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Laughter Induced Syncope: A Case Report

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Abstract
Syncope is a transient loss of consciousness and postural tone secondary to inadequate cerebral perfusion that spontaneously resolves without medical intervention . Laughter-induced syncope is rare and likely goes unrecognized by many health care providers. It is thought to be another form of valsalva induced syncope. The laughter-induced syncope is an actual medical term, and it is also known as the "Seinfeld syncope" because one reported case occurred when an unfortunate patient died while watching a particularly funny episode of the popular TV show Seinfeld*. Laughter-induced or gelastic (derived from the Greek word for laughter, 'gelos') syncope is extremely rare. It is a sub-type of the situational syncopes hypothesized to be the result of a neurally mediated reflex triggered by increased intrathoracic pressure.*

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I. Introduction

Syncope is defined as a transient loss of consciousness resulting from global hypoperfusion of the cerebral cortex or reticular activating system. The loss of consciousness generally lasts less than a minute, and is followed by a rapid return to baseline neurologic status without any medical intervention . It is a relatively common clinical problem accounting for 1% to 1.5% of emergency department visits and around 6% of hospital admissions annually . Among the various classifications, neurally mediated, cardiac and unexplained etiologies appear to be the most common diagnoses. In a prospective study of 341 patients presenting with syncope, a cardiac cause of syncope was established in 23% of the patients, a neurally mediated cause in 58% and the cause of syncope remained unexplained in 18% .

CLASSIFICATION OF SYNCOPE

Cardiac
-Aortic stenosis, hypertrophic cardiomyopathy, pulmonary embolism, aortic dissection, myocardial infarction, left atrial myxoma, cardiac tamponade, atrioventricular block, sick sinus syndrome, tachyarrhythmia, bradyarrhythmia

Neurally mediated (reflex mechanisms)
Vasovagal, situational (micturition, laughter, tussive, defecation, postprandial, sneeze, swallow), orthostatic syncope, carotid sinus syncope

Neurologic
Transient ischemic attack, subclavian steal syndrome, Takayasu disease, seizure

Metabolic
Hypoxia, hypoglycemia, hyperventilation

Psychiatric
Panic disorder, conversion reaction, hysteria

Drug-induced Vasodilators (nitrates, calcium channel blockers, angiotensin-converting enzyme inhibitors), phenothiazines, antidepressants (tricyclic agents, monoamine oxidase inhibitors), central nervous system depressants (barbiturates), drugs associated with torsades de pointes (quinidine, procainamide, disopyramide, amiodarone, sotalol, flecainide), diuretics, digitalis, insulin, marijuana, alcohol, cocaine .

Unknown Origin

II. Case Study

A 52-year-old, obese (body mass index of 35) man with a past medical history of hypertension and hyperlipidemia who suffered from syncope secondary to intense laughter. Staff nearby who had witnessed the event immediately found his blood pressure to be 109/60 mm Hg with a heart rate of 96 beats/min. He was sent to the emergency department, and electrolyte, blood glucose, complete blood count, electrocardiogram, and echocardiogram testing results were normal. The patient also had a history of syncope in the distant past when he collapsed on the floor for several seconds. There were no prodromal symptoms such as coughing before the syncope and patient completely loss and could not arosed for 2-3 minutes. He then awoke spontaneously and felt rather weak for further 20-30 min. After the attack, there were no seizure-like movements, or bowel or bladder incontinence. He had no episodes of narcolepsy or sleeping disturbance. An electrocardiogram (ECG) was normal, and a 24-hour Holter ECG showed neither arrhythmia nor bradycardia leading to syncope. No abnormality (regional wall , valvular lesion) was detected by echocardiography. The results of other examinations, including electroencephalography, cranial computed tomography, and carotid artery ultrasonography, were normal. To rule out ischemic heart disease, Tread mill Test was performed, but the result was negative for ischemia and arrythmia. Since the syncope occurred during laughing, we assumed he had a hypersensitive carotid sinus, but carotid artery massage did not lead to hypotension or syncope.

