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Gabapentin-induced congestive heart failure

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Abstract

A 60 year-old man presented with acute congestive heart failure and atrial flutter 7 days after taking gabapentin prescribed for peripheral neuropathy. Gabapentin was discontinued and he became asymptomatic after standard therapy for heart failure and atrial flutter ablation. Although the patient probably had some degree of previously asymptomatic tachycardia-induced cardiomyopathy, the timing suggests that gabapentin played a role in precipitating heart failure.

Keywords: Peripheral neuropathy; Gabapentin; Congestive heart failure; Atrial flutter; Tachycardia-induced cardiomyopathy.

Introduction

Several studies involving gabapentin and its congener pregabalin have demonstrated that these agents may rarely cause acute congestive Heart Failure (HF) in patients with and without compensated HF [1,4]. This report describes a patient who presented with apparent gabapentin-induced precipitation of acute congestive HF only 7 days after starting a small dose of therapy. The patient also had atrial flutter and probable previously asymptomatic tachycardia-induced cardiomyopathy. The patient became asymptomatic upon discontinuation of gabapentin and appropriate therapy for heart failure.

Case report

A 60 year-old man was prescribed gabapentin 100 mg a day for non-diabetic peripheral neuropathy. There was no past history of alcohol abuse, hypertension, diabetes or heart disease. Apart from neuropathy, he was asymptomatic until he developed dyspnea on effort about a week after the start of gabapentin therapy. This quickly progressed to orthopnea, and paroxysmal nocturnal dyspnea for which he was hospitalized for acute congestive heart failure. The electrocardiogram on admission revealed atrial flutter that was not present at a routine examination 37 days before when the pulse rate was regular at 80 beats per minute. On physical examination, the jugular venous pressure was elevated, the pulse rate was irregular with an average rate of about 120 beats per minute and the blood pressure was 130/70. There was peripheral edema. The initial electrocardiogram revealed atrial flutter with varying degrees of atrioventricular block and an average rate ventricular rate of about 100 per minute. The chest X ray revealed pulmonary venous congestion and no cardiomegaly. The following laboratory investigations were normal: hematocrit, white cell and platelet counts, random glucose (89 mg/dL), electrolytes, calcium, cholesterol, triglycerides and troponin x 2. Other tests: BUN 36 mg/dL, creatinine 1.17 mg/dL, Pro-B-Natriuretic (BNP) 2711 pg/ml and abnormal liver function tests compatible with hepatic venous congestion. Test for coronavirus was negative. Echocardiography revealed global left ventricular dysfunction with an ejection fraction of 32%, mild mitral regurgitation and mildly dilated right ventricle, right and left atria. Rest and pharmacologic stress myocardial perfusion with wall motion analysis revealed

global hypokinesis and no fixed or reversible defects. A pulmonary angiogram revealed no evidence of pulmonary emboli. He was treated with furosemide, losartan, empagliflozin, sotalol, spironolactone and apixaban with marked improvement. Successful ablation of atrial flutter was performed before hospital discharge. The patient became asymptomatic. Five months later an echocardiography revealed a normal left ventricular function with an rejection fraction of 57%, normal diastolic parameters, a trace of mitral regurgitation, normal left atrial size and slightly dilated right atrium and right ventricle.

Discussion

The pharmacokinetics and side effects of pregabalin and gabapentin (gabapentinoids) tend to be similar. Several case reports have suggested that both pregabalin and gabapentin are associated with the development of acute congestive Heart Failure (HF), [1,5]. This has now been confirmed in number of large studies showing that both drugs can rarely precipitate acute congestive HF in patients with or without pre-existing compensated heart failure [6,10]. Our patient was found to be in sinus rhythm 37 days before the acute clinical event. Therefore, atrial flutter might have lasted 37 days or less but also longer if the arrhythmia was paroxysmal. The question then arises as to whether HF was unrelated to gabapentin therapy and caused only by tachycardia-induced-cardiomyopathy that eventually resolved because of atrial flutter ablation. Although the patient was asymptomatic before the sudden onset of HF, he may have had already a degree of myocardial dysfunction well before the acute clinical event. It is also possible but unlikely that the HF itself by causing acute left atrial dilatation might have created electrophysiologic disturbances responsible for the initiation of atrial flutter. A direct effect of gabapentin on the atria is remotely possible in view of the report of Ortiz de Landaluce et al who found a higher incidence of atrial fibrillation in patients taking gabapentin [11].

The patient was asymptomatic prior to the symptoms of HF. The early onset days of acute HF 7 days after the start of a relatively a small dose of gabapentin suggests a causal relationship. Indeed, the timing is compatible with the observations of Largeau et al who reported 9 cases of pregabalin-induced acute HF in whom the onset was 1 to 112 days (median 17 days) after the start of therapy [7]. Most of their patients that included two with pre-existing HF had a favorable outcome after discontinuation of pregabalin. The early timing of HF in the study of Largeau et al is consistent with that in previous case reports with pregabalin [1,2]. In this respect, the timing of acute HF caused associated with only a small dose of gabapentin (rather than pregabalin) was not stated in the large studies [8,10].

The impressive clinical presentation could be explained in terms of the superimposition of an acute depressive effect of gabapentin upon prevailing asymptomatic left ventricular dysfunction. This effect is comparable to the acute HF decompensation that may occur when gabapentin is administered to patients with compensated HF.

Conclusion

Gabapentin in small doses may precipitate acute HF if administered in a patient with underlying asymptomatic myocardial dysfunction. This effect is akin to the pregabalin or gabapentin-induced decompensation in patients with pre-existing compensated HF as reported in large series of patients.

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