Rapid onset intracranial hypertension in a young woman after starting nitrofurantoin

Introduction

A 24-year-old woman presented to the eye department with a 2-week history of headache and photophobia. This started 2 days after she had been started on a course of nitrofurantoin by her GP for a urinary tract infection. At presentation bilateral papilloedema was noted on examination. Neuroimaging was normal with the exception of prominent optic nerve sheaths. A lumbar puncture revealed a CSF pressure of over 40 cm. A diagnosis of nitrofurantoin-induced pseudotumour cerebri was made. Symptoms improved following the lumbar puncture, but the patient did require a tapering course of oral acetazolamide.

This case adds to the existing literature of nitrofurantoin in the aetiology of raised intracranial pressure. It highlights the speed at which symptoms may arise following commencement of the drug and demonstrates the possible ongoing effect despite cessation of the medication.

Discussion

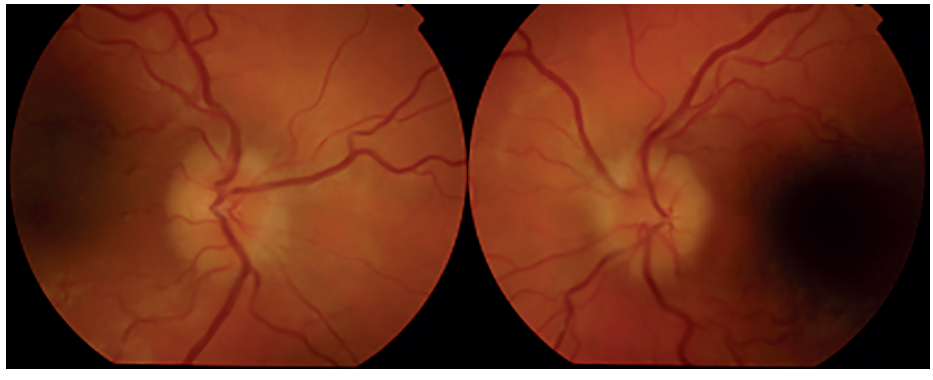
This article presents an interesting case of pseudotumour cerebri syndrome secondary to nitrofurantoin. This case highlights the unusual rapidity of onset, and the long duration it may take for full resolution of symptoms after stopping the medication. Nitrofurantoin as a cause of pseudotumour cerebri has been described in the literature since 1974 (Sharma and James, 1974; Mushet, 1977; Pringle et al, 2008). Previous cases report varying duration of antibiotic use before onset of symptoms, from 10 days to several months. This patient's symptom onset was within 2 days of starting nitrofurantoin. Other medications reported to cause intracranial hypertension include nalidixic acid, indomethacin, isotretinoin and lithium (Ball and Clarke, 2006).

This patient's case highlights the 'coasting' effect of many months before full resolution of symptoms despite stopping the offending medication. Lumbar puncture is diagnostic and may be therapeutic in some cases. Acetazolamide can be useful to manage symptoms during this period as the effect of nitrofurantoin tails off, as seen in this patient.

Conclusions

This case report highlights the potential adverse effect of pseudotumour cerebri syndrome secondary to nitrofurantoin. The onset of symptoms may be rapid, within 2 days in this patient, and may last for months despite stopping the medication. Acetazolamide can be useful for symptom treatment during this period. BJHM

Figure 1. Colour fundus photographs at presentation showing bilateral papilloedema.



CASE REPORT

A 24-year-old woman, with no previous past medical history, presented to the eye department with a 2-week history of a severe headache and photophobia. The headache was a frontal pressure, which worsened on bending forwards. She also complained of occasional blurry vision. Two weeks earlier she was started on a course of nitrofurantoin by her GP for a urinary tract infection. Two days into this course, she developed a continuous severe headache and blurred vision. She was referred to the eye department by her GP and was subsequently admitted for further investigations.

On examination, she had bilateral papilloedema with peripapillary folds (*Figure 1*). The rest of her ocular examination was normal. Humphrey visual fields showed enlarged blind spots and superior arcuate scotomas in both eyes. All other neurological examination was normal.

Computed tomography venogram excluded venous sinus thrombosis. Magnetic resonance imaging of the brain showed mild prominence of both optic nerve sheaths and heads with flattening of the posterior globes. A lumbar puncture performed in the left lateral position had an opening pressure of over 40 cm with normal CSF constituents.

She was diagnosed with pseudotumour cerebri syndrome, secondary to nitrofurantoin.

She was discharged but re-presented a week later with recurrence of headache and pulsatile tinnitus. She was therefore started on oral acetazolamide 500 mg twice daily.

On review 1 month later, her headaches had improved with occasional blurred vision. Papilloedema was still mildly present. The acetazolamide was gradually reduced over 6 weeks. At review 2 months later, she had full resolution of signs and symptoms.

