# Incidental carotid sinus hypersensitivity in a patient presenting with syncope: A case report

# Abstract

# Carotid sinus hypersensitivity is one of the unexplained causes of syncope in old age. A 56-year-old male, known case of coronary artery disease (CAD) and diabetes mellitus (DM), presenting with syncope, was diagnosed as carotid sinus hypersensitivity incidentally. Hence, the rare disorder like carotid sinus hypersensitivity should also be considered.

# Keywords

Carotid sinus hypersensitivity (CSH); Carotid sinus syndrome (CSS); Carotid sinus massage (CSM); Orthostatic hypotension (OH); Vasovagal syncope (VVS); Electrocardiogram (ECG)

# Introduction

The etiology behind syncope is diverse. The carotid sinus hypersensitivity (CSH) is one of the causes for unexplained syncope in elderly patients. CSH along with the reproduction of syncopal event by carotid sinus massage has been referred to as carotid sinus syndrome (CSS). CSH is considered as a treatable cause of undiagnosed syncope (1). Since Weiss and Baker described CSH in 1933, several studies have been conducted regarding its clinical relevance and management options (2). The three varieties of CSH have been described in literature as cardioinhibitory, vasodepressor or mixed type (3).

The prolonged stimulation of carotid sinus has been seen to produce hypotension or syncope. The prevalence of CSH according to some studies has been shown in the range of 0-62% (4). The prevalence of CSH is found to increase with age. (5). It accounts for about one-third of symptoms in older patients presenting with syncope (4).

Here, we report a case of a 56-year-old male presenting with loss of consciousness, having two underlying co-morbidities – CAD and diabetes mellitus. This created a diagnostic dilemma among the treating physicians regarding the cause of the syncopal event.

# Case presentation

A 56-year-old Hindu male, known case of coronary artery disease (CAD) and type 2 diabetes mellitus (DM), presented to our emergency department with a complaint of loss of consciousness (LOC). It was sudden in onset, lasting for a few minutes, and was preceded by sweating. There was a history of two episodes of LOC. It was not associated with any position or activity. There was no history of chest pain, palpitation, headache, weakness, abnormal body movements, fever, trauma, frothing at mouth, up rolling of eyes, tongue bite and tinnitus. He was non-smoker and non-alcoholic.

On clinical examination, he was hemodynamically stable, with normal blood pressure on supine (126/90 mmHg), and standing position (110/88 mmHg). There was no significant postural drop in blood pressure, ultimately ruling out postural hypotension. The general and systemic examinations were unremarkable. He was admitted in medical ward for further evaluation.

The baseline laboratory investigations are shown in table 1.

Table 1: Baseline laboratory investigations

|  |  |  |  |
| --- | --- | --- | --- |
| **Laboratory tests** | **Result** | **Unit** | **Reference range** |
| Total Leukocytes Count | 4.6 | 10˄3/µL | 4-11 |
| Neutrophil | 70 | % | 40-80 |
| Lymphocyte | 20 | % | 20-40 |
| Hemoglobin | 13.7 | g/dl | 13-17 |
| Platelet Count | 137 | 10˄3/µL | 150-450 |
| Urea | 19 | mg/dl | 17-43 |
| Creatinine | 1.0 | mg/dl | 0.7-1.3 |
| Sodium | 142 | mEq/L | 135-145 |
| Potassium | 4.0 | mEq/L | 3.5-5.5 |
| Bilirubin Total | 0.8 | mg/dl | 0.1-1.2 |
| Bilirubin Direct | 0.3 | mg/dl | 0.0-0.2 |
| Alkaline Phosphatase (ALP) | 48 | U/L | 53-128 |
| Alanine Transferase (ALT) | 34 | U/L | 0-35 |
| Aspartate Transferase (AST) | 32.7 | U/L | 0-35 |
| Random Blood Glucose | 99 | mg/dl | 70-140 |
| Prothrombin time (PT) | 17.3 | seconds | 11-13.5 |
| CPK NAC | 144 | U/L | 20-200 |
| CPK MB | 28 | U/L | < 35 |
| Troponin I | Negative |  |  |
| FT3 | 3.14 | pg/ml | 2.3-4.2 |
| FT4 | 0.99 | ng/dl | 0.89-1.76 |
| Thyroid Secreting Hormone (TSH) | 5.10 | µIU/ml | 0.35-5.50 |
| Total Cholesterol | 201.4 | mg/dl | 0-200 |
| High Density Lipoprotein (HDL) | 32.3 | mg/dl | 40-60 |
| Low Density Lipoprotein (LDL) | 139.0 | mg/dl | 0-100 |
| Tri-glyceride (TG) | 129.3 | mg/dl | 0-180 |
| Serum magnesium | 2.98 | mg/dl | 1.8-2.6 |
| Serum Phosphorus | 2.81 | mg/dl | 2.4-4.4 |
| Serum Calcium | 9.21 | mg/dl | 8.6-11.8 |

On etiological workup for syncope, magnetic resonance imaging (MRI) of head showed a small locus of blood degradation product in the right frontal lobe. The electroencephalogram (EEG) showed normal background activity and was reactive to eye opening and closing. The carotid doppler showed normal scan. And, 2D echocardiography showed a reduced ejection fraction of 40% with no any outflow tract obstruction.

