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Case Report

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Reflex Abdominal Syncope: A Case Series

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Abstract

The etiology of syncope in the acute care setting requires considerable input from numerous specialties. During a recent analysis of patients presenting to the acute care setting with syncope and identification of underlying associated disorders, we identified a subdiagnosis presenting with gastrointestinal associated bradycardia. We report here four cases of this syndrome, where exacerbation of abdominal pain was associated with bradycardia and subsequent syncope. Patients were evaluated with European Society of Cardiology-based standards, including cardiovascular and neurologic testing, which was unremarkable. Treatment of patients for their underlying gastrointestinal disorders (gastritis, gastroenteritis, diverticulitis) resulted in resolution of syncopal symptoms. Such reflex abdominal syncope should be considered in patients presenting to an acute care setting with abdominal pain and transient losses of consciousness.

Keywords: Syncope; Gastroenteritis; Gastritis; Bradycardia; Reflex abdominal syncope

Introduction

Syncope is defined as an abrupt, transient, and complete loss of consciousness associated with inability to maintain postural tone followed by rapid and spontaneous recovery [1]. The underlying mechanism is presumed to be cerebral hypoperfusion which may occur due to systemic vasodilation, decreased cardiac output, or both, although various cellular and molecular mechanisms exist [2–5].

Syncope represents a frequent presentation to the emergency room with up to 3% of all cases entering an emergency department (ED) presenting with transient loss of consciousness [6]. Classifications of syncope have been primarily grouped into cardiac etiology, reflex syncope, and orthostatic, although other manifestations exist [4, 7].

Presentations to the acute care setting often prompt cardiac and neurologic consultation, but recent data suggest other areas associated with the diagnosis of syncope [7]. Indeed, recent emphasis from international bodies (e.g., European Society of Cardiology (ESC)) highlights the assessment of syncope as a symptom, and the need to determine the underlying etiology [1]. Identification of these other etiologies could expedite diagnosis and management.

In particular, gastrointestinal (GI) etiologies have limited data, despite representing up to 6% of presenting cases to the ED. We present four cases of significant GI symptoms (abdominal pain) associated with resultant witnessed syncopal episodes. These cases are in addition to other cases recently presented [7]. Each had prominent GI symptoms, and witnessed recurrent losses of consciousness after presenting to the ED. Such "reflex abdominal syncope" was ameliorated with treatment of the underlying GI disorder.

Case Reports

Case 1

A 27-year-old man with no significant medical history presented to the emergency room with significant epigastric abdominal pain. History revealed a syncopal event several hours prior while sitting in bed, witnessed by his spouse, lasting for approximately 1 min. The patient did not recall the event. There were no associated diaphoresis or palpitations. The patient endorsed lightheadedness. In the ED, patient was given intravenous (IV) fluids and placed on cardiac monitoring. Initial blood pressure was 128/84, heart rate 72, temperature 37.8, and respiratory rate 16. While in the ED, patient experienced another acute episode of severe abdominal pain and sat up; he experienced another acute episode of syncope. Heart rate decreased to approximately 35. IV fluids were administered and patient recovered in 1 to 2 min. Patient was admitted and evaluated by Cardiology and Neurology. Workup included electrocardiogram (EKG), echocardiogram, tilt table testing, computed tomography/computed tomography angiography (CT/CTA), magnetic resonance imaging (MRI), and electroencephalogram (EEG), all of which were unremarkable. The patient was diagnosed with gastroenteritis and treated empirically with no further episodes during the remainder of his hospitalization. No recurrence of these symptoms was noted in follow-up 8 months later.

