

Case Report

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'Heart Rate Deficit' from Dysautonomia in a Bariatric Surgery Patient - An Unusual Cause of Recurrent Syncope

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Introduction

Since development of laparoscopic sleeve gastrectomy in 1999, bariatric surgery (BS) for morbid obesity has become increasingly common due its minimally invasive nature in conjunction with demonstrated mortality benefit.¹ The greatest weight loss occurs 6-24 months postoperatively.^{2,3} Patients undergoing BS are prone to develop symptomatic autonomic dysfunction with associated orthostatic intolerance potentially causing syncope.^{4,5,6} We report a case of bradycardia-mediated syncope without associated hypotension in a patient who had prior BS.

Case

A 61-year-old female with hyperlipidemia, fibromyalgia, morbid obesity status post gastric sleeve surgery 2 years prior, was seen for recurrent episodes of syncope over the past 2 months. Patient had significant weight loss (~170 lbs.) post-surgery over the 2-year period. She began noticing increasing fatigue and exercise intolerance in the four months before syncope initiation. Episodes occurred while sitting and while standing but most occurred without prodrome.

Baseline ECG showed sinus bradycardia at 41 bpm (Figure 1) with no other abnormalities. Thyroid function was normal. Other laboratory values were within normal limits. The echocardiogram was normal. During hospitalization, multiple syncopal episodes were witnessed while she tried to sit up or lift her head 30-60 degrees from a recumbent position. This was reproducible. Her baseline heart

Key Words

Syncope, Dysautonomia, Bariatric Surgery.

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rate (HR) with telemetry monitoring was 36-42 bpm and her blood pressures were in the normalrange (e.g., 126/78 mm Hg).

During witnessed syncopal episodes, the HR remained unchanged (35-40 bpm) with no asystolic pauses. Her eyes were closed. The systolic blood pressure also remained unchanged (120-140 mm Hg). Symptoms were so severe and was only relieved by either lying flat on the bed without a pillow or placing bed in a Trendelenburg position.

Psychogenic psuedosyncope was suspected given the unusual presentation. There was no history of psychological problems noted otherwise. However, video electroencephalogram showed loss of brain electrical activity during the syncopal episode (Figure 2). No seizure activity was noted. No other cause for syncope was noted despite extensive investigation

Review of prior ECGs during the peri-procedural period following bariatric surgery in 2017 showed a baseline HR ranging between 75-90 bpm (Figure 3). Progressive decline in baseline HR since BS as well as baseline bradycardia raised concern for weight loss related dysautonomia and was suspected as a potential cause for syncope.

She underwent a dual chamber permanent pacemaker implantationprogrammed MVP-R (AAIR>>DDDR) at 60-130 bpm along with rate drop response. Dramatic improvement was noted in her symptoms. She was discharged the following day as she was able to stand up and walk with no dizziness or orthostatic intolerance. Over a 7-month follow-up, she has not had any further recurrence of syncope and remains atrially paced.

Discussion

Bariatric surgery has been increasingly used for patients with morbid obesity, and has been associated with improved outcomes.¹ Studies have shown changes in autonomic indices following BS,

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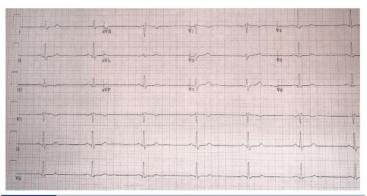


Figure 1: Baseline ECG at presentation showing sinus bradycardia at 41 bpm with normal QTc

specifically an increase in vagal tone and relative vagal efferent flow after gastric bypass surgery. Studies on heart rate variability parameters following BS show that autonomic indices overall tend to change toward a parasympathetic predominance. Multiple studies have reported on orthostatic intolerance and associated symptoms following BS but little information is available regarding other mechanisms causing syncope. Moreover, exact mechanisms underlying autonomic dysfunction in these patients remain unclear. Whether the increased vagal tone is secondary to weight loss or another mechanism remains to be elucidated. Nerve redistribution during gastric bypass surgery as well as the impact of BS on various gut hormones such as insulin, leptin, ghrelin, gastrin, GLP-1, CCK has been proposed as potential mechanisms increasing vagal tone.

Multiple mechanisms thus may be in play which can alter the intricate autonomic balance following BS and create dysfunction.

We report, for the first time to our knowledge, a post-bariatric surgery patient who had progressive weight loss resulting in progressive decline in baseline HR leading to recurrent, debilitating syncope without any long pauses/asystole or vasodepression. It appears that there was a relative 'HR deficit' where baseline HR fell below a critical threshold for adequate cerebral perfusion with any postural changes despite a blood pressure that remained within the normal range, both during recumbent position as well as with postural changes. Pacemaker implantation completely resolved symptoms by increasing baseline HR and restoring adequate cerebral perfusion. Further studies are needed to better understand

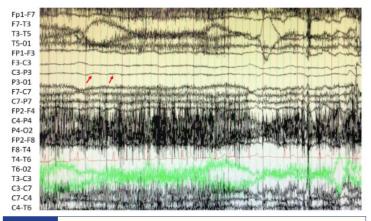


Figure 2: Video electroencephalogram showing loss of electrocerebral activity during the syncopal episode (red arrows).



Figure 3: ECG obtained during peri-procedural period following bariatric surgery 2 years ago showing sinus rhythm at 72 bpm

mechanistic underpinnings behind progressive bradycardia without vasodepression following BS.

Conclusion

We describe a unique case in which BS was associated with marked resting sinus bradycardia causing debilitating symptoms. This 'HR deficit' and resting bradycardia, likely due to dysautonomia, resulted in recurrent syncope without asystole or vasodepression, and completely resolved with permanent pacemaker implantation.

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