Table 1 Laboratory findings of immune-mediated thrombocytopenic purpura related to *Toxoplasma gondii* infection

Variable	Period of the toxoplasmosis and immune-mediated thrombocytopenic purpura			
	Mononucleosis-like	Immune purpura	1 week after	1-month follow-up
Hemoglobin (g/dl)	12.0	12.4	11.8	13.4
Hematocrit (%)	36.4	39.3	38.2	43.6
White cell count ($\times 10^9$/l)	9.46	6.74	7.74	7.77
Neutrophils ($\times 10^9$/l)	4.90	2.40	3.90	4.30
Lymphocytes ($\times 10^9$/l)	3.00	3.90	2.20	2.50
Platelets ($\times 10^9$/l)	395	5	172	299
C-Reactive protein (mg/l)	29.4	2.58	1.59	0

Acknowledgement

We thank Helena Petta, André Siqueira Machado, and Silvia Goulart for clinical and laboratory assistance.

Conflict of interest: No conflict of interest to declare.

References

1. Bhopale GM. Pathogenesis of toxoplasmosis. *Comp Immunol Microbiol Infect Dis* 2003;**26**:213–22.
2. Deutsch M, Nezi V, Kountouras D, Belegrati M, Kalmantis T, Dourakis SP. Immune-mediated thrombocytopenic purpura associated with *Toxoplasma gondii* infection in an immunocompetent patient. *Hematol J* 2004;**5**:538–9.
3. Hohlfeld P, Forestier F, Kaplan J, Tissot J, Daffos F. Fetal thrombocytopenia: a retrospective survey of 5,194 fetal blood samplings. *Blood* 1994;**84**:1851–6.
4. Borderon JC, Pesnel G, Canet J, Fabre A, Solle R, Lajouanine P. Thrombopenic purpura and acquired toxoplasmosis. *Ann Pediatr (Paris)* 1969;**16**:129–32.
5. Gurkan E, Baslamisli F, Guvenc B, Bozkurt B, Unsal C. Immune thrombocytopenic purpura associated with Brucella and Toxoplasma infections. *Am J Hematol* 2003;**74**:52–4.
6. Michez D, Quintart C, Noel E, Lepage P. Peripheral thrombopenic purpura associated with acquired toxoplasmosis. *Rev Med Brux* 1998;**19**:135–7.
7. Yenicesu I, Yetgin S, Ozyurek E, Aslan D. Virus-associated immune thrombocytopenic purpura in childhood. *Pediatr Hematol Oncol* 2002;**19**:433–7.

Felipe Francisco Tuon
Hermes Ryoiti Higashino
Antonio Carlos Nicodemo*
Department of Infectious and Parasitic Diseases, Medical School, University of São Paulo, São Paulo, SP, Brazil

Valdir Sabbaga Amato
Infectious and Parasitic Diseases Clinic, Hospital das Clínicas, Medical School, University of São Paulo, São Paulo, SP, Brazil

*Corresponding author.
Current address: Rua Barata Ribeiro, 414, Conjunto 104, CEP 01308-000, São Paulo, SP, Brazil.
Tel.: +55 11 32144251; fax: +55 11 32591318
E-mail address: ac_nicodemo@uol.com.br (A.C. Nicodemo)

Corresponding Editor: William Cameron, Ottawa, Canada

23 August 2007

doi:10.1016/j.ijid.2007.12.009

An unusual case of *Corynebacterium striatum* endocarditis and a review of the literature

An 83-year-old man was admitted with a 3-day history of fever and joint pain (mostly knee and shoulder). Over the previous three weeks he had been treated in the community with ciprofloxacin for a presumed recurrent urinary tract infection. He was known to have metastatic prostate cancer and had developed secondary hyperfibrinolysis syndrome with an intracerebral bleed from which he had made a good recovery.

On examination, he was afebrile. His pulse rate was 85 per minute and his blood pressure was 98/60 mmHg. An ejection systolic murmur was heard. His knees and shoulders were tender and hot to the touch. Respiratory and abdominal examinations were normal. Neurological examination revealed increased tone in the muscles.

Polymyalgia rheumatica, polyarthritis, rheumatoid arthritis, and metastatic effects of prostate cancer were considered in the differential diagnosis. Laboratory studies revealed elevated urea and creatinine (12.5 mmol/l and 148 μmol/l, respectively), high alkaline phosphatase (351 U/l), and a markedly elevated C-reactive protein (CRP) (565 mg/l). He had a mild leukocytosis (11.4×10^9/l) and a raised erythrocyte sedimentation rate (74 mm/h). Tests for rheumatoid factor and anti-neutrophil cytoplasmic antibody were negative. A chest roentgenogram showed clear lung fields, but extensive bony sclerosis was noted consistent with bony metastases.

