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Stress-Induced Cardiomyopathy After an Attempted Trigger-Point Injection

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Background: Stress-induced cardiomyopathy (SIC), also known as Takotsubo cardiomyopathy, resembles myocardial infarction with transient regional wall-motion abnormalities of the left ventricle and is often associated with physical or emotional stress. Unlike myocardial infarction, coronary angiography reveals no significant evidence of coronary artery disease or plaque rupture. The pathogenesis of SIC remains unclear, but it is proposed to be related to increased catecholamine release causing cardiotoxicity.

Case Report: An 85-year-old woman presented with dizziness, nausea, and weakness immediately after an attempted trigger-point injection procedure. She was later found to have elevated cardiac markers and a negative coronary angiogram, and was subsequently diagnosed with SIC.

Conclusions: Increased awareness of SIC during common medical procedures may improve its recognition and underscore the importance of managing patients' procedural stress.

Key words: Stress-induced cardiomyopathy, trigger point injection, procedural anxiety

BACKGROUND

Stress-induced (aka Takotsubo) cardiomyopathy (SIC) is triggered by emotional or physical stress and has a clinical presentation which imitates an acute myocardial infarction (2). SIC is associated with transient left ventricular apical ballooning with the absence of ischemic coronary occlusion (1). Often referred to as "broken-heart syndrome," this rare hyperreaction to increased catecholamine release occurs most commonly in postmenopausal women (3). An evaluation of the 2008 Nationwide Inpatient Sample database by Deshmukh et al (3) found 90.4% of SIC admissions were women. Moreover, women over the age of 55 had 4.8 times higher odds for developing SIC compared to women under the age of 55 (3). Trigger-point injection therapy is a common low-risk intervention used for patients with a distinct, hyperirritable area located in a contracted band of the skeletal muscle. We describe a case of SIC provoked by an attempted trigger-point injection in an outpatient pain clinic.

CASE

An 85-year-old woman with a past medical history of hypertension on triamterene-hydrochlorothiazide and cervical spondylosis without myelopathy was seen in an ambulatory pain clinic for a cervical trigger-point injection for cervical myofascial pain. She did not have a history of coronary artery disease, congestive heart failure, anxiety, or depression. Preprocedure vitals were as follows: blood pressure was 131/74, heart rate was 78, and oxygen saturation was 97% on room air. Prior to the procedure, she expressed anxiety about the procedure, as she previously had cervical medial branch block injections, which were very painful and did not provide any relief. The patient was counseled about the trigger-point injections and procedural pain, and she was placed in an upright and seated position for the injection. A 30-gauge 1-inch needle with a mixture of 8 mL of bupivacaine 0.25% and 20 mg of methylprednisolone was prepared for the injection.

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After negative aspiration 0.5 mL was injected into the right cervical paraspinal muscle, the patient began to complain of dizziness, lightheadedness, and nausea. The procedure was aborted immediately, and the needle was removed. The patient's vital signs were stable with adequate oxygen saturation (98%), heart rate (64), and blood pressure (144/83). She was taken to our recovery room, where she had multiple episodes of emesis. The patient was given 4 mg of sublingual ondansetron. Her vital signs remained stable throughout this period. After monitoring the patient in our recovery room for over an hour, she continued to feel nauseated, dizzy, and weak. She was transported to the emergency department (ED) for further evaluation.

In the ED, she had mild improvement of nausea but continued to complain of lightheadedness. Her blood pressure was 155/77, heart rate was 61, and oxygen saturation 99%. She denied chest pain, chest pressure, shortness of breath, or focal neurologic deficits. An electrocardiogram was negative for ST elevation, depression, or T-wave inversions; however, her troponin was 68 with a 2-hour repeat of 247, and 6-hour troponin of 344 (Table 1) indicating myocardial injury. Brain natriuretic peptide was less than 50 suggesting this reaction was not related to heart failure. She was admitted for further evaluation. Transthoracic echocardiogram showed an ejection fraction of 58% with regional wall-motion abnormalities in the inferior, inferio-septal, and inferolateral segments of her mid-ventricle (Fig. 1). Coronary angiogram did not show evidence of obstructive disease and no intervention was required (Fig. 2). She was diagnosed with SIC. The patient did not experience any seizures, ringing in the ears, or metallic taste in her mouth after 0.5 mL of the injectate was administered and she was hemodynamically stable, thus making local anesthetic toxicity an unlikely cause of her symptoms. She was discharged home 2 days after her injection and was started on a beta blocker, ACE inhibitor, and aspirin. Upon follow-up with cardiology, it was determined there was no need for aspirin as she did not have an ischemic event. This allowed her to resume naproxen for her neck pain.

Table 1. Troponin levels of patient indicating myocardial injury.

Component	Patient's Result
Troponin T, Baseline (< = 10 ng/L)	68
Troponin T, 2 hour (< = 10 ng/L)	247
Troponin T, 6 hour (< = 10 ng/L)	344

On 4-month follow-up, the patient was asymptomatic. She denied resting or exertional chest pain, shortness of breath, palpitations, or orthopnea. Repeat echocardiogram showed an ejection fraction of 60% and an improvement of the regional wall-motion abnormalities. She will follow-up in one year for a repeat echocardiogram.

