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Prolonged sinus pause following ingestion of a button battery

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ABSTRACT

Para-esophageal inflammation secondary to button battery ingestion may rarely irritate the vagus nerve causing prolonged sinus pauses. We report such a case in a two-year-old who presented with prolonged sinus pauses noted on telemetry following button battery ingestion. A comprehensive evaluation deemed hyper-stimulation of the parasympathetic input to the sinus node as the possible etiology. This was determined to be most likely due to irritation and excessive stimulation of the vagus nerve secondary to para-esophageal inflammation. Because of the underlying mechanism of the sinus pauses, treatment with anti-cholinergic/anti-inflammatory agents was pursued with significant improvement in the clinical presentation. The case highlights the need to delineate the mechanism of sinus pauses in patients, as it may be reversible. Treatment directed at the underlying etiology is indicated to prevent inappropriate consideration/placement of a permanent pacemaker and avoid its associated co-morbidities, especially in the pediatric population.

1. Introduction

Foreign body ingestion is most common in children six months to three years of age [1]. Though serious morbidity is rare, occurring in less than 1% of all patients, approximately 1500 deaths per year are attributed to foreign body ingestion in the United States [1,2]. Button battery ingestion is one of the more serious types of foreign body ingestion as it may lead to rapid necrosis of the surrounding tissue and requires urgent endoscopic removal [1]. We report an extremely rare case of prolonged sinus pause following button battery ingestion presumably caused by vagus nerve irritation due to para-esophageal inflammation.

2. Case report

A two-year-old previously healthy female was admitted to our pediatric intensive care unit (PICU) after removal of an ingested button battery at an outside hospital. Prior to her presentation to our PICU, the parents noted that she had labored breathing and took her to an outside emergency room. There she had a chest radiograph that demonstrated a foreign body with a double density in the mid-lower esophagus (Fig. 1). She immediately went to the operating room and an esophagogastroduodenoscopy demonstrated a button battery in the mid esophagus with significant inflammatory changes and necrotic tissue around the site. The button battery was removed uneventfully. A computed tomographic (CT) scan of the chest demonstrated significant para-esophageal inflammation and edema. Given the degree of scarring and the risk of an aorto-esophageal fistula, the patient was transferred to our PICU because of monitoring in case of need for cardio-thoracic surgery. The child arrived at the unit intubated. Vital signs upon admission included a weight of 10 kg, temperature of 98.4 F, heart rate of 97 bpm, blood pressure 103/58 and an oxygen saturation of 100% on an Fio2 of 40%. Initial laboratory tests included a capillary blood gas (7.42/35.5/44/23/-1), a normal complete blood count, a normal complete metabolic panel and mildly elevated C-reactive protein (1.9 mg/dl). A magnetic resonance imaging (MRI) scan performed shortly after admission demonstrated extensive inflammatory changes involving the mid intrathoracic esophagus starting just below the carina and extending to approximately the level of the gastroesophageal junction and extending circumferentially around the descending thoracic aorta (Fig. 2). Twelve hours after admission, the telemetry monitor captured two prolonged sinus pauses of 16.5 s and 14 s (Fig. 3). Further review of telemetry revealed at least 3 other shorter sinus pauses of 3–5 s. All these episodes started with sinus slowing followed by the prolonged pause. Recovery started with junctional escape rhythm and gradually increased rate with resumption of normal sinus rhythm. The anesthetic medications received during button battery removal were reviewed to evaluate for medication reaction/effect. They included propofol (120 mg), fentanyl (15 μg), rocuronium (10 mg), dexmedetomidine (10 μg) and sevoflurane for induction and maintenance. Detailed past medical and family history failed to elicit any prior similar episodes or any obvious familial predisposition for sinus node

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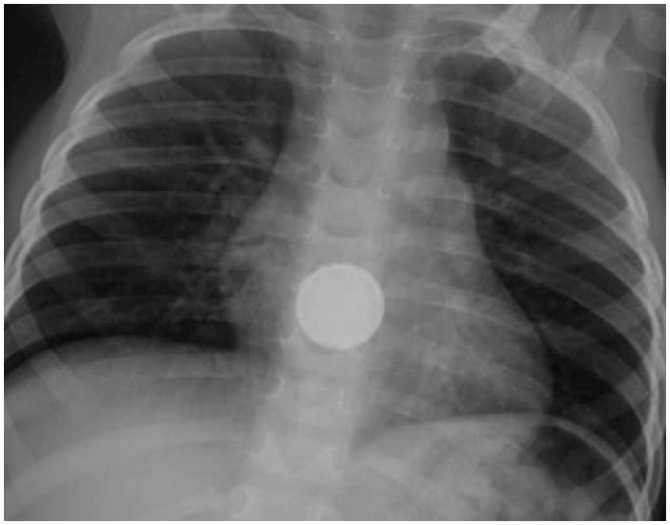

Fig. 1. Chest radiograph demonstrating a foreign body with a 'halo' sign in the mid-lower esophagus.