Four separate sets of blood cultures taken at different times grew *Corynebacterium striatum*. In the absence of definitive laboratory guidelines for determining the antibiotic susceptibility of diphtheroids, we established the minimum inhibitory concentrations (MIC) for penicillin,

Table 1 Summarizing previously reported cases of *Corynebacterium striatum* endocarditis

Reference	Age	Sex	Associated illness	Valve	Intervention	Outcome
4	76	M	None	Aortic	Medical	Died
5	54	M	Hypertension	Aortic	Medical and surgical	Survived
6	73	M	Pacemaker	Tricuspid	Medical and surgical	Survived
7	24	M	Ventricular shunt	Pulmonary	Medical	Survived
8	68	M	Hypertension	Mitral	Medical	Survived
9	72	F	Prosthetic valve	Aortic	Medical	Died
10	62	F	Prosthetic valve	Aortic	Medical	Survived
11	50	M	Mycotic aneurysm	Aortic	Medical and surgical	Survived
2	61	F	Rheumatic fever	Mitral	Medical	Survived
2	72	F	Prosthetic valve	Mitral	Medical	Survived
3	46	F	Hemodialysis	Tricuspid	Medical	Survived
12	68	M	Prosthetic valve	Mitral	Medical	Survived
13	69	F	Endometrial cancer	Mitral	Medical and surgical	Survived
14	77	F	None	Mitral	Medical	Survived
15	62	M	Hypertension	Aortic	Medical and surgical	Survived

gentamicin, and vancomycin. These values were 1.5 mg/l, 0.032 mg/l, and 0.5 mg/l, respectively. A transthoracic echocardiogram revealed a mobile oscillating mass at the tip of the mitral valve leaflet. A diagnosis of infective endocarditis (IE) was made and therapy with vancomycin and rifampin was instituted.

As the patient showed no signs of improvement despite 5 days of treatment, therapy was switched to intravenous benzyl penicillin in combination with gentamicin in doses prescribed for IE. Combination therapy was used because the MIC of penicillin was considered to be on the higher side. Penicillin and gentamicin combination is known to be synergistic in the treatment of diphtheroid endocarditis irrespective of the MIC of penicillin provided the strains are gentamicin-susceptible.[1] However, the patient remained febrile on this combination although his CRP and total leukocyte count continued to fall. An abdominal CT scan in the third week of admission was reported as normal. A repeat echocardiogram at this stage failed to show any vegetation. Six sets of blood cultures (two while on vancomycin and four on penicillin) taken on different days after the initiation of treatment did not grow any pathogens. As there was a temporal relationship between the administration of penicillin and the fever spikes, β-lactam induced fever was considered and combination therapy was discontinued following which the patient defervesced. However, since the therapy for endocarditis was thought to be incomplete at this point, treatment was switched to intravenous daptomycin 6 mg/kg once daily. The MIC of daptomycin for this strain was found to be 0.064 mg/l. He remained apyrexial and his inflammatory markers continued to improve while on daptomycin. An additional blood culture set taken 7 days after daptomycin was started did not grow any bacteria. Daptomycin was given for a total of 17 days following which the patient was discharged. Although he appeared to recover from endocarditis based on his clinical status, repeat echocardiogram findings, and negative microbiology, he died of myocardial infarction 2 weeks following discharge. Emboli to the coronary arteries can complicate the course of IE but this is unlikely to have complicated the present case because of the long interval between the repeat negative echocardiogram and the occurrence of myocardial infarction.

A literature search in Medline revealed only 15 reports of *C. striatum* endocarditis (Table 1).[2–15] In our patient, multiple joint pain coupled with a history of prostate cancer pointed towards a primary rheumatologic condition or the effects of metastasis of the primary carcinoma. Prostate cancer is prone to metastasize in long bones. Repeated isolation of *C. striatum* in blood cultures and the subsequent echocardiogram finding clinched the diagnosis of IE.

IE is associated with immune complexes. Deposition of the antigen–antibody complexes can give rise to joint manifestations or vasculitis. Stoddart and colleagues reported two cases of endocarditis due to *C. striatum* that presented with symptoms suggestive of vasculitis and systemic sclerosis.[2] It is possible that because diphtheroids are often dismissed as contaminants even when isolated from sterile sites, the diagnosis is delayed, thereby allowing a full-blown immunological process to manifest in the absence of antibiotic treatment. By reducing the number of organisms, antibiotics cause a fall in the number of immune complexes.[16] Early and specific intervention in IE due to more common causes may often prevent the onset of severe immunologic manifestations.

Penicillin and vancomycin have been used for the treatment of diphtheroid endocarditis, but neither was found to be suitable in our patient. Daptomycin is a rapidly bactericidal lipopeptide antibiotic that is currently licensed in the UK for severe skin and soft tissue infections and has recently been licensed in the USA for endocarditis. A recent study found daptomycin to be useful for right-sided IE caused by *Staphylococcus aureus*.[17] Its long half-life allows once-daily administration. The combination of daptomycin and rifampin has been used in one previously reported case of *C. striatum* endocarditis.[3] Our patient seemed to respond to daptomycin monotherapy and completed a total course of 5 weeks of treatment that included vancomycin, penicillin, and daptomycin.

