

Tonsillar Inflammation, a Rare Cause of Bradycardia and Hypotension

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Introduction

Tonsillitis is a frequently encountered pathology in the outpatient setting, usually caused by viruses [1]. When bacterial, the most common causatory microbe is streptococcus group A [1]. Tonsillar and peritonsillar abscess (PTA) on the other hand are never viral, and are usually caused by streptococcus pyogenes, Streptococcus melleri, fusobacterium necrophorum and staphylococci [1,2]. The overall incidence of PTA is suggested to be 37/100,000 patients, with the highest incidence between ages 14-21 at 124/100,000 [3].

Reflex bradycardia is a homeostatic response mediated by baroreceptor activation in the carotid sinus and aortic arch. Vagal nerve stimulation triggers inhibition of sympathetic stimulation, which causes a decrease in heart rate and blood pressure [1]. Carotid sinus syndrome (CSS) is an overstimulation of this carotid response, resulting in bradycardia, hypotension, dizziness, syncope, and rarely, cerebral ischemia [1]. Stimuli can be external (carotid sinus massage), or internal (local tumors, inflammation and swelling). Interestingly, the carotid sinus can have a variable location in the carotid artery [2]. Here, we report a case of PTA leading to carotid sinus syndrome.

Case

A 23 year old lady with a past medical history (PMH) of PCOS (polycystic ovarian syndrome), Hashimoto's disease, and MTHFR gene mutation with clotting disorder, presented to the hospital with a two day history of sore throat and cough, associated with dysphagia. Physical exam was significant for anterior cervical lymphadenopathy. Her vitals were: BP 135/65, HR 92, Temp 35.9 and RR 18. At that time. She was diagnosed with pharyngitis, and discharged on clindamycin and prednisone.

Two days later she returned, with complaints of worsening dysphagia and odynophagia, associated with right neck tenderness, ear pain and subjective fevers and chills. She had been taking the clindamycin throughout this time. Temperature was 36.4 (97.6 F), HR 64, BP 107/52. Physical exam showed persistent anterior cervical lymphadenopathy, markedly swollen tonsils, and right sided neck tenderness. She was given decadron in the er. CT neck showed generalized tonsillar prominence with tonsillar and paratonsillar phlegmon/ abscess, and she was started on vancomycin and unasyn.

The next day, she developed persistent sinus bradycardia, reaching HR as low as 34 on the 4th day of treatment. She was transferred to the cardiac care unit and workup was initiated. Troponins were negative. EKG showed sinus bradycardia without heart block, ECHO was normal. CTA showed no pulmonary embolism. Telemetry revealed persistent sinus bradycardia, without evidence of pauses or blocks.

The bradycardia finally started improving around day 5 of antibiotics, and was subsequently attributed to direct pressure from the abscess/ phlegmon, as well as suspected abnormal location of carotid sinus. The abscess was not large enough to be drained, and the symptoms gradually resolved as inflammation resolved. By day 6 of treatment, her HR was back to normal range.

Image 1: Neck CT with 9.3 mm phlegmon/abscess



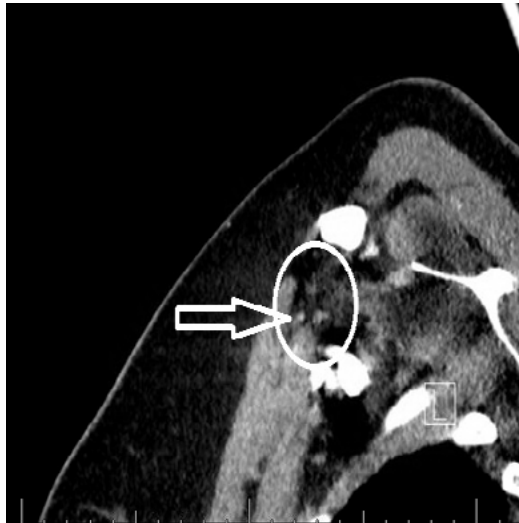


Image 2: Sagittabsection showing same are of inflammation adjacent to the carotid sinus

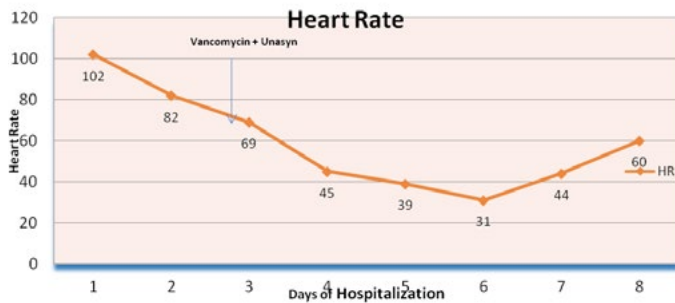


Figure 1: Heart rate over the duration of therapy

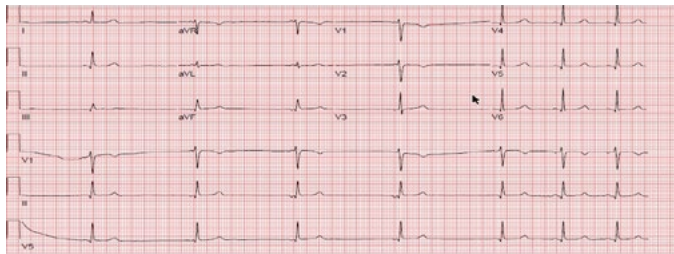


Image 3: EKG showing Sinus Bradycardia with HR of 43

Discussion

Parasympathetic stimulation of the heart is controlled by the vagus nerve. The right vagus nerve innervates the SA node, while the left vagus nerve blocks heart conduction through its effect on the AV node. The carotid sinus, containing baroreceptors, is usually present at the carotid bifurcation; However, cases documenting variability in the origin and location of the carotid sinus in the carotid wall have also been reported. These baroreceptors are innervated by the sinus nerve of Hering (branch of the glossopharyngeal (IX) nerve). Stimulation of the carotid sinus leads to stretching of the baroreceptors, resulting in impulse conduction through the afferent nerve of Hering, to the Nucleus trachus Solitarius (brainstem) and then via the vagus. Subsequently, bradycardia ensues [4-10]. Overstimulation due to external and internal mechanical compression leads to the carotid sinus syndrome [2]. Thus, carotid sinus syndrome can present with bradycardia, hypotension and syncope

[1]. Any neck mass can potentially lead to carotid sinus stimulation, given it is a tight space and mechanical compression can easily occur [2]. This can include neck abscesses, tonsillitis, phlegmon, and primary and metastatic cancers [2].

Typically, if an abscess is thought to cause bradycardia, the treatment would be abscess drainage, and implantation of a temporary or permanent pacemaker for heart rate control. Our patient was believed to have an aberrantly located carotid sinus, which was likely impinged upon by the tonsillar abscess. Given the small abscess size, treatment was mainly antibiotics and supportive care. She didn't require a pacemaker as she started improving quickly, without symptomatic bradycardia.

Conclusion

Tonsillitis is a very common disease, which can lead to peritonsillar abscess formation, potentially resulting in carotid sinus stimulation due to mechanical compression. Treatment of bradycardia in any patient with a neck mass would entail prompt treatment of the mass itself, along with evaluation for temporary pacemaker.

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