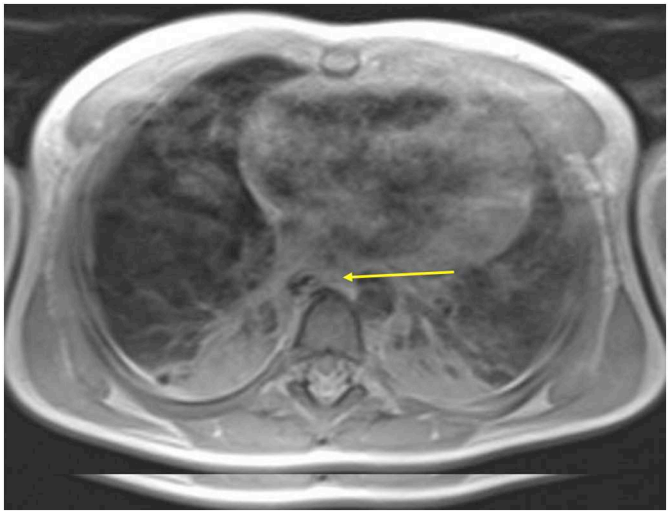

Fig. 2. Magnetic resonance imaging showing esophageal wall thickening and surrounding inflammatory changes involving the mid-lower esophagus directly anterior to the descending thoracic aorta (yellow arrow).

Table 1
Common etiologies of sinus pause.

Etiology	
Medications and toxins	β-Blockers
	Calcium channel blockers
	Digoxin
	Anti-arrhythmic
	Methyldopa
	Clonidine
Familial disease	Familial sick sinus syndrome
	HCN4 mutation
Infiltrative diseases	Amyloidosis
	Sarcoidosis
	Hemochromatosis
	Tumor
Inflammatory diseases	Pericarditis
	Myocarditis
	Rheumatic fever
Trauma	Cardiac surgery
Sinus node artery disease	
Sinus node fibrosis	
Miscellaneous	Hypothyroidism
	Hypothermia
	Hypoxia

dysfunction. An echocardiogram was performed which revealed a structurally and functionally normal heart. The cardiology consult team in discussion with cardiac electrophysiology determined that the anesthetic medications used at the time of button of battery removal were unlikely to be the etiology of the sinus pauses given the doses used and their duration of use. A detailed family history made a familial syndrome for sinus node dysfunction low on the differential and a normal echocardiogram eliminated structural heart disease as the etiology. The fact that the episodes started with sinus slowing and recovery started with junctional escape rhythm with a gradually increasing heart rate suggested hyper-stimulation of the parasympathetic input to the sinus node as the possible etiology. This was deemed most likely to be due to irritation and excessive stimulation of the vagus nerve secondary to para-esophageal inflammation. The patient was thus started on glycopyrrolate (10 μg/kg/dose IV Q6H) for anti-cholinergic effect and dexamethasone (0.5 mg/kg/dose IV Q8H) for anti-inflammatory effect. Because she continued to have some heart rate variability and occasional episodes of bradycardia, she was also started on IV isoproterenol at 0.02 μg/kg/min. After being on this therapy for 24 h, telemetry did not reveal any further episodes of sinus pauses. She was weaned off of isoproterenol in 24 h and dexamethasone and glycopyrrolate were discontinued after 24 and 72 h respectively. She was observed on telemetry for 48 h after discontinuation of treatment with no further events.

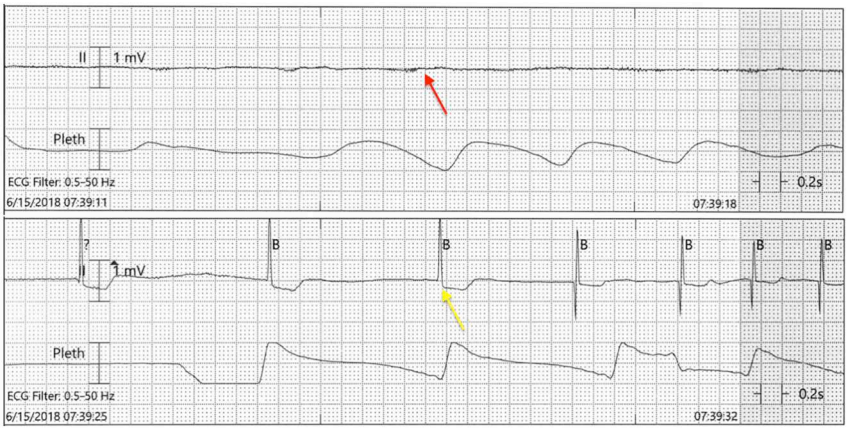

Fig. 3. Telemetry recording showing prolonged sinus pause (red arrow), followed by recovery with junctional escape rhythm (yellow arrow) before reverting to normal sinus rhythm.

