Missile Alarm as a Trigger for Vasovagal Syncope: A Case Report from a Conflict Zone

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> We report a case of true syncope with documented vasovagal syncope which occurred at the time of an alarm due to missile attack. This case shows a severe vasovagal reaction to emotional stress and fear.

The vasovagal response (neurocardiogenic response) involves an abnormal interplay of bradycardia and paradoxical vasodilation. This intense vagal stimulation can result in syncope, which may have dramatic consequences, especially in elderly patients. Such an event was triggered during a missile alarm in an 80-year-old man. To the best of our knowledge, no similar case has been previously reported [1,2]. A sinus pause is a temporary cessation of electrical impulses from the sinus node, resulting in a brief but complete halt of heart contraction. The excessive vagal input diminishes sinus node automaticity, potentially causing pauses that vary in duration from seconds to even longer periods, depending on individual susceptibility and the length and intensity of the vagal response.

Although typically transient, sinus pauses may also result in significant symptoms like lightheadedness or syncope. In severe cases, they can compromise cerebral perfusion, necessitating immediate intervention. Recognizing the risk of sinus pause in susceptible patients is essential for preventing potential complications, particularly during procedures that may provoke a strong vasovagal reaction.

PATIENT DESCRIPTION

An 80-year-old male with a medical history of hypertension, dyslipidemia, diabetes, and peripheral vascular disease was hospitalized due to unstable angina. Three years prior, he underwent transcatheter aortic valve implantation due to severe aortic stenosis. Echocardiography demonstrated preserved ejection fraction with anterior wall motion abnormalities, and no significant gradients across the aortic valve. Coronary angiography revealed severe narrowing of 80% in the left main coronary artery (LM-CA), and 80% in the proximal left anterior descending artery (LAD). He underwent intravascular ultrasound-guided percutaneous coronary intervention (PCI) from the LMCA to the LAD, achieving good angiographic results. He recovered well post-PCI. No arrhythmias were recorded. Troponin was not elevated.

The next day, 25 October 2024, at 18:15, a loud siren alerting a possible missile attack sounded across the entire region and the medical center due to rockets fired in the area. The patient and the rest of the coronary intensive care unit patients and staff were promptly evacuated to a designated protective area. This required a short walk of 25 meters. Moments after arrival, with no prodrome, the patient collapsed. The patient appeared pale, with no urine or fecal incontinence, cardiopulmonary resuscitation was initiated. He regained consciousness after less than one minute. During this short event, no blood pressure measurement was done. Tele-monitoring revealed two episodes of ventricular arrest lasting 6 and 4 seconds [Figure 1]. Of note, in the tracing it is not excluded that a blocked P was present at two instances in the middle tracing. Following the event, a head computed tomography scan excluded intracranial bleeding.

Following multidisciplinary discussion, it was decided that, due to patient's advanced age and current pacemaker guidelines [3] as well as exclusion of other causes of asystole, to implant a dual-chamber pacemaker. No electrophysiology study was conducted due to the nature of the collapse, which was highly suspicious of vasovagal syncope. The patient was subsequently discharged in stable condition.



Figure 1. Tele-monitoring during syncope showing the severe vasovagal sinus arrest

on syncope state that permanent pacemaker therapy may be effective if asystole is a dominant feature of reflex syncope, and that the efficacy of pacing depends on the clinical setting [3]. This case broadens the understanding of indirect cardiovascular risks associated with war beyond direct physical harm.

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COMMENT

Vasovagal response is the most common mechanism of syncope. A variety of mechanisms can cause vasovagal responses [1-3]. Central triggers, such as severe pain, anxiety, fear, and emotional stress, can cause vasovagal response. Syncope and hemodynamic collapse can occur due to pain, anxiety, and fear during medical procedures, such as vascular access or induction of neuraxial anesthesia [4,5]. Usually, past medical history reveals previous vasovagal episodes and susceptibility.

The present case underscores an unusual cause of vasovagal syncope triggered by the severe physiological stress associated with recurring missile alarms. The combination of isolation, repetitive alarms, and the physical exertion of swift evacuation to a safe area, likely increased vagal tone, precipitating vasovagal syncope. Pacemaker implantation offered a preventive measure against further episodes, underscoring the importance of considering cardiac protection strategies for at-risk individuals. Current ESC guidelines

Capsule

Neoadjuvant triplet immune checkpoint blockade in newly diagnosed glioblastoma

Glioblastoma is an aggressive primary adult brain tumor that rapidly recurs after standard-of-care treatments, including surgery, chemotherapy and radiotherapy. **Long** et al. presented a case of newly diagnosed IDH-wild-type, *MGMT* promoter unmethylated glioblastoma treated with a single dose of neoadjuvant triplet immunotherapy (anti-programmed cell death protein 1 plus anti-cytotoxic T-lymphocyte protein 4 plus anti-lymphocyte-activation gene 3) followed by maximal safe resection 12 days later. The anti-programmed cell death protein 1 drug was bound to tumor-infiltrating lymphocytes (TILs) in the resected glioblastoma and there was marked TIL infiltration and activation compared with the baseline biopsy. After 17 months, there is no definitive sign of recurrence. If used first line, before safe maximal resection, checkpoint inhibitors are capable of immune activation in glioblastoma and may induce a response.

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