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Case 2

A 73-year-old woman with a history of Alzheimer dementia, hypertension, and chronic obstructive pulmonary disease (COPD) was sent to the ED with a witnessed syncopal event while at memory care center. She had been complaining of abdominal pain and had vomited twice before arriving at the ED. Vital signs included a respiratory rate of 20, heart rate of 78, blood pressure of 140/88, and a temperature of 37.8 °C. The patient was admitted to telemetry for further assessment; on the night of admission, the patient developed acute abdominal pain while sitting, and experienced syncope. Heart rate was noted to be 48 with a systolic blood pressure of 70. A stroke alert was activated, with CT and CTA acquired and both negative for acute findings. The patient recovered within approximately 1 min. Heart rate improved to 88 and blood pressure rose to 140/80. Patient was subsequently evaluated by cardiology; EKG, tilt table testing, and echocardiogram were negative. MRI was notable for diffuse atrophy but otherwise unremarkable. She was subsequently diagnosed with acute gastritis and treated symptomatically with pain medications and IV fluids. The patient was discharged without recurrence of syncope. Outpatient follow-up 3 months later was negative for recurrence of transient losses of consciousness.

Case 3

A 46-year-old man with a history of hypertension, diabetes, 20-pack-year history of smoking, undomiciled status was brought to the emergency room by emergency medical services (EMS) secondary to complaints of abdominal pain and witnessed loss of consciousness at the bus station. EMS noted his blood pressure at the scene was 120/70, with a heart rate of 90; the patient was afebrile. The patient recounted that he had had 2 days of diarrhea and vomiting; it was unclear if the patient had a fever. In the ED, the patient had acute abdominal pain and collapsed while trying to get up out of the gurney. Patient's heart rate was noted to be approximately 48. The patient suffered a bruise on the left side of his forehead with a CT and CTA being unremarkable at the time. EKG and bedside echocardiogram were performed, which showed no other abnormalities and a heart rate of 88. The patient subsequently eloped from the ED, so no further workup was possible.

Case 4

An 81-year-old man with a history of hypertension, diabetes, prostate cancer, and marijuana use was brought in by ambulance because of a syncopal event. The patient had complained of intermittent abdominal pain during the week and had a history of "irritable bowel disorder." That evening, approximately 1 h after eating, the patient developed severe abdominal pain and was unresponsive while watching television with his family. In the ED, the patient's blood pressure was 180/90, with a heart rate of 90, respiratory rate of 20, and a temperature of 37.4 °C. The patient recalled the event, noting that he was lightheaded and "the lights went out" and he woke up with his family around him. In the ED, he experienced an acute episode of abdominal pain and lightheadedness with subsequent brief loss of consciousness. Heart rate was at the time 50, and the patient was repositioned from a sitting position to a supine position with administration of IV fluids. Patient noted that this was similar to his previous episode. The patient was admitted; CT/CTA and EEG were unremarkable, as were echocardiogram, EKG, and tilt table tests. MRI showed microvascular changes but otherwise was unremarkable. Patient had a GI workup and was diagnosed with diverticulitis. The patient was treated symptomatically with no further syncopal episodes. Follow-up 5 months later reported no syncope.

Discussion

The diagnosis of syncope has typically been associated with cardiovascular etiologies and vasovagal components. However, syncope may also be associated with numerous other disciplines including infectious, renal, GI, endocrine, and psychiatric etiologies. Our prior study [7] aimed to evaluate the acute therapeutic areas involved with findings of syncope in the ED; during our data analysis, we identified a cohort of patients with a subdiagnosis presenting with gastrointestinal-induced bradycardia, with stereotypic symptoms of acute abdominal pain, bradycardia, and syncope, with associated GI diagnosis and negative cardiac and neurologic workup. We report four additional cases herein noting negative workups as per ESC guidelines for syncope, with symptomatic treatment of the GI disorders resulting in resolution of the syncope; outpatient follow-up in three of these cases revealed no additional episodes of syncope.

In our experience, GI-associated diagnoses with syncope have been noted to represent up to 6% of cases presenting to the ED [7]. Such associated involvement spanned all age groups, and overall was the fourth most common associated diagnosis. While GI bleeds were frequent, the most common associated clinical scenario noted has been severe abdominal pain and vomiting with or without nausea. This particular subgroup was observed to have episodic bradycardia and syncope during exacerbations of their abdominal pain (a reflex abdominal syncope).