DISCUSSION

While the exact pathophysiology of "stress-induced" or "Takotsubo" cardiomyopathy is not well established, it is proposed to be secondary to high serum catecholamine concentration resulting from exogenous or endogenous stress (4). An acute flux of plasma epinephrine may act on the densely concentrated beta-adrenoreceptors in the apical myocardium, causing reversible regional left ventricular dysfunction and abnormal wall movement (4,5). SIC typically presents similarly to myocardial infarction, with common symptoms including chest pain, dyspnea, elevated cardiac biomarkers, and transient echocardiogram abnormalities (1). However, further assessment will reveal a lack of ischemic coronary occlusion or necrosis. Indeed, approximately 1%-2% of patients with SIC (1) are initially misdiagnosed with acute coronary syndrome or myocardial infarction. The patient did not present with the most common symptoms of SIC: chest pain (75.9%), dyspnea (46.9%), or syncope (7.7%) (6), although she did report lightheadedness and feeling weak. Despite her atypical presentation, she meets all 4 of the Mayo Clinic's diagnostic criteria for SIC (7): transient hypokinesis of the left ventricular mid segments often in the setting of a stressful trigger, absence of obstructive coronary disease, or angiographic evidence of acute plaque rupture, new electrodiagnostic abnormalities, or a modest elevation in cardiac troponin, and absence of pheochromocytoma and myocarditis. We propose that our patient's emotional stress in anticipation of receiving trigger-point injections increased her serum catecholamine concentrations, precipitating SIC.

Local anesthetic systemic toxicity and vasovagal reaction were also considered as potential etiologies. Inadvertent intravascular injection of local anesthetic is a possibility in the cervical area. Aspiration was negative before injection; however, a 30-gauge needle was used, making aspiration difficult. The patient had dizziness and nausea, which could have been early central nervous system signs of local anesthetic toxicity (8). Nonetheless, she did not display the classic initial symptoms of local

anesthetic systemic toxicity, which include ringing in the ears, a metallic taste in the mouth, or circumoral tingling (8). She was also hemodynamically stable throughout the process. Our patient had dizziness, lightheadedness, and nausea, which are symptoms of vasovagal syncope. Her heart rate and blood pressure did not decrease, which is commonly seen during a vasovagal reaction. Additionally, vasovagal reactions are generally brief, and our patient's symptoms continued for over an hour, which prompted transportation to the ED.

Trigger-point injections are generally considered safe procedures. Complications of trigger- point injections include infection, pneumothorax, myonecrosis, myopathy, or pain at the injection site (9). This case displays examples where caution should be considered in elderly patients and nonimage-guided injections in the cervical area, which both increase risk of trigger-point injection adverse reactions.

After an extensive review of the literature, this case is, to our knowledge, the first published report of SIC due to a stress-response and/or procedural anxiety preceding a trigger-point injection. Fortunately, the prognosis is typically favorable with supportive treatment, and in-hospital mortality is from 0% to 10% (2). Increased awareness of SIC during common medical procedures may improve its recognition and underscore the importance of managing patients' procedural stress.

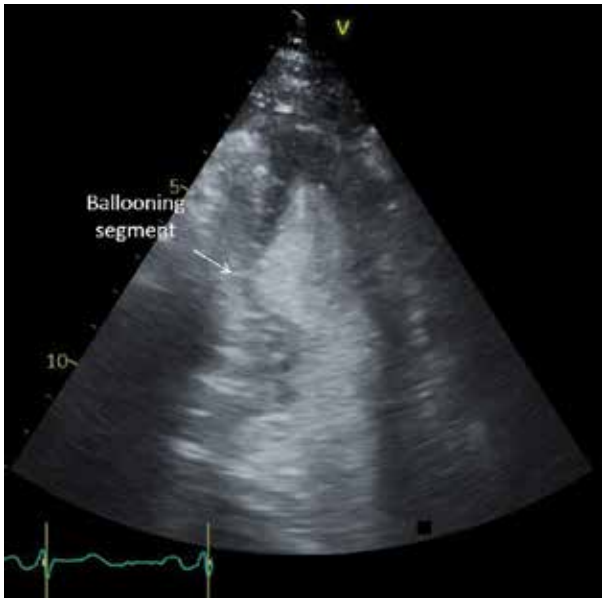

Fig. 1.Echocardiogram shows an isolated inferior/inferolateral midventricle wall motion abnormality.

CONCLUSIONS

A case of SIC after an attempted trigger-point injection therapy emphasizes the significance of managing a patient's procedural anxiety.

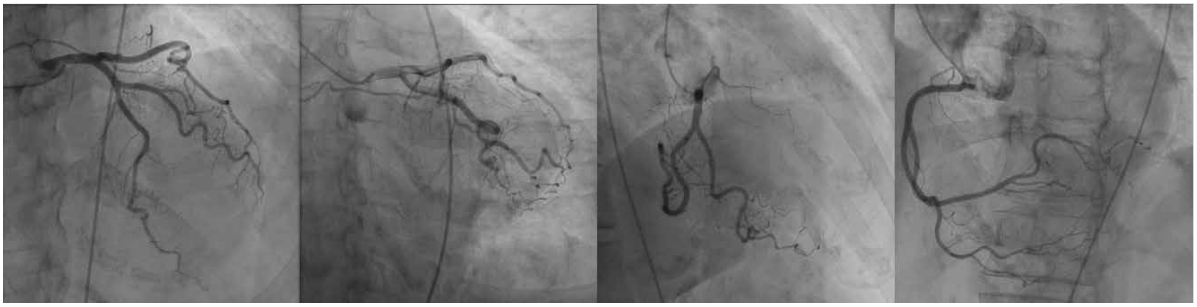
Fig. 2. Coronary Angiogram.

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