

Recurrent Syncope Due to Carotid Sinus Hypersensitivity and Sick Sinus Syndrome

Feng-Yu Kuo¹, Hsiang-Chiang Hsiao¹, Chuen-Wang Chiou^{1,2}, Chun-Peng Liu^{1,2*}

¹Cardiovascular Medical Center, Kaohsiung Veterans General Hospital, Kaohsiung, and

²National Yang-Ming University School of Medicine, Taipei, Taiwan, R.O.C.

Syncope is a sudden and brief loss of consciousness with postural tone. Its recovery is usually spontaneous. There are various causes of syncope including cardiac, vascular, neurologic, metabolic and miscellaneous origins. The tracing is usually time-consuming and costly. The diagnosis of carotid sinus syncope may sometimes be difficult since the symptoms are nonspecific, especially in older persons. Here, we report the case of a 72-year-old woman who sought medical attention at our hospital due to repeated syncope episodes over the previous 5 years. Neurologic examinations showed negative results (including brain computed tomography). Twenty-four-hour ambulatory electrocardiogram monitoring showed atrial and ventricular premature contractions only. Electrophysiologic study disclosed prolonged corrected sinus node recovery time (1,737 ms) with poor atrioventricular conduction. Drop of blood pressure together with sinus bradycardia developed after left side carotid sinus massage. Both carotid sinus hypersensitivity with sick sinus syndrome contributed to this patient's syncope, and after pacemaker placement together with selective serotonin reuptake inhibitor treatment, she was free from syncope thereafter. [*J Chin Med Assoc* 2008;71(10):532–535]

Key Words: hypersensitive carotid sinus syndrome, sick sinus syndrome, syncope

Introduction

Syncope is a common clinical issue with complex and heterogeneous etiologies.¹ In 1933, Weiss and Baker established carotid sinus hypersensitivity as a causative factor in syncope, and then called further attention to the relationship between carotid sinus pathophysiology and syncope.² We know that excessive carotid sinus activity can cause recurrent syncope through sudden onset bradycardia (cardioinhibitory type) or hypotension without change in heart rate (vasodepressor type), or both (mixed type). Sick sinus syndrome refers to a combination of symptoms such as dizziness or confusion caused by sinus node dysfunction, which is a frequent cause of syncope at the same time. Here, we describe the first ever reported case of syncope, which was contributory to both hypersensitive carotid sinus syndrome, mixed type, and sick sinus syndrome.

Case Report

A 72-year-old woman was brought to our medical emergency department due to repeated syncope attacks in the past month. She had a 20-year history of hypertension and had undergone regular medical control. She denied having other medical illnesses in the past and lived well in her daily life. She was not allergic to any medication or food, and had never smoked in the past.

Five years prior to this admission, she started to have symptoms of occasional dizziness and unsteady gait. She paid little attention to it due to limited symptoms. One year later, she fainted when riding a motor scooter. She called our neurologist for help. Transcranial Doppler and Dopscan showed patency of bilateral carotid arteries without significant stenosis, and computed tomography of the brain was negative. In December



*Correspondence to: Dr Chun-Peng Liu, Cardiovascular Medical Center, Kaohsiung Veterans General Hospital, 386, Dar-Chung 1st Road, Kaohsiung 813, Taiwan, R.O.C.

E-mail: cpliu@isca.vghks.gov.tw • Received: August 6, 2007 • Accepted: May 16, 2008

