

Letter to the Editor

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Amiodarone and Reversible Benign Intracranial Hypertension

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In December 1985, amiodarone was approved in the United States for treatment of life-threatening arrhythmias to be used only when other drugs were ineffective. Amiodarone has a wide spectrum of antiarrhythmic activity against both supraventricular and ventricular tachyarrhythmias and its efficacy led to much wider use in the United Kingdom. The high incidence of adverse reactions restricted the use of amiodarone in the United States of America. Amiodarone causes a variety of pulmonary, thyroid, ocular, hepatic, dermatologic and neurologic side effects [1]. Rarely optic neuritis, tremors, ataxia and pseudotumor cerebri have been reported to the manufacturer (< 1 % of patients) [2]. Nevertheless, to my knowledge, this appears to be the first report on a patient with this adverse drug reaction. Benign intracranial hypertension is a rare, usually self-limiting condition. Its exact cause is not known, but certain conditions such as pregnancy, Addison's disease and hypoparathyroidism, are usually associated with increased intracranial pressure [3]. Certain drugs such as corticosteroids, tetracycline, isotretinoin, TMP-SMX, danazol and nitroglycerin [4, 5] have been implicated in the causation of this problem. Review of the literature has not revealed any report in which the amiodarone was the culprit.

Report of a Case: I report a case of reversible benign intracranial hypertension associated with amiodarone therapy. In addition to enalapril, lovastatin and furosemide, amiodarone (400 mg/daily) was included in the treatment regimen of a 49-year-old man for control of congestive heart failure with ventricular arrhythmias. In 1994, he had undergone implantation with an automatic implantable cardioverter defibrillator (AICD) for life-threatening ventricular arrhythmias. Eight weeks after amiodarone therapy was initiated, the patient reported that he had severe headache for which he was reassured and advised to take paracetamol. The headache was continuous and accompanied by projectile vomiting. He was rather lethargic and weak, and was forced to lay flat in his bed because these symptoms were aggravated by change in posture such as bending forward. At this time, he also noticed multiple brief episodes of dimming or loss of vision. Neurologic examination revealed papilloedema and unilateral nonlocalizing sixth-nerve palsy and a diagnosis of intracranial hypertension was entertained. Amiodarone was withdrawn forthwith, and 3 days later, neurologic symptoms disappeared.

Physicians treating patients with amiodarone should be aware of this potentially dangerous adverse drug reaction, which has not been described before.

References

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