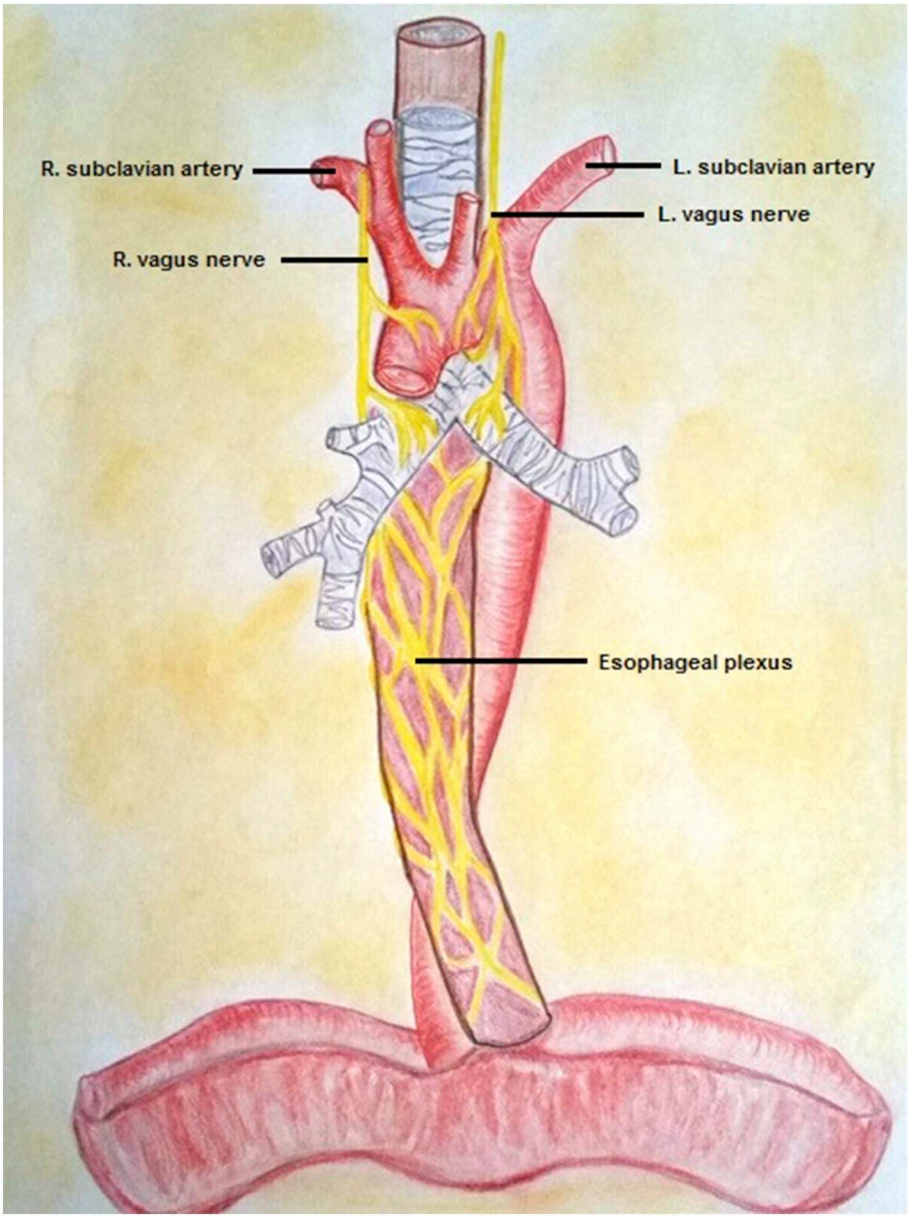

Fig. 4. A diagrammatic representation of the course of the right and left vagus nerves and the formation of the esophageal plexus.