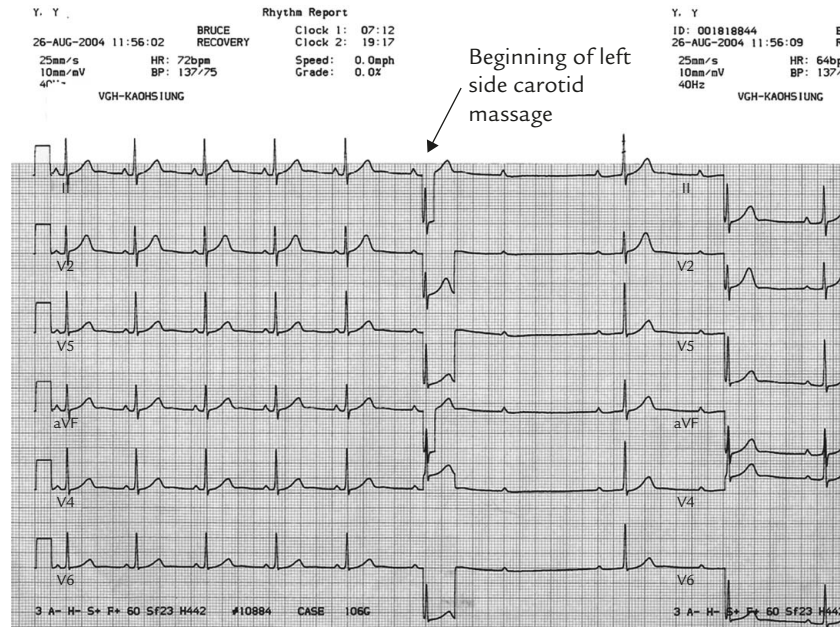


Figure 1. Electrocardiography (ECG) tracing while patient was receiving left-side carotid massage. The ECG showed sinus rhythm initially. Immediately after left carotid massage (arrow), atrioventricular block with prolonged ventricular pause developed. The patient experienced blackout with dizziness.

2003, sudden onset of chest tightness, diaphoresis and loss of consciousness occurred while she was swimming. Hypotension (76/48 mmHg) was noted at the local hospital. Further 24-hour ambulatory electrocardiogram monitoring (Holter) disclosed sinus bradycardia in the daytime (35 beats/min).

This time, the patient presented with repeated syncope in the past 1 month. She thus sought medical attention at our emergency department. The physical examination revealed clear consciousness. No orthostatic hypotension was found. We heard no bruits on carotid auscultation. Heart beat was regular on examination, with a grade II/VI systolic murmur presented at apex. The radial pulse was symmetric bilaterally, and no neurologic deficit was present. Laboratory examination showed mild normocytic anemia and normal glycemia.

We arranged a series of examinations for the patient after admission. Both awake and asleep electroencephalography revealed normal results. Echocardiography showed thickened aortic valve with mild mitral regurgitation and essentially preserved left-ventricular systolic function. Only rare atrial premature contractions (75 beats/23 hours and 58 minutes) and rare ventricular premature contractions (12 beats) were recorded on Holter exam. No sinus pause was found, nor was tachyarrhythmia recorded. Confirming that no significant carotid stenosis was present, we performed carotid massage to assess the possibility of carotid sinus hypersensitivity. Electrocardiography (ECG) showed normal

sinus rhythm before massage, but complete atrioventricular (AV) block with prolonged ventricular pause developed on left carotid sinus massage (Figure 1). The patient felt dizziness and nearly fainted immediately. Blood pressure was 137/75 mmHg initially, but 30 seconds after left carotid massage, it was 84/50 mmHg as recorded by noninvasive blood pressure monitoring, while the ECG tracing continued to show normal sinus rhythm with a heart rate of 66 beats/min.

The patient underwent coronary angiography, which disclosed insignificant coronary artery disease, and left ventriculography showed preserved left-ventricular systolic function without focal wall motion abnormality. Further electrophysiologic study revealed immediate fall in blood pressure after carotid sinus massage (from 176/69 mmHg, mean 108 mmHg to 120/50 mmHg, mean 73 mmHg after carotid massage) by simultaneous arterial monitoring. Both atrial and ventricular rates dropped (Figure 2). The corrected sinus node re-entry time was 1,737 ms, and atrial to ventricular one-to-one conduction time was 540 ms (Figure 3). Thus, sinus node dysfunction and poor AV node function were impressed.

Owing to the above findings, we suggested that she undergo permanent pacemaker (DDDR mode) implantation. We prescribed selective serotonin reuptake inhibitor (SSRI) under the impression of mixed carotid sinus hypersensitivity with satisfactory results. She did not suffer from dizziness or syncope after

treatment. She now enjoys her daily life quite normally, including riding a motor scooter and swimming.

