in a female patient in her late 50s. They state that they were called to her home where they found her complaining of sudden onset chest pain and shortness of breath. According to the patient, she had accidentally left the door to her house open, and her dog had escaped while she had her back turned. The patient states that she then chased after the dog when it ran into the middle of the street and unfortunately was struck and killed by a car. The patient states that her symptoms developed immediately after. En route, EMS states the patient's 12 lead did not show any ST elevations and her vitals were within normal limits. The patient has no history of ischemic heart disease and no family history of it either. Thinking that this is related to anxiety, you give the patient a small dose of Ativan and send off the chest pain labs and get a formal EKG. The EKG once again shows non-specific ST segment changes but no obvious STEMI; however, her troponin and BNP both return elevated. Being an astute physician, you place the ultrasound probe on the patient to perform an echo. You see that the left ventricular apex is bulging out and the base of the heart is hypercontractile. Now that you know the diagnosis, you give a call to your cardiology friends to discuss the case of a broken heart.

More commonly called Takotsubo cardiomyopathy, the disease is associated with **physical or emotional stress** 85% of the time. It is also called **transient apical ballooning syndrome**, apical ballooning cardiomyopathy, Gebrochenes-Herz syndrome, and stress cardiomyopathy. It is non-ischemic and is caused by weakening of the muscular portion of the heart. Takotsubo is a known cause of **acute heart failure, as well as ventricular arrhythmias and rupture**. Patients usually present with CHF like symptoms, as well as **changes on EKG that appear similar to MIs** of the anterior aspect of the heart. Echo will show the findings seen in the patient above. The name "tako tsubo" is the Japanese word for the pots used to catch octopuses, which is similar in appearance to the heart in this disease. It is most common in postmenopausal female patients. It is usually caused by emotional stressors; however, it has also been seen after physical stressors (i.e. surgery, strokes), as well. Although numbers as high as 85% have been quoted, a paper published in the Journal of the American College of Cardiology entitled "Four-Year Recurrence Rate and Prognosis of the Apical Ballooning Syndrome" by Elesber, M.D., et. al. contradicts this statement. The study was performed to determine how likely the disease was to occur in patients; however, the more interesting aspect of the paper was that they studied 100 patients and when looking at their clinical characteristics, they found that 26 had an emotional stressor prior to the event and 30 had a physical stressor. That left almost half of their patient population without an identifiable stressor. There is definitely a correlation between stressors and the disease, but it is important to have a high index of suspicion no matter the precipitating factors.

The cause of Takotsubo is not entirely clear, but likely is multifactorial. It is more common in patients with the anatomical variant where the LAD supplies the inferior wall of the heart. It is also thought that there is a component of transient vasospasm, microvascular dysfunction, and apical stunning. Also these patients are thought to have an abnormal response to epinephrine and norepinephrine, which are both released during times of stress. Diagnosing these patients is difficult when they present. EKG changes can mimic an anterior wall infarct. It can also

demonstrate T-wave inversion or QT prolongation. Typical symptoms include chest pain and shortness of breath. Bedside ultrasound helps tremendously in the diagnosis, as you can see LV dysfunction and the ballooning of the apex of the ventricle on exam. It is not uncommon for these patients to receive a coronary angiogram, which will show no blockages.

Treatment is usually supportive. No controlled data is available that defines an optimal treatment regimen. These patients are occasionally hypotensive, but use of inotropes can cause the disease to progress and worsen. The **usual treatment is fluids versus diuretics (depending on hemodynamic status and volume status), beta blockers and ACE inhibitors, and in the worst cases, intra-aortic balloon pumps**. Aspirin has not been proven to help the disease; however since the patients first appear like a MI, it is usually given as a precaution. **Most patients will survive the initial event with very good long-term prognosis. Ventricular systolic function usually normalizes within months**. Once they are initially treated, it is important to teach stress reduction techniques, as this is one of the main causes of recurrence.

References

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