CARBAMAZEPINE INDUCED SINUS NODAL DYSFUNCTION - A CASE REPORT

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ABSTRACT
Carbamazepine, a common drug used for the treatment of seizure disorders and neuralgias. It can cause sinus nodal dysfunction in some cases. We report a case of sinus nodal dysfunction occurring in a 47-years-old female while on therapy with carbamazepine with no structural heart disease.

KEYWORDS: Carbamazepine; sinus nodal dysfunction; seizure disorder.

INTRODUCTION
Sinus node dysfunction (SND) refers to abnormalities in SN impulse formation and propagation and includes sinus bradycardia, sinus pause/ arrest, chronotropic incompetence, and sinoatrial exit block.[1] Drugs such as beta blockers and calcium channel blockers can suppress SN function, causing marked SN bradycardia, as well as prolonged SN recovery time (SNRT) and sino-atrial conduction time (SACT). Carbamazepine, a tricyclic antidepressant derivative, is widely prescribed for the treatment of epilepsy and trigeminal neuralgia. It is also known to have antiarrhythmic action. In addition, suppression of sinus nodal automaticity and atrioventricular conduction has been reported in few subjects.[2,3]

Sinus bradyarrhythmia is a major complication of carbamazepine, mainly involving individuals with latent sinus node dysfunction. Suppression of sinus node function can occur at the normal therapeutic level of carbamazepine. Dysfunction could appear approximately 10 to 20 hours after the maximum plasma level of the drug is reached. This delay might
constitute the time required for carbamazepine to affect the sinus node cells and to induce marked sinus bradyarrhythmia.\[4\]

**CASE REPORT**

A 47 year old housewife presented to medical emergency with history of two episodes of syncope and around 15-20 episodes of giddiness in last 3 months. The patient was conscious and oriented at the time of presentation. She was a diagnosed case of trigeminal neuralgia for which she was taking carbamazepine 600 mg/day. She did not have any other neurologic symptoms. She was found to have sinus arrhythmia on electrocardiogram and was planned for further evaluation with a provisional diagnosis of sick sinus syndrome. Her echocardiogram did not reveal any structural heart disease. Holter monitoring for 24 hours showed a sinus pause of 3.5 seconds with corresponding event of giddiness. A diagnosis of carbamazepine induced sinus nodal dysfunction. Carbamazepine was replaced with lamotrigine. The patient was discharged in a satisfactory condition. On follow up visits at 3 months and 1 year, she was asymptomatic without any complaints of giddiness or syncope. Holter monitoring for 24 hours, repeated at 3 months showed no abnormality.

**DISCUSSION**

Carbamazepine (CBZ) is known to produce negative chronotropic and dromotropic effects on the cardiac conduction system which can cause various types of conduction disturbances including sinus node dysfunction and atrioventricular nodal block. These cardiac dysfunction are noted after 1-15 months of CBZ therapy.\[4\] There is a direct correlation between the plasma concentration of CBZ and the frequency of sinus arrest. CBZ aggravated lithium-induced sinus node dysfunction.\[5\] Elderly women are particularly susceptible to the bradyarrhythmogenic effect of CBZ. Life-threatening brady-arrhythmias or AV conduction delay had been described in elderly women, even at therapeutic levels.\[6\] Many case reports of bundle branch block, AV blocks and symptomatic sinus bradyarrhythmia in elderly subjects on CBZ, resolved after stopping CBZ.\[7\] The potential cardiotoxic effects have generally been observed in elderly female patients and at concentrations >40 mg/ml. The cardiotoxicity in young patients with no structural heart disease is not usually seen. In young persons with epilepsy but no underlying cardiac diseases, CBZ apparently had no significant effect on the conduction system.\[8\] But it can cause bradyarrythmias in younger patients very rarely. Our case shows a relationship between use of carbamazepine and occurring of sinus nodal dysfunction.
Carbamazepine is a chemical derivative of tricyclic antidepressants. It exerts its effects by interacting with phase I and III of the electric potential by primarily acting as a sodium channel blocker, similar to class 1A antiarrhythmic agents. The other drugs like barbiturates, valproate, succinamides, and benzodiazepines are not associated with significant effect on cardiac conduction system. Phenytoin may fasten the AV conduction. A prolongation of atrioventricular conduction and a decreased rate of phase 4 depolarization of autonomic fibers, suggests that CBZ may induce bradycardia in structurally normal cardiac conduction systems.

CONCLUSION
Carbamazepine can result in sinus node dysfunction and it is more common in elderly women. A detailed history is always important in all patients as it can search a rare cause of common disease. Permanent pacemaker implantation can be avoided in patients who have been on CBZ and their sinus nodal dysfunction reverts back to normal after cessation of carbamazepine.

REFERENCES