In the current case series, each patient with reflex abdominal syncope recovered spontaneously; one patient eloped before further workup, but the other three underwent subsequent detailed workup, including cardiac (echocardiogram, EKG, tilt table evaluation) and neurologic (MRI, CT/CTA, EEG), all of which was unremarkable. With treatment of the underlying disease, no recurrence of syncope was observed in the remaining patients, including subsequent outpatient follow-up.

Telemetry data were available in three of the four cases (cases 1, 2, and 4), as each patient was placed on continuous cardiac monitoring upon admission. In each instance, the syncopal episodes were temporally associated with acute drops in heart rate to the 35–50 beats per minute range, with concurrent hemodynamic compromise documented in case 2 (systolic blood pressure 70 mm Hg). Heart rates normalized spontaneously within 1–2 min. No sustained arrhythmias, conduction abnormalities, or ST-segment changes were identified

on telemetry review. Formal orthostatic vital signs were not systematically obtained in all cases; however, vital signs at presentation were hemodynamically stable in all patients, and IV fluid resuscitation was administered in each case. Volume status assessment was limited by the retrospective nature of these observations, though clinical documentation indicated that patients were not overtly volume-depleted at the time of their syncopal events, with the possible exception of case 3, where the patient reported 2 days of diarrhea and vomiting prior to presentation.

A review of medications in each case was performed to evaluate for pharmacologic contributors to bradycardia or syncope. Case 1 had no significant medical history and was on no medications. Case 2 was on antihypertensive therapy, though the specific agents were not documented to include beta-blockers or calcium channel blockers; her donepezil for Alzheimer dementia may have cholinergic effects that could theoretically lower heart rate, though this had been a stable medication without prior hemodynamic effects. Case 3 had a history of hypertension and diabetes, though his undomiciled status and subsequent elopement limited detailed medication review. Case 4 was on antihypertensive therapy without documented beta-blocker or rate-limiting calcium channel blocker use, and his marijuana use was noted but unlikely to contribute to bradycardia. In no case was a clear pharmacologic contributor to the bradycardia or syncopal episodes identified.

We propose that reflex abdominal syncope, as described in this series, represents a subtype of reflex (vasovagal) syncope rather than a distinct pathophysiologic entity. The 2018 ESC syncope guidelines classify reflex syncope broadly, with subcategories including vasovagal (mediated by orthostatic or emotional triggers) and situational (mediated by specific stimuli such as cough, micturition, or deglutition) [1]. The pattern observed in our patients—acute visceral pain triggering a vagally mediated cardioinhibitory response with bradycardia and syncope—is most consistent with a situational reflex syncope in which abdominal pain serves as the afferent trigger. The distinction is clinically relevant because it identifies a treatable underlying cause (the GI pathology), and resolution of the GI disorder was associated with cessation of syncopal episodes in all cases with follow-up.

The risk of recurrence of reflex abdominal syncope appears to be closely linked to the persistence of the underlying GI pathology. In our series, treatment of the GI disorder (gastritis, gastroenteritis, diverticulitis) resulted in resolution of syncopal symptoms, with follow-up ranging from 3 to 8 months without recurrence. This finding suggests that the prognosis for syncope recurrence is favorable when the inciting GI condition is identified and adequately treated. However, patients with chronic or recurrent GI disorders may remain at risk for subsequent episodes. Volume status likely plays a contributory role, particularly in the acute setting where diarrhea, vomiting, and reduced oral intake may compound the vagal response. IV fluid administration was part of the acute management in all of our cases, and adequate hydration should be considered an important component of both acute treatment and preventive strategy in patients presenting with reflex abdominal syncope. Counseling patients on maintaining adequate oral hydration in the setting of GI illness may help reduce the risk of recurrence.