3. Discussion

Sick sinus syndrome is characterized by sinus node dysfunction and may present clinically as sinus bradycardia, sinus pause, sino-atrial exit block or an inadequate chronotropic response [3]. A sinus pause is defined as the temporary absence of 'P' waves on the surface electrocardiogram. This pause may be as short as 2 s but may last longer and is believed to be secondary to an alteration in discharge by the sinus node. During the pause, escape beats may originate in an ectopic fashion from the atrium, the atrioventricular node, or the ventricles [4,5]. The differential diagnosis of sinus pause includes sinus node fibrosis as a result of a degenerative process (seen mostly in adults), medications and toxins, familial disease, infiltrative and inflammatory diseases, sinus node artery disease and trauma during cardiac surgery (Table 1).

To our knowledge, this is the first reported case of prolonged sinus pauses due to excessive irritation of the vagus nerve secondary to extensive para-esophageal inflammation following button battery ingestion. Foreign body ingestion is most common in children six months to three years of age [1]. Button battery ingestion is one of the more serious types of foreign body ingestion as it may lead to rapid necrosis of the surrounding tissue and thus warrants urgent endoscopic removal [1,2]. Button batteries lodged in the esophagus may lead to rapid and extensive mucosal damage, necrosis, and perforation [6,7]. A button battery has a "halo sign" or double density at its periphery when viewed en face on a chest radiograph [8].

The vagus nerve descends vertically in the carotid sheath after exiting the medulla oblongata. The right vagus nerve crosses anterior to the first part of the subclavian artery, travels posterior to the innominate vessels, reaches the thorax to the right of the trachea and then forms the esophageal plexus after traveling medially and joining the left vagus nerve [9]. The left vagus nerve crosses anterior to the left subclavian artery and enters the thorax between the left common carotid and subclavian arteries. It then continues its descent to the left of the aortic arch and joins the right vagus nerve to form the esophageal plexus [9] (Fig. 4). Thus, any significant inflammation of the para-esophageal region may stimulate and irritate the vagus nerve.

Heart rate is dependent on the activity of the sinus node which in turn is largely determined by the net result of its sympathetic and

parasympathetic stimulation [10]. The primary parasympathetic nerve supply to the sinus node is via the vagus nerve. Thus, with excessive stimulation of the vagus nerve, one may expect to see sinus bradycardia or, in extreme cases, prolonged sinus pauses. The placement of a permanent pacemaker is indicated for sinus node dysfunction with documented symptomatic bradycardia including frequent symptomatic sinus pauses [11]. Although the guidelines are not clear about the frequency/duration of sinus pauses that would prompt placement of a pacemaker, they state that it is not uncommon to detect resting heart rates as low as 40 bpm and sinus pauses as long as 2.8 s in trained athletes and these findings are not pathologic. The guidelines do, however, emphasize the importance of determining the underlying etiology to rule out reversible causes. Hence, prompt recognition and treatment directed at the underlying etiology is indicated to prevent inappropriate consideration or placement of a permanent pacemaker and avoid its associated co-morbidities, especially in the pediatric population.

Our patient had prolonged sinus pauses as long as 16.5 s with no escape beats during that time. The fact that there was slowing of the heart rate prior to the pause suggested excessive parasympathetic stimulation of the sinus node as the mechanism. As a result, we opted to treat with glycopyrrolate, which is an anticholinergic drug, to counter this effect [12]. Because we believed the excessive parasympathetic drive was from over-stimulation of the vagus nerve secondary to esophageal inflammation and edema, corticosteroids were started. Isoproterenol is a β_1 and β_2 adrenoceptor agonist and has almost no effect on α adrenergic receptors [13]. It has positive inotropic and chronotropic effects on the heart and was used as a bridge to support the heart rate while allowing the glycopyrrolate and corticosteroids to take effect due to continued sinus bradycardia after the first doses of those medications. It was weaned off 24 h after initiation.

This case emphasizes the need of a comprehensive evaluation, which is not limited to the gastrointestinal and respiratory system in cases of button battery ingestion. It also highlights the importance of attempting to identify the mechanism of sinus bradycardia and sinus pause in every patient. Although the sinus pauses would have likely improved over time, the prolonged episodes may have been life threatening, demanded temporary pacing and even prompted consideration of a permanent pacemaker in the absence of an identifiable underlying treatable etiology.

4. Conclusion

We describe a rare case of excessive vagus nerve stimulation secondary to para-esophageal inflammation leading to prolonged sinus pauses which was rapidly reversed with anti-inflammatory and anticholinergic pharmacologic therapy. While placement of a permanent pacemaker is indicated for prolonged symptomatic sinus pauses, it is essential to delineate the mechanism of the sinus pause as it may be reversible. Treatment directed at the underlying etiology is indicated in such cases to prevent consideration/placement of a permanent pacemaker and avoid its associated co-morbidities, especially in the pediatric population.

Declaration of Competing Interest

None of the authors have any conflict of interest to disclose with respect to publication of this manuscript.

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