CASE REPORT

Carbamazepine induced atrial tachycardia with complete AV block

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Abstract

Background/Aim: Carbamazepine, a widely used antiepileptic drug that has been used for the treatment of both partial and generalized seizures, for trigeminal neuralgia, as a mood stabilizer and for treatment of neuropathic pain syndromes, may have negative chronotropic and dromotropic effects on the cardiac conduction system.

Description of case: We report a case of cardiac syncope due to atrial tachycardia combined with complete atrioventricular block as a consequence of carbamazepine administration for trigeminal neuralgia.

Conclusion: Although sinus tachycardia is the most frequently observed cardiac side effect of carbamazepine, sinus and nodal bradycardia, atrioventricular block, premature ventricular contractions, ventricular tachycardia and junctional escape rhythms have been reported in patients due to carbamazepine toxicity. Hippokratia 2014; 18 (2):185-186.

Keywords: Carbamazepine, atrial tachycardia, complete atrioventricular block

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Introduction

Carbamazepine is a wide used antiepileptic drug which has been used for the treatment of both partial and generalized seizures, for trigeminal neuralgia, as a mood stabilizer and for treatment of neuropathic pain syndromes[1]. Carbamazepine may have negative chronotropic and dromotropic effects on the cardiac conduction system[2]. We report a case of cardiac syncope due to complete atrioventricular block aggravated by carbamazepine prescribed for trigeminal neuralgia.

Description of case

An 82-year-old woman was admitted following a syncopal episode at home. She had a past medical history of coronary heart disease (myocardial infarction 4 years before) and during the last year she had been treated with aspirin 100 mg o.d, enalapril 5 mg o.d, atorvastatin 40 mg o.d and a low dose of carvedilol 3.125 mg o.d. Two days before her admission, carbamazepine 400 mg t.i.d. was prescribed for trigeminal neuralgia and she began to experience exhaustion. She reported losing consciousness on two occasions. On admission she had symptoms of congestive cardiac failure with low blood pressure (90/50 mmHg), a Glasgow Coma Scale of 11/15, and bradycardia noticed on Electrocardiography (ECG). The atrial rate was 135 beats per minute and the ventricular rate was 30 beats per minute with narrow QRS. There was isoelectric intervals between P waves in all leads, the rhythm was regular (constant RR and PP intervals) but the QRS complexes were independent to atrial P waves. The diagnosis of atrial tachycardia with complete AV block was made based on the ECG findings (Figure 1).

Laboratory investigations revealed that the patient had normal renal function with an estimated glomerular filtration rate of >60 ml/min. The hemoglobin, white cell count differentials and coagulation screen were all within the normal range. Serum concentration of carbamazepine could not be estimated by the hospital's laboratory. Thyroid function tests were normal. Isoproterenol was given as a bridge to temporary pacing, but due to gradual improving of patient's hemodynamics and mental status, temporary pacemaker was not placed. Twenty four hours later, sinus rhythm has been restored and she was completely free of symptoms (Figure 2).

Discussion

Carbamazepine therapeutic effect results from binding to sodium channels, reducing the phase 4 of depolarizationand thus affects the automaticity of pacemaker cells of the heart. In this way it may suppress the atrioventricular node and aggravate the bradycardia to complete heart block[3,4].

In carbamazepine toxicity, sodium channel blockade may cause prolongation of the QRS interval and predisposes patients to ventricular arrhythmias and hypotension[5].

Although sinus tachycardia is the most frequently observed cardiac side effect of carbamazepine, sinus and nodal bradycardia, atrioventricular block, premature ventricular contractions, ventricular tachycardia, and junctional escape rhythms have been reported in patients due to carbamazepine toxicity[6]. Reviewing literature two

