Pergolide and coronary artery dissection

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Introduction

This report describes a 48-year-old woman with Parkinson's disease who developed coronary artery dissection. We believe that dissection in this patient was probably caused by treatment with the anti-Parkinsonian drug pergolide.

Case report

A 48-year-old woman developed skeletal muscle tremor in 1996, when the diagnosis of Parkinson's disease of uncertain cause was made. Her symptoms were not well controlled with maximal doses of co-beneldopa, and supplementary treatment with pergolide was added in May 1999. She developed slight chest pain after the first 50 mcg dose.

Treatment with pergolide was continued in increasing dosage without further symptoms until six weeks later, when she had a further, more severe, episode of pain when taking 250 mcg pergolide three times daily. She was admitted to hospital as an emergency. Physical findings in the cardiovascular system were unremarkable and cardiac enzyme measurements were normal but a resting ECG showed extensive anterolateral ST segment and T wave changes consistent with the diagnosis of acute coronary insufficiency. Her blood pressure on admission was 100/70 mmHg. Conventional risk factors were negative.

She was treated conventionally with nitrates, atenolol, nifedipine and low-molecular-weight heparin, and pergolide treatment was reduced and ultimately stopped five days after admission. She continued to experience attacks of chest pain, and coronary angiography was performed. The findings on angiography were dissection in the distal half of a large anterior descending artery. The coronary angiogram was otherwise normal. In particular, no atheroma was observed in the coronary arteries.

Nifedipine was replaced by nicorandil after angiography, and over the next three or four days her symptoms slowly settled without further intervention. She was then mobilised gradually, and discharged from hospital some four weeks later on co-beneldopa with atenolol, nicorandil and aspirin.

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Discussion

Spontaneous coronary artery dissection is a rare condition that has been described in women of child-bearing age without conventional risk factors for premature vascular disease.[1,2] The condition has also been reported in young women treated in the puerperium with the ergopeptide derivative bromocriptine to prevent lactation.[3]

The mechanisms by which spontaneous coronary artery dissection may occur are not fully understood. Ergot alkaloids may cause coronary artery spasm in susceptible patients, and coronary artery spasm has been demonstrated at angiography after a bromocriptine challenge.[4] Dissection may therefore occur in association with coronary artery spasm in susceptible individuals with underlying vascular wall abnormalities, and those abnormalities could be potentiated by hormonal changes in pregnancy and the puerperium. To the best of our knowledge, this is as yet unproven in any clinical studies.

Pergolide is structurally similar to bromocriptine. We are unaware of any documented cases of coronary artery spasm of spontaneous dissection with the use of pergolide but dose-related angina has been reported.[5] We believe that pergolide-induced coronary spasm was the likely cause of dissection in this patient.

Parkinson's disease is very rare in women of child-bearing age. We recommend caution in the use of agents based on ergot derivatives in such patients, at least until there is a better understanding of the predisposing factors and pathological mechanisms involved in the development of coronary dissection.

The Committee on Safety of Medicines has been informed about this case.

References

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