Dr Patrick Hughes, Specialist Trainee in Ophthalmology, Department of Ophthalmology, Royal Devon and Exeter Hospital, Exeter, EX2 5DW

Dr Sui Wong, Consultant Neuro-ophthalmologist, Department of Ophthalmology, Moorfield's Eye Hospital and St. Thomas' Hospital, London

Correspondence to: Dr P Hughes (pw.hughes@yahoo.co.uk)

Ball AK, Clarke CE (2006) Idiopathic intracranial hypertension. *Lancet Neurol* **5**(5): 433–442. https://doi.org/10.1016/S1474-4422(06)70442-2

Mushet GR (1977) Pseudotumor and nitrofurantoin therapy. *Arch Neurol* **34**(4): 257. https://doi.org/10.1001/archneur.1977.00500160071019

Pringle Ho H, O'Sullivan E, Soper C (2008) Reversible bilateral optic disc swelling in a renal patient treated with nitrofurantoin. *NDT Plus* **1**(5): 344–345. https://doi.org/10.1093/ndtplus/sfn045

Sharma DB, James A (1974) Letter: benign intracranial hypertension associated with nitrofurantoin therapy. *BMJ* **iv**(5947): 771. https://doi.org/10.1136/bmj.4.5947.771-c

LEARNING POINTS

- Nitrofurantoin, among other medications, can cause raised intracranial pressure. Clinical features include headache, pulsatile tinnitus, transient visual obscurations and visual loss.
- This case demonstrates that the effects of nitrofurantoin can occur rapidly and may persist even after cessation of the medication.
- Taking a full drug history is imperative in patients presenting with headache and should include recently stopped medication.
- A lumbar puncture is both diagnostic and therapeutic in patients suspected of having idiopathic intracranial hypertension.
- Follow up in the eye department is required to ensure there are no ongoing effects which may necessitate a course of acetazolamide.

Ventricular bigemini or something less common?

A 55-year-old man was admitted with heart failure. He had undergone a heterotopic heart transplant 21 years earlier. The electrocardiogram shows two independent morphologies for the QRS complex, one from the native heart and a second from the donor heterotopic heart transplant (*Figure 1*).

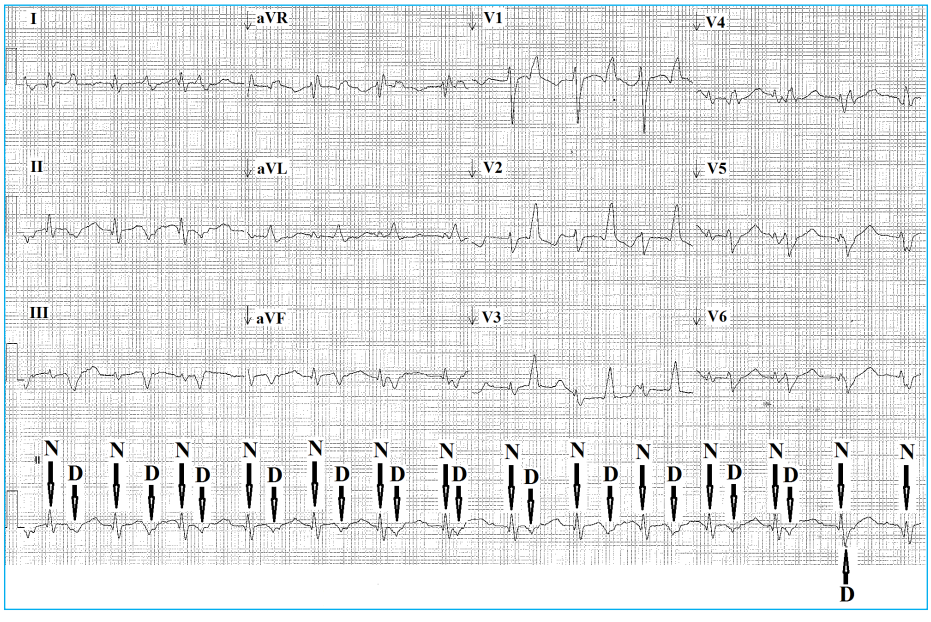
Heterotopic heart transplantation (which is also known as a 'piggyback' procedure) involves the surgical connection of a donor heart to the recipient's heart in a parallel fashion. The procedure was developed with the advantages that the donor heart is 'shielded' from refractory pulmonary hypertension and that it helps to compensate for significant donor–recipient size mismatch.

Further advantage derives from the native heart remaining 'viable' in the event of acute graft dysfunction with the native heart assisting the donor heart through the post-ischaemic recovery period. This advantage also applies in the event of severe graft rejection and malignant ventricular arrhythmias of the native heart. In effect, the native heart maintains the right circulation, often in the presence of significant pulmonary vascular resistance and pulmonary hypertension, while the donor heterotopic heart functions as a biological left ventricular assist device.

Heterotopic transplantation is far less common than the more conventional orthotopic procedure, being performed in around 0.3–0.4% of heart transplantations (Jahanyar et al, 2014). **BJHM**

Jahanyar J, Koerner MM, Ghodsizad A, Loebe M, Noon GP (2014) Heterotopic heart transplantation: The United States experience. *Heart Surg Forum* **17**(3): E132–E140. https://doi.org/10.1532/HSF98.2014328

Figure 1. 12-lead electrocardiogram showing two alternating, and mutually independent, morphologies for the QRS complex. D = QRS from the heterotopic donor heart; N = QRS from the native heart.



Dr Simon Dubrey, Consultant Cardiologist, Department of Cardiology, Hillingdon Hospital, Uxbridge, Middlesex UB8 3NN

Dr Georgios Karagiannis, Consultant Cardiologist, Department of Cardiology, Hillingdon Hospital, Uxbridge, Middlesex

Correspondence to: Dr S Dubrey (simon.dubrey@thh.nhs.uk)