Reflex Syncope Manifesting as Orthostatic Complete Heart Block

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Abstract
A 52 year old patient presented with orthostatic dizziness and syncope caused by postural heart block. When the patient was supine, atrioventricular conduction was normal but when she assumed the upright posture she developed advanced atrioventricular block rapidly progressing to complete heart block. We are presenting a case of syncope caused by orthostatic heart block.

Introduction
Syncope is defined as a transient loss of consciousness, the underlying mechanism being a transient period of global cerebral hypoperfusion. A sudden cessation of cerebral blood flow for 6–8 s has been shown to be sufficient to cause complete loss of consciousness.

It is a common medical problem and accounts for 6% of hospital admissions. The elderly are particularly at higher risks for syncope having a 6 per cent annual incidence of syncope. 10-year cumulative incidence of syncope is 11% for both men and women at age 70–79, and 17% and 19% respectively for men and women at age ≥ 80.

Syncope can be caused by a variety of conditions with cardiac causes having the highest mortality.

Vascular causes of syncope are most common, followed by cardiac causes. Among cardiac causes of syncope, arrhythmias are most common.

Orthostatic hypotension, which is defined as a 20-mmHg drop in systolic blood pressure or a 10-mmHg drop in diastolic blood pressure within 3 minutes of standing, results from a defect in any portion of the blood pressure control system. It can be due to drugs/volume depletion/autonomic failure. But posturally related heart block is only very rarely reported as the cause of orthostatic syncope.

We present a case of postural heart block in a 52 year old woman.

Case Report
A 52 yr old woman, a known diabetic of 3 yrs duration presented to the medicine OP with H/O recurrent fainting episodes 1 month back. ECG taken in Medicine OP showed complete heart block and patient was referred to the Cardiology Department.

Clinically patient was conscious, oriented, afebrile, not anaemic or jaundiced. She had no pedal oedema. JVP showed intermittent cannon a waves. She was stable, had otherwise no significant findings at time of examination.

Her pulse rate was 42/mt and regular, her BP was 130/70 mmHg in all four limbs. No other abnormal cardiac findings were present.

Patient had a hyperpigmented rash over cheeks and forehead. Other systemic examinations were normal.
ECG showed complete heart block with an atrial rate of 84/mt and a regular narrow QRS rhythm rate of 40/mt.

Since patient was now asymptomatic, she was kept in observation. We noticed that CHB reverted to sinus rhythm intermittently (Figure 1 & 2). On continuous observation, we noticed that patient had sinus rhythm during recumbent posture (Figure 1) and heart block developed in upright posture.

Patient did not develop any symptoms during upright posture and BP remained stable during upright position without hypotension.

Continuous video recording was done when sinus rhythm during supine position quickly transformed to high grade AV block and then to complete heart block (Figure 2) on assuming the sitting position and again the complete heart block resolved to sinus rhythm on assuming the supine position. The changes occurred within 30 seconds of change in posture. This phenomenon we were able to reproduce a number of times.

The patient did not develop symptoms during the development of complete heart block and there was no fall in blood pressure.

Investigations

- FBS 86 mg/dl
- Hb 11 g/dl
- TC 5600
- DC P 65, L 33%
- Renal function tests were normal
- TSH 2.5 mIU/ml
- T4 1.15 ng/dl
- CRP + (6 mg/L)
- ANA negative

Echo
- Showed normal chamber dimensions. LV showed normal systolic contraction and function with grade 1 diastolic dysfunction. The valves were normal.

Patient was treated with aspirin, clopidogrel, atorvastatin and alupent.

After a few days, the CHB resolved. Sinus rhythm was restored and she remains in continuous sinus rhythm for 10 days and when she came up for review after a month, the orthostatic heart block was present again.

As the patient is asymptomatic, she is being followed up.

Discussion

Syncope is due to a transient fall in cerebral perfusion. Syncope may be classified as
1. Reflex syncope (neurally mediated e.g.: vasovagal, situational, carotid sinus hypersensitivity)
2. Syncope due to orthostatic hypotension
   a. Primary autonomic failure (Parkinson’s disease)
   b. Secondary autonomic failure (diabetes)
   c. Drug induced hypotension
   d. Volume depletion
3. Cardiac causes
   a. Ischaemia related
   b. Arrhythmia related
   c. Obstructive lesions

Neurocardiogenic syncope or Reflex syncope is caused by an abnormal or exaggerated autonomic response to various stimuli of which most common are standing and emotion.

The mechanism involves reflex mediated changes in heart rate or vascular tone and by activation of cardiac “C” fibres. Stimulation of “C” fibres results in vasodilation and increased vagal tone with consequent fall in cardiac filling and bradycardia with ensuing syncope.

The differential diagnoses of reflex syncope are carotid sinus hypersensitivity and orthostatic hypotension.

Before a diagnosis of reflex syncope is established, structural heart disease and arrhythmias must be ruled out.

In contrast to reflex mediated syncope, orthostatic hypotension is a failure of autonomic reflex response, manifesting as a drop in blood pressure on assuming upright posture due to failure of autonomic nervous system to compensate for venous pooling in lower limbs with reduced venous return and cardiac output with consequent cerebral hypoperfusion.

In carotid sinus hypersensitivity, syncope or presyncope results from extreme response to carotid sinus stimulation.

In neurally mediated syncope (also called vasovagal or reflex mediated syncope) different types of manifestation is explained by different types of activation or depression of autonomic nervous system. A more intense activation of parasympathetic nervous system provokes bradycardia and loss of sympathetic tone causes hypotension.

Vasovagal episodes may occur without any identifiable trigger even in patients who are sitting.
The pathophysiology of Reflex Mediated Syncope

The pathophysiology is not completely understood. Reflex activation triggers a rapid decrease in heart rate and a reduction in vascular tone. The concept of depressor reflexes originating in heart was described by von Bezold in 1867 and later revised by Jarisch in 1937.

The change to an upright position causes venous pooling, up to 500 ml of blood flows down to the legs. By activation of autonomic nervous system, contraction and heart rate increases to maintain sufficient circulating volume.

In the first moments of a vasovagal syncope, an empty heart is seen in echo because of acute loss of preload (empty heart syndrome)

Mechanoreceptors in the wall of LV, aorta and pulmonary trunk are activated. These vagal inhibitory sensory receptors mainly found in LV are also seen in bladder, lungs and esophagus. Stimulation of these vagal C fibres occur in response to stretch, chemical substances and drugs. These C fibres connect the heart with brainstem, wherein vagal neurons are stimulated and activity of sympathetic system is depressed. Activation of this reflex mechanism provokes bradycardia, vasodilatation and hypotension.

95% of vasovagal syncope show a dominant vasodepressor response. 5% of patients show a predominant cardio inhibitory response. Cardiac effects of vagal stimulation include depression of rhythmicity of both sinus and AV node, prolongation of SA and AV conduction.

In the present case, the predominant effect of orthostatic stress is cardio inhibitory. The vagal inhibitory effect is seen involving the AV junction manifesting as transient complete heart block. The overwhelming reflex vagal effect is seen only on the AV node and not on the SA node. The reasons for this phenomenon could not be explained.

We have searched the literature and we were able to find only a few publications citing postural heart block without causing symptoms.

Conclusion

This case is presented because of the interesting phenomenon of orthostatic stress like change from recumbency to sitting up posture producing transient complete heart block. This most likely could be an extreme manifestation of exaggerated autonomic response in the form of vagal effect on the atrioventricular junction resulting in complete heart block.

References