Unilateral Uveitis Following Streptokinase Treatment For Myocardial Infarction

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Citation

Abstract
A 61-year old lady, presented to a hospital with chest pain, shortness of breath, sweating and nausea. She was diagnosed with an inferolateral myocardial infarction (MI), and thrombolysed with streptokinase (1.5 million units). Streptokinase is a protein agent used to dissolve thrombi in patients with myocardial infarctions or other thromboembolic events involving veins or arteries. Its use has been associated with immediate allergic reactions, such as bronchospasm and anaphylaxis,\(^1\) as well as delayed reactions, such as vasculitis and illness like serum sickness.\(^2,3\) Ocular involvement, although rare, can form part of this delayed reaction. We report a case of unilateral uveitis with iris atrophy, following the use of streptokinase for a myocardial infarct.

CASE REPORT
A 61-year old lady, presented to a hospital with chest pain, shortness of breath, sweating and nausea. She was diagnosed with an inferolateral myocardial infarction (MI), and thrombolysed with streptokinase (1.5 million units). She was also given diamorphine, aspirin and metoclopramide. The next day she complained of a painful, red left eye and reduced vision. On examination she was found to have corneal oedema in the left eye, a raised intraocular pressure (IOP), and a mid-dilated pupil. A presumed diagnosis of acute angle closure glaucoma was made and treated with intravenous and oral diamox and guttae pilocarpine.

However, although the IOP was normal the following day, she developed a hypopyon and vision dropped to hand movement. She was given depomedrone subcutaneously and commenced on guttae predforte hourly, guttae ciprofloxacin q.d.s., guttae chloramphenicol q.d.s., guttae atropine q.d.s., oral acetazolamide b.d and oral co-amoxiclav. After sufficient recovery from her MI, she was discharged from hospital and referred to our hospital for further ophthalmic care as this was closer to her home.

On presentation to our hospital 2 days later, she was found to have a visual acuity of counting fingers in the left eye. She was also found to have a marked anterior uveitic reaction with a blood-tinged hypopyon and sectoral atrophy on the nasal pupillary margin (Figures 1 and 2). Although hazy, examination of the posterior segment appeared to be normal.

Treatment was changed from guttae predforte to guttae maxidex hourly by day and 2 hourly by night, leaving her other medication unchanged. Two doses of subcutaneous betnesol were given on consecutive days and signs and symptoms gradually improved to a visual acuity of 6/9.
DISCUSSION

Although rare, unilateral and bilateral uveitis ± raised intraocular pressure has been reported as a side-effect of streptokinase infusion following treatment of acute MI. One case report describing this reaction following treatment with streptokinase for an assumed acute MI which subsequently was found not to be an MI provides evidence for the uveitis being a side-effect of the drug, rather than the MI.

The uveitis is a delayed response that typically presents within 24 hours of commencement of the streptokinase infusion. Although the mechanism is uncertain, due to lack of histopathological evidence, it has been postulated that it may represent an immune-complex hypersensitivity reaction, or may form part of the spectrum of drug-induced serum sickness.

One group found that up to 6% of patients treated with streptokinase had some degree of serum sickness.

To our knowledge there has not been any report in the literature, were iris atrophy occurred. Due to the potential sight-threatening complications that could arise due to delayed presentation, or secondary to unnecessary investigations such as anterior chamber tap to exclude endophthalmitis, it is important that this rare side-effect of streptokinase infusion is recognized. Appropriate treatment with topical ± subconjunctival steroids is the mainstay of treatment and results in rapid resolution of the uveitis.

References

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