Discussion

Carotid sinus syncope, a kind of neurally mediated syncope (Table 1), was first raised in 1933 as a cause of syncope.² Tea et al³ suggested that the neurovascular

structures surrounding the carotid mechanoreceptors are involved in carotid sinus syndrome, such as carotid sinus massage, head turning, neck extension and wearing tight collars. It presents as dizziness or syncope owing to transient cerebral hypoperfusion. It occurs predominantly in elderly males. Concurrent morbidity and mortality includes increased risk of head injury

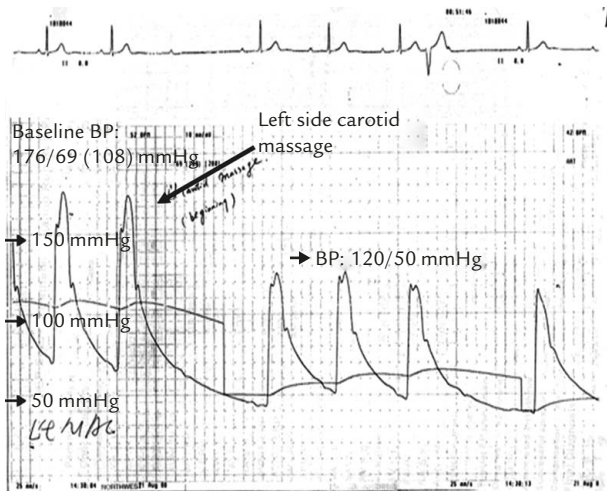


Figure 2. Blood pressure monitoring during carotid massage with electrocardiography tracing is shown in the top panel. Blood pressure dropped immediately after massage with atrioventricular conduction block and prolonged ventricular pause. The patient experienced blackout with severe dizziness at the same time.

Table 1. Neurally mediated syncopal syndromes

Vasovagal syncope (common or emotional faint)
Carotid sinus syncope
Postmicturition syncope
Airway stimulation
Cough syncope
Sneeze syncope
Gastrointestinal stimulation
Swallow syncope, defecation syncope
Raised intrathoracic pressure
Trumpet playing, weight lifting
Glossopharyngeal neuralgia
Miscellaneous
Syncope associated with aortic stenosis
Syncope accompanying onset of certain tachyarrhythmias (atrial fibrillation, paroxysmal supraventricular tachycardia, and possibly certain episodes of ventricular tachycardia)

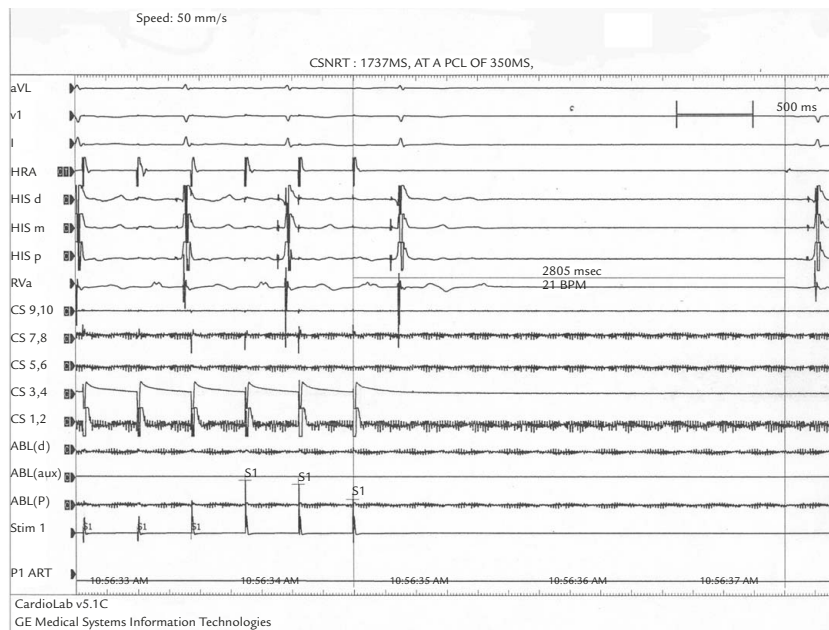


Figure 3. Corrected sinus node recovery time (CSNRT) recorded during electrophysiologic study. CSNRT was markedly prolonged (1,737 ms), which was suggestive of sick sinus syndrome.

