Digoxin toxicity: an unusual presentation of infective endocarditis

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Abstract

We describe a case of infective endocarditis, which presented with digoxin toxicity. This case is of interest since the patient only became pyrexial six days after admission when blood cultures grew *Streptococcus viridans*. We believe this is the first case of infective endocarditis presenting with digoxin toxicity.

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Case report

A 64-year-old gentleman presented with a one week history of general malaise, blurred vision, anorexia and dizziness. His past medical history included hypertension, atrial fibrillation and congestive cardiac failure. He was taking digoxin 250 µg daily, enalapril 40 mg daily, doxazosin 4 mg daily, spironolactone 25 mg daily, frusemide 120 mg daily and warfarin. He was a non-smoker and drank 20 units of alcohol per week.

Examination revealed his blood pressure was 90/60 mmHg, he had atrial fibrillation (95 bpm), his temperature was 36.9°C and he had an ejection systolic murmur over the entire precordium. There was no lymphadenopathy or splinter haemorrhage; his chest was clear and abdomen was normal. He appeared dehydrated.

Urine analysis was negative including culture. Investigations showed concentrations of sodium 123 mmol/L, potassium 6.6 mmol/L, urea 35.1 mmol/L and creatinine 362 µmol/L. He was digoxin toxic, with a digoxin concentration of 3.0 µg/L, (range 1.0–1.4 µg/L). His white cell count was 13.7×10^9/L, neutrophils 11.5×10^9/L, haemoglobin 12.0 g/L, mean corpuscular volume 81.9, and platelets 301×10^9/L.

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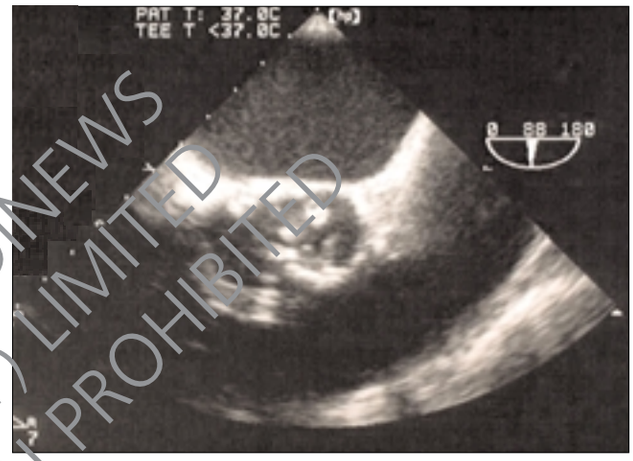
Figure 1. Echocardiogram showing vegetations on the aortic valve



Digoxin and spironolactone were stopped, frusemide was reduced to 40 mg daily and enalapril to 20 mg daily.

Six days later he became pyrexial. Three blood cultures grew *Streptococcus viridans*. He was started on benzylpenicillin and gentamicin intravenously. A transthoracic echocardiogram showed mixed aortic valve disease but no vegetations. A transoesophageal echocardiogram demonstrated three supravalvular vegetations on the aortic valve (figure 1) and there was mild aortic reflux. Other valves were normal.

He was continued on intravenous benzylpenicillin for six weeks and gentamicin for four weeks. Renal function normalised. Four weeks later, he was still apyrexial and his renal function was normal.

Discussion

On admission this patient did not have any signs of infective endocarditis. Renal impairment in infective endocarditis may be caused by renal infarction (septic emboli), glomerulonephritis, amyloidosis and renal abscess.[1-4] In a study, some 13.2% of patients who had infective glomerolunephritis were also found to have endocarditis.[5]

This renal impairment makes patients with infective endocarditis at risk of drug toxicity. In our case, we believe that renal impairment was caused by endocarditis leading to digoxin toxicity. On presentation, apart from an ejection systolic murmur – which is common in this age group – there were no signs of endocarditis. This

Key messages

- Digoxin (or other drug) toxicity could be an unusual presenting sign of infective endocarditis
- Early diagnosis of renal impairment caused by bacterial endocarditis is very important, as it usually responds dramatically to antimicrobial therapy
- If endocarditis is suspected, a transoesophageal echocardiogram should be performed

case illustrates that digoxin or other drug toxicity could be a presenting feature of endocarditis. Antimicrobial therapy results in a rapid decrease of circulating immune complexes and subsequent dramatic improvement of renal function.[2,6] Immunosuppression or plasmophoresis is rarely needed.[2] Our patient's renal function improved dramatically after antibiotics.