In summary, patients presenting with features suggestive of a rheumatologic condition should be thoroughly reviewed before blood culture isolates are dismissed as contaminants. In recalcitrant cases of diphtheroid endocarditis, daptomycin may be a useful alternative for patients who fail to respond to standard therapy. There is also an urgent need to establish

antibiotic susceptibility guidelines for diphtheroids that would help infectious diseases specialists to choose the most appropriate antibiotics.

This paper was presented in part at the Scottish Microbiology Association meeting, Dunblane, November 11, 2006.

Conflict of interest: No conflict of interest to declare.

References

1. Murray BE, Karchmer AW, Moellering Jr RC. Diphtheroid prosthetic valve endocarditis. A study of clinical features and infecting organisms. *Am J Med* 1980;**69**:838–48.
2. Stoddart B, Sandoe JA, Denton M. *Corynebacterium striatum* endocarditis masquerading as connective tissue disorders. *Rheumatology (Oxford)* 2005;**44**:557–8.
3. Shah M, Murillo JL. Successful treatment of *Corynebacterium striatum* endocarditis with daptomycin plus rifampin. *Ann Pharmacother* 2005;**39**:1741–4.
4. Markowitz SM, Coudron PE. Native valve endocarditis caused by an organism resembling *Corynebacterium striatum*. *J Clin Microbiol* 1990;**28**:8–10.
5. Rufael DW, Cohn SE. Native valve endocarditis due to *Corynebacterium striatum*: case report and review. *Clin Infect Dis* 1994;**19**:1054–61.
6. Melero-Bascones M, Munoz P, Rodriguez-Creixems M, Bouza E. *Corynebacterium striatum*: an undescribed agent of pacemaker-related endocarditis. *Clin Infect Dis* 1996;**22**: 576–7.
7. Tattevin P, Cremieux AC, Muller-Serieys C, Carbon C. Native valve endocarditis due to *Corynebacterium striatum*: first reported case of medical treatment alone. *Clin Infect Dis* 1996;**23**:1330–1.
8. Juurlink DN, Borczyk A, Simor AE. Native valve endocarditis due to *Corynebacterium striatum*. *Eur J Clin Microbiol Infect Dis* 1996;**15**:963–5.
9. de Arriba JJ, Blanch JJ, Mateos F, Martinez-Alfaro E, Solera J. *Corynebacterium striatum* first reported case of prosthetic valve endocarditis. *J Infect* 2002;**44**:193.
10. Houghton T, Kaye GC, Meigh RE. An unusual case of infective endocarditis. *Postgrad Med J* 2002;**78**:290–1.
11. Kocazeybek B, Ozder A, Kucukoglu S, Kucukates E, Yuksel H, Olga R. Report of a case with polymicrobial endocarditis related to multiresistant strains. *Chemotherapy* 2002;**48**: 316–9.
12. Mashavi M, Soifer E, Harpaz D, Beigel Y. First report of prosthetic mitral valve endocarditis due to *Corynebacterium striatum*: successful medical treatment. Case report and literature review. *J Infect* 2006;**52**:e139–41.
13. Tibrewala AV, Woods CJ, Pyrgos VJ, Ruiz ME. Native valve endocarditis caused by *C. striatum*. *Scand J Infect Dis* 2006;**38**:805–7.
14. Elshibly S, Xu J, Millar BC, Armstrong C, Moore JE. Molecular diagnosis of native mitral valve endocarditis due to *Corynebacterium striatum*. *Br J Biomed Sci* 2006;**63**:181–4.
15. Belmares J, Detterline S, Pak JB, Parada JP. Corynebacterium endocarditis species-specific risk factors and outcomes. *BMC Infect Dis* 2007;**7**:4.
16. Bayer AS, Theofilopoulos AN, Dixon FJ, Guze LB. Circulating immune complexes in experimental streptococcal endocarditis: a monitor of therapeutic efficacy. *J Infect Dis* 1979;**139**:1–8.
17. Fowler Jr VG, Boucher HW, Corey GR, Abrutyn E, Karchmer AW, Rupp ME, et al. Daptomycin versus standard therapy for bacteremia and endocarditis caused by *Staphylococcus aureus*. *N Engl J Med* 2006;**355**:653–65.

Yaqoob Bhat
Stuart Rochow
Department of Elderly Medicine, Woodend General Hospital, Aberdeen, UK

Abhijit M. Bal*
Ian M. Gould
Department of Medical Microbiology, Aberdeen Royal Infirmary, Foresterhill, Aberdeen AB25 2ZN, UK

*Corresponding author. Tel.: +44 1224 553819; fax: +44 1224 550632
E-mail address: a.bal@abdn.ac.uk (A.M. Bal)

Corresponding Editor: Craig Lee, Ottawa, Canada

17 August 2007

doi:10.1016/j.ijid.2007.12.010