III. Discussion

Laughter-induced or gelastic (derived from the Greek word for laughter, 'gelos') syncope is extremely rare. It is a sub-type of the situational syncopes as the result of a neurally mediated reflex triggered by increased intrathoracic pressure. Intense laughter causes repetitive forced expirations in a staccato pattern with a Valsalva-type effect. The associated increase in intrathoracic pressure reduces venous return resulting in decreased cardiac output and a transient reduction in cerebral perfusion. It has also been proposed that strenuous laughter might produce isometric muscle contraction resulting in acute vascular dilatation, thereby exacerbating the reduction in venous return . In one of the most well-known reflex arcs, reduced cardiac output leads to decreased stimulation of carotid sinus and aortic arch baroreceptors, as well as mechanoreceptors in the left ventricle wall . The resulting increase in sympathetic tone maintains blood pressure for adequate cerebral perfusion. However, in neurally mediated syncopes, there is acute and inappropriate hypotension and bradycardia exacerbating the reduction in cerebral perfusion, resulting in a transient loss of consciousness.

It is hypothesized that increased ventricular contraction in response to reduced venous return stimulates the left ventricle mechanoreceptors to a degree that is able to override the baroreceptor reflex and cause an inappropriate increase in parasympathetic tone . Aside from laughter-induced syncope, this mechanism is also thought to account for syncope secondary to coughing, sneezing and other Valsalva-related activities.

Laughter-induced syncope is rare and has only been described a few times in the literature.[2] The cases described in the literature had a similar presentation as in this case, with the exception that some patients had a short prodrome before the syncope occurred. The described cases also share negative laboratory, cardiac, neurological and imaging studies including negative tilt table studies.

The diagnosis of laughter-induced syncope presents a challenge as the rarity of this disorder means there are no standard investigations. The evaluation of patients with syncope remains a frequent challenge facing emergency physicians. Careful history taking, examination, and the judicious use of testing, if needed, will allow physicians to diagnose many patients with benign, neurally mediated, reflex syncope events.

The patient's physical examination should include orthostatic blood pressure, heart murmurs and carotid bruits, and a basic neurological examination; an electrocardiogram should also be considered. Basic laboratory testing such as complete blood count and a comprehensive metabolic panel should be considered (Strength of Recommendation Taxonomy (SORT), level C). History and symptoms that are suggestive of more serious causes of syncope include a family history of sudden death, chest pain or palpitations before or during the event, shortness of breath, seizure activity, heart murmur, focal neurological deficits and a loss of consciousness lasting more than 5 min.−Depending on the findings from this initial evaluation and risk stratification, a decision can be made as to whether further more advanced testing is required.

To the best of our knowledge, no therapeutic approaches have been established for laughter-induced syncope. This disorder is treated by prevention and patient awareness of the problem. All patients described had symptoms lasting seconds to a few minutes and returned to consciousness when they were in a head down position or lying flat. Few reported case of laughter-induced syncope successfully treated with midodorine hydrochloride, an alpha1-stimulator, and propranolol. The alpha1-stimulator increases venous return to the heart through the vasoconstriction of veins and elevates blood pressure via the vasoconstriction of peripheral arteries. As a consequence, it could increase brain blood flow and prevent the Neurally mediated syncope .

The prognosis for patients presenting with syncope varies according to the underlying etiology One population-based study found that cardiac and neurologic syncope were associated with an increased risk of death from any cause and an increased risk of cardiovascular events and stroke, respectively . By comparison,

patients with vasovagal, orthostatic, medication-induced and situational syncope had no increase in the risk of death from any cause compared with patients without syncope . Although laugh syncope was not specifically addressed in this study, as a type of situational syncope, we extrapolate that its prognosis is likely benign.

IV. Conclusion

-Laughter-induced syncope is a rare entity.

- Laughter-induced syncope should be treated through prevention and patient awareness.

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