Conclusions

Digoxin toxicity is an unusual presenting sign of infective endocarditis. Toxicity with other drugs excreted by the kidneys can also potentially indicate endocarditis. Early diagnosis of such renal impairment is very important as it usually responds dramatically to antimicrobial therapy.

In this case, although the patient was apyrexial, more attention to the murmur and mild neutrophilia on admission might have led to an earlier diagnosis. We believe this case is the first reported case of endocarditis presenting with digoxin toxicity.

A transoesophageal echocardiogram should always be considered if endocarditis is suspected, even if the transthoracic echocardiogram is negative.

References

1. Kumar P, Clark M. *Clinical medicine*. 4th edn. London: W.B. Saunders, 1998.
2. Weatherall DJ, Ledingham JGG, Warrell DA. *Oxford textbook of medicine*. 3rd edn. Oxford: Oxford University Press, 1996.
3. Yoshida K, Matsubara H, Shibatani N *et al*. Immune complex-mediated glomerulonephritis associated with infective endocarditis with aortic valve vegetation. *Nippon J Inzo Gakkai Shi* 1998;**40**:359-63.
4. Barnes E, Frankle A, Brown EA, Woodrow D. Glomerulonephritis associated with permanent pacemaker endocarditis. *Am J Nephrol* 1995;**15**: 436-8.
5. Montseny JJ, Meyrier A, Kleinknecht D, Callard P. The current spectrum of infectious glomerulonephritis. Experience with 76 patients and review of literature. *Medicine Baltimore* 1995;**74**:63-73.
6. Masuda T, Suzuki A, Obana M, Matsuoka Y, Irimajiri S. A case of infective endocarditis with purpura and macroscopic haematurea as initial manifestations. *Kansenshogaku Zasshi* 1996;**70**:366-70.

Diary

2003

9th-10th September: Coronary Heart Disease Collaborative Workshops, Manchester (free to NHS staff)
1. Rehabilitation Workshop. Contact: Linda Binder, tel: 07747 603978, email: linda.binder@npat.nhs.uk
2. Angina/PCI Workshop. Contact: Sheelagh Machin, tel: 07771 980846, email: Sheelagh.Machin@npat.nhs.uk
3. Cardiac Surgery Workshop. Contact: Julie Harries, tel: 07810 836305, email: Julie.Harries@npat.nhs.uk

17th-19th September: Joint British Atherosclerosis Society and British Society for Haemostasis & Thrombosis, Cambridge
Contact: Natasha Dougall, tel: 01922 457984, fax: 01922 455238, email: natashadougall@wheldonevents.freeserve.co.uk, website: www.britathsoc.ac.uk

19th-20th September: 2nd Joint European/North American Symposium on Congenital Heart Disease in the Adult, Santorini, Greece.
Contact: Michael Gatzoulis, tel: 020 7351 8227, fax: 020 7351 8629, email: m.gatzoulis@rbh.nthames.nhs.uk, website: www.rbh.nthames.nhs.uk/ACHD 2003/

2nd October: Coronary Heart Disease Collaborative: Heart Failure Workshop, Birmingham (free to NHS staff)
Contact: Jim Heys, tel: 07810 836302, email: Jim.Heys@npat.nhs.uk

3rd-4th October: Primary Care Cardiovascular Society Annual Scientific Meeting and AGM, Dublin, Ireland.
Contact: PCCS, email: office@pccs.org.uk, website: www.pccs.org.uk

19th-22nd October: 5th International Congress on Coronary Artery Disease from Prevention to Intervention, Florence, Italy
Contact: Secretariat, tel: +41 22 908 0488, fax: +41 22 732 2850
email: coronary@kenes.com, website: www.kenes.com

24th-25th October: British Association of Cardiac Rehabilitation Annual Conference, Harrogate
Contact: The Affiliated Office, British Cardiac Society tel: 020 7692 5413, fax: 020 7383 5961 web: www.bcs.com

9th-12th November: Scientific Sessions 2003 of the American Heart Association, Orlando, Florida, USA
Contact: Scientific Sessions, tel: 214/706 1543, fax: 214/706 5262, email: sessions@heart.org