Dilated Cardiomyopathy and sudden loss of vision

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Abstract

Heart Failure is a common cardiac condition managed by medications, most of which also cause hypotension as a side effect. Treatment aims to relieve symptoms thus improving exercise capacity, reduce incidence of acute exacerbations and mortality.

We present an interesting case of an uncommon side effect of hypotension leading to sudden loss of vision due to non arteritic ischemic optic neuropathy (NAION). It also highlights that once systolic blood pressure falls to 90 regular monitoring of blood pressure is required.

Background

Heart failure is common. Treatment is in the form of medications and device therapy. Medications which form the mainstay of treatment have a common side effect in the form of hypotension. Symptoms due to renal and cerebral hypo-perfusion are commonly seen. Rarely there can be sudden loss of vision caused by excessive decline in nocturnal blood pressure[1].

Case report

A 60 year man was referred with symptoms of increasing shortness of breath over a period of ten weeks with orthopnea and increase in weight up to 10 kg. He was a non-smoker, not a diabetic and had no significant family or alcohol history. On examination his blood pressure (BP) was 113/80 mmHg with a regular pulse of 110/min. On auscultation there was decreased air entry at both lung bases slightly more on the right. Chest Xray showed cardiomegaly with bilateral pleural effusions. There was left bundle branch block (LBBB) pattern on electrocardiogram (ECG). Echocardiography demonstrated left ventricular end systolic (LVES) and, left ventricular end diastolic (LVED) dimensions of 62 and 66 mm respectively, with a fractional shortening of 6%, suggesting severe left ventricular systolic impairment. Coronary angiogram was normal and a rest/stress single isotope SPECT scan with pharmacological stress and gated SPECT imaging showed dilated cardiomyopathy, most probably of non-ischemic origin.

He was started on evidence-based treatment for heart failure and the medications were titrated to: Furosemide 80 mg, candersartan 8mg, spironolactone 25mg, and aspirin 75 mg. On his last clinic appointment his BP was 90/70mm Hg supine, urea 14.8 and creatinine 117. He denied any symptoms of dizziness or light headedness.

Four months later he presented to eye casualty with sudden onset of general discomfort and ache in the right eye associated with symptoms of flashing lights. He also complained of visual loss in the lower field of the right eye. On examination of the right eye; visual acuity was 6/36 with no improvement with pin hole. Fundoscopy showed superior disc edema with sparing of the macula (Figure 1). There was right inferior altitudinal visual field defect. Examination of the left eye was normal with acuity of 6/4, normal fundus and visual fields. Ocular pressure was normal in both eyes.

His ESR was slightly raised at 40, but there were no other features to suggest giant cell arteritis. MRI scan of the brain was normal.

A 24 hour ambulatory BP measurement showed daytime (0700-2200hrs) mean arterial BP of 97/60, and night time (2200-0700hrs) of 87/50 mmHg, with minimum systolic of 79mmhg and minimum diastolic of 37 mmHg at 0120 hrs.

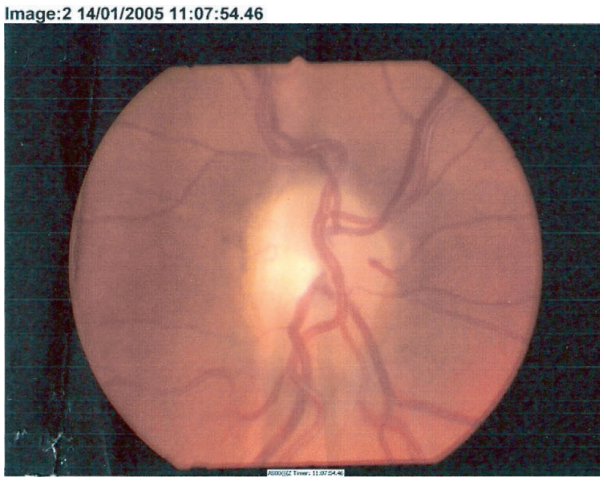

Figure 1. *Optic disc photograph of the right eye showing superior disc oedema. This caused an inferior altitudinal field defect.*

Diagnosis of non arteritic anterior ischemic optic neuropathy (NAAION) was made; possibly due to nocturnal hypotension. Heart failure medications were adjusted to improve BP, and his vision gradually improved over six months to 6/9 in the right eye.

Discussion

Treatment of heart failure aims to relieve symptoms, improve exercise capacity, reduce incidence of acute exacerbations and reduce mortality. Medications generally used for this are angiotenisn converting enzyme inhibitors (ACE inhibitors), loop and (sometimes) thiazide diuretics, beta blockers, aldosterone antagonist and digoxin. Except for digoxin, they also lower blood pressure, as most are efficient antihypertensive agents. Common practice is to titrate up the doses of these drugs according to symptom improvement, as long as the systolic BP is more than 90 and cerebral and renal perfusion are not compromised. European Society of Cardiology (ESC) guidelines on chronic heart failure 2005, suggest monitoring for symptomatic hypotension while patients are on treatment for heart failure[2]. Our patient did not complain of any symptoms due to hypotension prior to his acute visual loss.

NAAION is a well-recognised but less well understood condition as a common cause of sudden loss of vision in middle aged and elderly men and women[3,4]. Among the various associated risk factors are optic disc morphology, diabetes, rheumatoid arthritis and systemic arterial hypertension and nocturnal hypotension[5,6]. It is caused by acute impairment to the circulation of the arteries supplying the optic nerve. A drop in blood pressure at night while asleep is an important precipitating factor for the development of NAAION. Landau et al found that on ambulatory measurement of diurnal blood pressure, patients with NAAION had consistently lower mean blood pressure in the morning than did control subjects and a lag in the usual rise in blood pressure in the morning to meet increasing daytime demands for perfusion[7].

Typical presentation is of sudden and painless loss of vision in one eye without premonitory symptoms. Early findings show optic disc oedema and corresponding visual field defects. Giant cell arteritis should be excluded in all cases since corticosteroids are required in these cases. There is no proven treatment of NAAION. Surgical decompression has been tried in the past, but a recent study showed no benefit[8]. Steroids have also been tried but have shown no benefit. In a retrospective cohort study, aspirin was observed to be beneficial in decreasing NAAION in the fellow eye over 2 years, but not at 5 years. However, although treatment was not randomised; aspirin was not associated with a lower rate of recurrence in the contra lateral eye in the Ischemic Optic Neuropathy Decompression trial[9]. Our case highlights the fact that in patients with severe

heart failure, NAAION is a possibility due to a combination of the underlying cardiac condition plus the medications used to treat it.

Ambulatory BP monitoring should be considered in all patients once the BP gets to 90 mmHg systolic or less. Medications should be adjusted to prevent this.

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