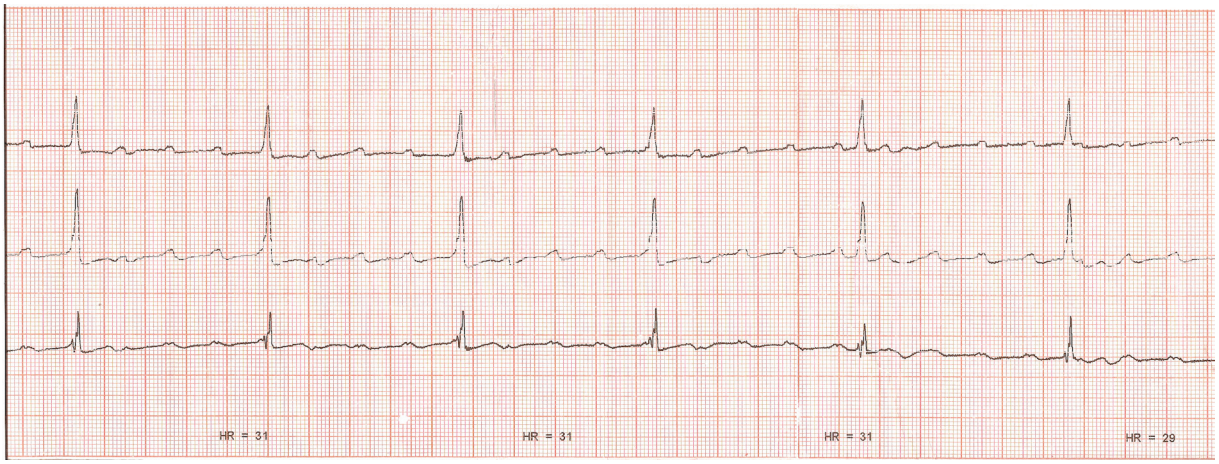

Figure 1: Atrial tachycardia with complete AV block. There are isoelectric intervals between P waves, the rhythm is regular (constant RR and PP intervals) and ventricular rhythm is independent to atrial rhythm with variable P-R intervals. Ventricular rate: 30 beats per minute, atrial rate: 135beats per minute.

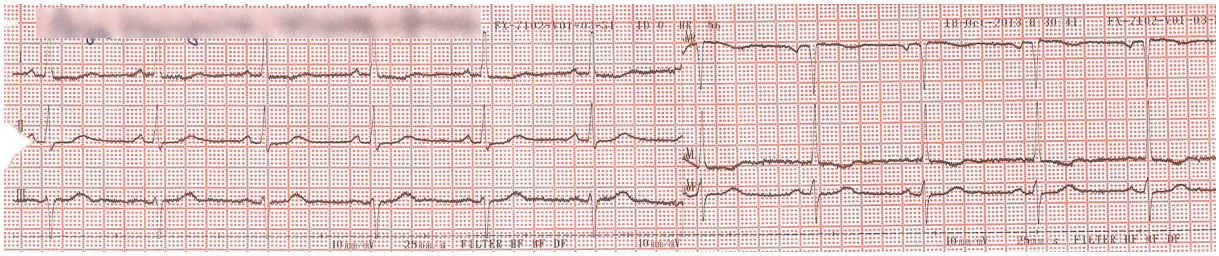

Figure 2: Restored sinus rhythm, twenty four hours later.

kinds of cardiac arrhythmias associated to carbamazepine were found. The first form occurs in young patients without pre-existing heart disease in which sinus tachycardia is common following carbamazepine overdose, taken usually with a suicidal intent. The second form occurs in most cases in elderly women who develop bradyarrhythmia or atrioventricular conduction delay in the setting of a therapeutic carbamazepine concentration, after long periods of therapy. In this form, symptoms rapidly resolve after discontinuation of the drug[7,8].

In our patient atrial tachycardia with complete AV block developed two days after administration of carbamazepine 400 mg t.i.d. B-blockers (as carvedilol) may contribute to complete AV block, but probable could not be the cause of atrial tachycardia at the same time. Additionally carvedilol had been taken for at least four years at the same dose by the patient without side effects. Carbamazepine and carvedilol probably contributed to complete AV block as the arrhythmia resolved rapidly following discontinuation of the drugs. No previous reports of cardiac conduction abnormalities with rapid atrial rhythm have been reported in patients receiving carbamazepine for trigeminal neuralgia or for any other reason.

Conflict of Interest

The authors declare no conflict of interest.

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