It is important to distinguish between acute and chronic GI pathology in the context of reflex abdominal syncope. In our series, the underlying GI diagnoses were predominantly acute conditions (gastroenteritis in case 1, acute gastritis in case 2, acute-on-chronic GI symptoms with diarrhea in case 3, and diverticulitis in case 4). Acute GI conditions, particularly those associated with diarrhea and vomiting, may predispose to relative hypovolemia and thus increase susceptibility to syncope through both a reduction in preload and enhancement of vagal tone. In contrast, the patients described by Wang et al [8] had chronic abdominal pain with recurrent syncopal episodes, suggesting a different clinical trajectory. The acuity and severity of the abdominal pain in our patients were notable: each syncopal event was temporally linked to an acute exacerbation of severe visceral pain, and in three of four cases, the recurrence of syncope in the ED was witnessed in direct association with a new pain episode. This reproducibility of the pain–bradycardia–syncope sequence under monitored conditions strengthens the case for a reflex mechanism.

There is a paucity of assessments of GI-associated syncope in the literature. Nguyen et al [9] reported a case of an 18-year-old man without any prior medical history who presented with an episode of syncope. After evaluation was completed, he was found to have GI bleeding and weight loss due to symptomatic anemia and physical exertion secondary to a gastric ulcer due to chronic *H. pylori* gastritis. Ischemic gastritis and perforation have also been identified as a potential source of syncope, though it is uncommon and likely underdiagnosed [10]. Deglutition syncope has also been noted as a rare, neurally mediated reflex syncope associated with swallowing, typically associated with pharyngoesophageal disorders with secondary abnormal vagal reflex causing atrioventricular cardiac block and cerebral hypoperfusion [11]. Finally, Wang et al [8] reported adult-onset syncopal syncope associated with abdominal pain in three patients, who all had chronic abdominal pain with recurrent syncopal episodes. In these patients, one patient was found to have evidence of a large right to left cardiac shunt, one revealed a partial right bundle branch block associated with multiple sources of premature beats, and one showed negative cardiac and neurologic workup.

Thus, the present cases were notable for the absence of cardiac or neurologic abnormalities on workup, yet demonstrated bradycardia associated with abdominal pain with subsequent syncope. These current findings and those previously reported [7], and potentially one from Wang et al [8], most likely reflect a reflex abdominal syncope associated with abdominal pain. Due to the frequency of transient loss of consciousness and GI pain as emergency room complaints, the two in concert should result in consideration of reflex abdominal syncope as an etiology with appropriate requisite monitoring and diagnostic evaluations.

Conclusion

Transient loss of consciousness remains a frequent presenting complaint in the acute care setting. Though most commonly categorized into cardiovascular, vasovagal, or orthostatic etiologies, numerous other systems may produce transient loss of

consciousness and should not be excluded from workup when a patient presents associated symptoms. GI-related syncope is frequent, and most often has been associated with severe abdominal pain and vomiting with or without nausea. Within this group includes patients with observed bradycardia associated with exacerbations of abdominal pain, a reflex abdominal syncope. We present four additional cases with outpatient follow-up. None had an identified cardiac or neurologic source of syncope; all were found to have gastritis, gastroenteritis, and diverticulitis. Our patients had no recurrence of syncope after appropriate management. These cases suggest that reflex abdominal syncope is not rare and should be considered in patients presenting to the ED with a history of GI pain and syncope with appropriate diagnostic workup and treatment.

Learning points

Syncope is a sign rather than a disease *per se*; GI etiologies are frequent; association of GI etiologies with bradycardia is a novel syndrome presenting in the acute care setting.

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Conflict of Interest

The authors declare that the work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Informed Consent

Not applicable.

Author Contributions

SG, BL: conceptualization; SG, BL: methodology, writing – first draft; SG, BL: writing –reviewing and editing; SG, BL: validation; BL: supervision/oversight.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

Generative Artificial Intelligence

During the preparation of this manuscript, Grammarly 1.2.235 for format and text support, and Semantic Scholar (semanticscholar.org) for context management. The authors have reviewed and edited the output and take full responsibility for the content of this publication.

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