and bone fracture. The condition can be classified into 3 types. The first is the cardioinhibitory type, which denotes ventricular asystole of more than 3 seconds on carotid sinus stimulation and is the most common type, accounting for 70–75% of all manifestations. The ECG manifestation includes sinus arrest, sinoatrial exit block and, less commonly, AV conduction block. The second type is the vasodepressor type, accounting for 5–10% in prevalence. It presents as a drop in systolic blood pressure of more than 50 mmHg without a drop in heart rate or drop in systolic blood pressure of 30 mmHg with reproducible symptoms. The third is the mixed type, consisting of both cardioinhibitory and vasodepressor types. It exists in 20–25% of all reported cases. To our knowledge, carotid hypersensitivity is a common cause of syncope; however, no previously reported case has shown both carotid hypersensitivity and sick sinus syndrome contributing to syncope, as demonstrated in our case.

Both pharmacologic and non-pharmacologic interventions have been used in the treatment of hypersensitive carotid sinus syndrome in correspondence to the disease nature. The pacemaker has maintained a key role in treating the cardioinhibitory type. It is also effective in sick sinus syndrome-related syncope, with dual-chamber pacing being more favored.⁴ Beta-blockers, SSRIs and alpha-agonists such as midodrine^{5,6} have shown clinical benefit in patients with neurally mediated syncope including carotid hypersensitivity syndrome.⁷ Grubb et al⁸ observed apparently beneficial effects of sertraline in 1 case and fluoxetine in another of carotid sinus hypersensitive syndrome that was unresponsive to dual-chamber pacing. Fludrocortisone, which increases sodium and fluid retention, has been reported to sensitize an adrenergic receptor (suggesting a possible synergism with midodrine), and was used as a first choice in younger individuals without other cardiovascular disease.^{9,10} However, its relation with hypersensitive

carotid sinus syndrome is not clear. In our case, we suggested the patient to have dual-chamber pacing with fluoxetine, which is the standard treatment for both sick sinus syndrome and hypersensitive carotid sinus syndrome, and she responded well clinically. This elderly patient was discharged in a stable condition and has been free from recurrent syncope thereafter.

References

1. Pai CH, Hu WH, Wang KY, Ting CT. Measurements of heart rate variability in patients with unexplained syncope. *J Chin Med Assoc* 1995;56:292–7.
2. Weiss S, Baker JR. The carotid sinus reflex in health and disease. *Medicine* 1933;12:297–351.
3. Tea S, Mansourati J, L'Heveder G, Mabin D, Blanc JJ. New insights into the pathophysiology of carotid sinus syndrome. *Circulation* 1996;93:1411–6.
4. Rinfret S, Cohen DJ, Lamas GA, Fleischmann KE, Weinstein MC, Orav J, Schron E, et al. Cost-effectiveness of dual-chamber pacing compared with ventricular pacing for sinus node dysfunction. *Circulation* 2005;111:165–72.
5. Gilden JL. Midodrine in neurogenic orthostatic hypotension. A new treatment. *Int Angio* 1993;12:125–31.
6. Low PA, Gilden JL, Freeman R, Sheng KN, McElligott MA. Efficacy of midodrine vs placebo in neurogenic orthostatic hypotension: a randomized, double-blind multicenter study. Midodrine Study Group. *JAMA* 1997;277:1046–51.
7. Benditt DG, Fahy GJ, Lurie KG, Sakaguchi S, Fabian W, Samniah N. Pharmacological therapy of neurally mediated syncope. *Circulation* 1999;100:1242–8.
8. Grubb BP, Samoil D, Kosinski D, Temesy-Armos P, Akpunonu B. The use of serotonin reuptake inhibitors for the treatment of carotid sinus hypersensitivity syndrome unresponsive to dual chamber pacing. *Pacing Clin Electrophysiol* 1994;17:1434–6.
9. Grubb BP, Kosinski D. Current trends in etiology, diagnosis, and management of neurocardiogenic syncope. *Curr Opin Cardiol* 1996;11:32–41.
10. Scott WA, Pongiglione G, Bromberg BI, Schaffer MS, Deal BJ, Fish FA, Dick M. Randomized comparison of atenolol and fludrocortisone acetate in the treatment of pediatric neurally mediated syncope. *Am J Cardiol* 1995;76:400–2.