Other full case

Can we really do without antibiotic prophylaxis for infective endocarditis?

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Summary

Current guidance recommends against the use of antibiotic prophylaxis around the time of dental intervention for the prevention of infective endocarditis. The authors report the case of a previously well-patient with an asymptomatic isolated ventricular septal defect (VSD) who developed evidence of infective systemic and pulmonary emboli following dental treatment. A diagnosis of severe endocarditis of a previously normal native mitral valve was made. She subsequently underwent surgical repair of her mitral valve, and closure of her VSD. She was deemed fit for discharge on parenteral antibiotics on the thirtieth postoperative day. The authors highlight the need for further re-evaluation of the issues surrounding antibiotic prophylaxis for endocarditis in the context of dental procedures.

BACKGROUND

Infective endocarditis is an uncommon disease with a high mortality and morbidity despite optimal treatment. The in-hospital mortality for infective endocarditis has been quoted to lie between 16% and 27%.¹

An association between infective endocarditis and dental treatment has long been postulated.² Until recently, antibiotic prophylaxis prior to dental procedures had been employed for the prevention of infective endocarditis in patients with pre-existing structural cardiac disease who were thought to be at risk. Indeed, this practice was once almost universal.³

In 2008, National Institute for Health and Clinical Excellence (NICE) published a guidance⁴ that departed

from the previously considered best practice. NICE recommended *the avoidance of antibiotic prophylaxis altogether in patients undergoing dental procedures*. This guidance included patients that were deemed to be at high-risk of developing infective endocarditis (table 1).

NICE guidelines were developed following a review of the available literature. The working group found that there was insufficient evidence to determine the effectiveness of prescribing antibiotic prophylaxis around the time of dental intervention in reducing the incidence of infective endocarditis.

We report the case of a patient with a known pre-existing asymptomatic ventricular septal defect (VSD) who underwent minor dental treatment without antibiotic

Figure 1 A CT slice of the abdomen demonstrating splenic infarcts (arrows).

Figure 2 A CT slice of the chest demonstrating pulmonary infiltrates (arrows).

prophylaxis and in whom life-threatening endocarditis subsequently occurred.

By reporting this case we aim to once again raise the issue of endocarditis prophylaxis. In addition we hope to prompt a re-evaluation of the current guidance on the prescription of antibiotic prophylaxis in the context of dental treatment.

CASE PRESENTATION

The patient is a 42-year-old female healthcare professional who had been diagnosed with a small isolated VSD from birth. Her native cardiac valves were otherwise normal. In view of her lack of symptoms this had been managed conservatively with observation and serial echocardiography. She had been otherwise fit and well, with no evidence of immunodeficiency. She had maintained regular contact with her dentist, and did not report any history of gingivitis or dental infection.

It is noteworthy that prior to 2008 she had received prophylactic antibiotics for all dental work. Following the publication in that year of the aforementioned NICE guidelines,⁴ and as she was not immunodeficient, she was no longer offered antibiotic prophylaxis. Indeed 6 months prior to her hospital presentation she had undergone a periodontal procedure without the cover of antibiotics.

Several days after this dental work was undertaken she presented to her family practitioner with symptoms of fever, loss of appetite, sweating and weight loss. These symptoms were initially attributed to early menopause. She described feeling more unwell over several months. With this background, some 5 months later, she presented to her local hospital with sudden onset of pain and numbness in her right leg.

On examination, she appeared unwell, with a tympanic temperature of 38.3°C. She was pale, tachycardic with a heart rate of 120 bpm and had a non-invasive blood pressure measurement of 97/56 mm Hg. She exhibited splinter haemorrhages on three fingers, and on auscultation

there was a soft pansystolic murmur heard loudest in the mitral area and radiating to the axilla. Oxygen saturation was measured to be 96% on air and her abdomen was soft and non-tender. Her right leg was cool and the dorsalis pedis, popliteal and femoral pulses were significantly reduced on the right hand side. She was unable to mobilise from bed.

INVESTIGATIONS

Initial blood investigations revealed a neutrophilic leukocytosis with a white cell count of $26 \times 10^9/l$, a C reactive protein (CRP) concentration of 256 mg/l and an erythrocyte sediment rate of 120 mm/h.

Her full blood count revealed a microcytic anaemia with a haemoglobin concentration of 7 g/dl. Plasma urea, creatinine and electrolyte concentrations were within normal limits.

Blood cultures taken at initial presentation yielded *Streptococcus mitis* and *Streptococcus oralis*.

An arterial Doppler ultrasound scan of her right leg revealed an embolus occluding the right external iliac artery. In light of this finding, and based on her poor clinical condition, a CT scan was ordered to delineate a potential source of the embolus.

CT of chest, abdomen and pelvis revealed multiple low attenuation lesions in the spleen and lungs (figure 1 and 2) consistent with splenic and pulmonary infarcts, respectively.

2D transthoracic echocardiography revealed a mobile hyperechoic mass on the anterior mitral valve leaflet. In addition, a small VSD was visualised, with associated hyperechoicity (figure 3). The appearances were highly suggestive of vegetations on the anterior mitral valve leaflet and in intimate relation to the VSD.

DIFFERENTIAL DIAGNOSIS

A diagnosis was made of infectious endocarditis affecting her VSD and mitral valve, with multiple emboli to the

Figure 3 A 2Dimensional transthoracic echocardiograph with colour-flow Doppler demonstrating hyperechoic material on the anterior mitral valve leaflet (arrow) and the presence of abnormal blood flow across the ventricular septum.