While the etiology for the syncope was in dilemma, an incidental carotid sinus massage resulted in sinus pause for more than three seconds in continuous electrocardiogram (ECG) monitoring. Carotid sinus massage (CSM) was performed sequentially over both left and right sides. It was performed both on supine and upright position for five seconds. The demonstration for sinus pause during CSM in ECG are shown in Figure 1 and Video 1. And reversion to the normal rhythm after CSM is shown in Video 2. The differential diagnoses such as vasovagal syncope and orthostatic hypotension were ruled out as the syncope was not associated with specific position or emotional and stressful events. He was then diagnosed as carotid sinus hypersensitivity (CSS). He was kept under medications as shown in table 2.

Table 2: Medications

|  |  |  |  |
| --- | --- | --- | --- |
| **Route** | **Drugs** | **Dose** | **Dosage** |
| Tablet | Aspirin | 75 mg | Once a day (OD) |
| Tablet | Clopidogrel | 75 mg | Once a day (OD) |
| Tablet | Atorvastatin | 20 mg | Hora Somnae (HS) |
| Tablet | Metoprolol | 37.5 mg | Once a day (OD) |
| Tablet | Isosorbide Mononitrate | 10 mg | Twice a day (BD) |

The definite treatment performed for CSH was dual chamber pacemaker placement. It was done in a heart center as the facility of cardiac catheterization was not available in our center. The pacemaker was placed through transvenous access via subclavian vein under local anesthesia. During the course on hospital, she was hemodynamically stable. He accepted the treatment and was recovering well. He was discharged on above mentioned medications after a week of stay in the hospital.

# Discussion

The diagnostic approach for syncope is complex and multidimensional. CSH has been found as a cause of unexplained syncope in elderly patients for a long time now. CSH is defined as sinus pause for more than 3 seconds and/or fall in systolic blood pressure (SBP) of 50 mmHg or greater in response to CSM (6). An asystole lasting for more than 3 seconds is termed as cardioinhibitory type of CSH. And, fall in systolic blood pressure (SBP) of 50 mmHg or greater is termed as vasodepressor type of CSH. If both of these are present, it is of mixed type. The term CSS is used when CSH is associated with spontaneous syncope. The diagnostic criteria for CSS is reproduction of spontaneous syncope during 10 seconds of CSM on both right and left carotid sinus sequentially (6).

The cardioinhibitory type is a common variant of CSH as compared to vasodepressor and mixed type. The vasodepressor type is a least common type of CSH (7). It is important to distinguish between cardioinhibitory and vasodepressor type of CSH. During CSM, atropine is given in order to distinguish between the above mentioned two types. If the blood pressures still declines despite not having bradycardia, it is suggestive of vasodepressor type (1,2). In our case, we could not delineate between cardioinhibitory and vasodepressor type due to technical infeasibility. We diagnosed it as CSH, cardioinhibitory type, as it was the commonest among the three.

The symptoms that patient can present with CSH include syncope or dizziness based on the perfusion to brain (7). Carotid sinus massage is a simple and reliable test to diagnose CSH. But, possibility of errors has been described in performing CSM. The precise anatomical location and position of the patient are some of the factors for errors in performing CSH (2). We performed CSM on both side in both positions sequentially. As there was no reproducible syncope during the massage, it did not fit into carotid sinus syndrome category.

According to a study, the symptoms are more profound in upright position as compared to supine position(8). In elderly population, CSH has been a modifiable risk factor for non-accidental falls (9). CSH has been shown to be associated with other causes of syncope like orthostatic hypotension (OH) and vasovagal syncope (VVS). A study by Maw Pin Tan et al, showed CSH, OH and VVS to be common causes for syncope in elderly and likely to co-exist in an affected individuals (10). In our case, there was no coexistence of these entities.

Both the pharmacological and interventional management strategies are being practiced for CSH. Before the pacing therapy, treatment options included anticholinergic drugs like atropine, radiation therapy and carotid sinus denervation (3,11). The preferred method of treatment that has been widely accepted is implantation of cardiac pacemaker (3,8,11,12). As dual-chamber pacemaker activates both atria and ventricles, it is a treatment of choice in cardioinhibitory type of CSH (8). The vasodepressor or mixed type can be treated with mineralocorticoids or radiation therapy and denervation of carotid sinus based on severity. Our case was treated with dual-chamber pacemaker placement.

There were certain limitations of our study. We could not distinguish between the two variants of CSH because of technical issues. Similarly, we could not follow up on the patient after discharge from the hospital.

# Conclusions

CSH is one of the unexplained causes of syncope in old age. It may create a diagnostic challenge, especially in patients with underlying co-morbidities like CAD and diabetes mellitus. So, the clinicians should be aware of this rare cause while attending a case of syncope with these characteristics, and management should be planned accordingly.

# Author Agreement Statement

We the undersigned declare that this manuscript is original, has not been published before and is not currently being considered for publication elsewhere.

We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

We understand that the Corresponding Author is the sole contact for the Editorial process. He/she is responsible for communicating with the other authors about progress, submissions of revisions and final approval of proofs.

# Ethical approval

As is a case report, therefore, it did not require ethical approval from ethics committee.

# Consent

Written [informed consent](https://www.sciencedirect.com/topics/medicine-and-dentistry/informed-consent) was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal on request.

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# Conflict of Interest

The authors report no conflicts of interest.

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Figure 1: ECG

Legend: Arrow (white) showing sinus pause during CSM

Video 1: Continuous ECG monitoring

Legend: Arrow (blue) showing sinus pause during CSM

Video 2: Continuous ECG monitoring

Legend: Arrow (blue) showing reversion to normal rhythm after CSM