Figure 4 A postoperative transoesophageal echocardiograph demonstrating the repaired anterior mitral valve leaflet, and closure of the ventricular septal defect. Right panel includes colour-flow Doppler.

systemic (spleen and the right common iliac artery) and to the pulmonary circulations.

TREATMENT

Antibiotic therapy with benzylpenicillin and gentamicin was initiated under microbiological guidance, and the organisms in question were later confirmed to be sensitive to both.

A vascular surgical opinion regarding the right leg was sought, following which it was elected to manage the limb conservatively with a continuous infusion of intravenous heparin.

Due to her continued constitutional deterioration, the size of the anterior mitral leaflet vegetation and the evidence of multiple emboli, it was decided that she should undergo surgical intervention.

At operation the VSD was identified and its surrounding tissues were also seen to be affected by the endocarditic process. The infective tissue of the septum had also infected the anterior mitral valve leaflet, which exhibited macroscopic features of severe infection. A large vegetation was found on the A2 segment.

The affected segment of the anterior leaflet of the mitral valve was resected and sent for microbiological

Table 1 Characteristics of those groups deemed to have a high risk of developing infective endocarditis⁴

Acquired valvular heart disease
Valve replacement
Structural congenital heart disease*
Previous infective endocarditis
Hypertrophic obstructive cardiomyopathy

*Including surgically corrected structural conditions, but excluding isolated atrial septal defect, fully repaired ventricular septal defect or fully repaired patent ductus arteriosus, and closure devices that are judged to be endothelialised.

examination. The anterior leaflet was patched with a small piece of autologous pericardium. The area around the VSD was debrided and the defect was closed, with an equine pericardial patch.

OUTCOME AND FOLLOW-UP

The intraoperative samples that had been sent for microbiological investigation later yielded growth of viridans streptococci—*S. mitis* and *S. oralis*. The patient therefore received a 4-week course of postoperative intravenous benzylpenicillin and gentamicin, to both of which the organisms concerned were found to be sensitive. During her postoperative course she was afebrile and white cell count and CRP normalised.

Her postoperative recovery was complicated by an atrioventricular block, which needed treatment using a permanent pacemaker. However, she was discharged home fully mobilised on the thirtieth postoperative day prior to which a transoesophageal echocardiogram revealed competent valvular function with no residual VSD (figure 4).

DISCUSSION

Current NICE guidelines do not recommend the routine use of antibiotic prophylaxis prior to dental intervention for the purpose of preventing infective endocarditis, even in those groups (table 1) deemed to be at a higher risk of developing the disease. This guidance has been issued based on the lack of consistent evidence to support the use of prophylactic antibiotics in these circumstances.

This case illustrates the morbidity associated with infective endocarditis when it does occur. While the dental work in question in this case was of a superficial hygienic nature and the hospital presentation was several months later, the onset of symptoms was temporally related to the dental work. At the very least this case should prompt a discussion about the wisdom of such guidelines recommending the undertaking of dental work in patients with known structural heart disease without prophylactic antibiotic cover.

There is good evidence that dental interventions result in significant bacteraemia in humans.⁵ In addition, these interventions do not necessarily need to be invasive. Even periodontal procedures are associated with transient bacteraemias.⁶

However, a causal relationship between this transient bacteraemia associated with dental interventions and the development of infective endocarditis in patients with structurally abnormal hearts has been neither proven nor disproven.

Several attempts have been made to analyse the role of prophylactic antibiotics in reducing the bioburden associated with dental intervention.⁷ Indeed the evidence reviewed by the NICE working group did appear to show that the administration of prophylactic antibiotics leads to a reduction in the level of bacteraemia associated with dental intervention.

Currently there is no evidence to suggest that prophylactic antibiotics could reduce the incidence of infective endocarditis in patients with structurally abnormal hearts. There has been no randomised controlled trial addressing this hypothesis. Furthermore it is unlikely that such a trial could be designed. In part, this is due to the very low incidence of infective endocarditis.

Several case reports, however,^{8–10} have suggested an association between dental intervention and the development of infective endocarditis.

It is in this context that we suggest that it may be time for a further re-examination of the issues highlighted here. If national data for the incidence of infective endocarditis before and after this change in accepted practice as a result of the published NICE guidelines could be examined, then any increase in the incidence of infective endocarditis might be enough for NICE to re-evaluate its conclusions.

It seems at the very least questionable to cite a lack of conclusive evidence as the basis for dismissing the idea that antibiotic prophylaxis might save lives in the context of high-risk patients undergoing dental procedures. If the evidence is indeed inconclusive, any recommendations should be made with caution. We suggest that reports such as this add voice to those who would question the rationale behind the current NICE guidelines.

Learning points

- ▶ Infective endocarditis is still accompanied by significant morbidity and mortality.
- ▶ Evidence reveals that dental treatment does cause transient bacteraemia.
- ▶ Evidence in support of, or against, antibiotic prophylaxis in the context of dental work is lacking.
- ▶ Current guidance does not recommend antibiotic prophylaxis prior to dental treatment for the purpose of preventing endocarditis, even in patients thought to be at high risk.
- ▶ The authors recommend a review of current UK guidance regarding this topic.

Competing interests None.

Patient consent Obtained.

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