

Unusual mechanism of myocardial infarction in prosthetic valve endocarditis

Fernando A Atik¹
Vanessa G Campos²
Claudio R da Cunha¹
Felipe Bezerra Martins de
Oliveira¹
Maria Estefânia Bosco
Otto²
Guilherme U Monte²

¹Department of Cardiovascular Surgery, Instituto de Cardiologia do Distrito Federal, Fundação Universitária de Cardiologia, Brasília, Distrito Federal, Brazil; ²Department of Cardiovascular Medicine, Instituto de Cardiologia do Distrito Federal, Fundação Universitária de Cardiologia, Brasília, Distrito Federal, Brazil

Abstract: A 46-year-old man with bicuspid aortic valve and severe calcific aortic stenosis was submitted to aortic valve replacement with a stented bioprosthesis. He developed *Staphylococcus epidermidis* prosthetic valve endocarditis a month later, presenting in the emergency room with acute myocardial infarction. The mechanism of myocardial ischemia was a large aortic root abscess causing left main extrinsic compression. He was urgently taken to the operating room, and an aortic root replacement with cryopreserved homograft was performed, associated with autologous pericardium patch closure of aortic to right atrium fistula and coronary artery bypass grafting of the left anterior descending. After a difficult postoperative period with multiple problems, he was eventually discharged home. At 36-month follow-up, he is asymptomatic with no recurrent infection, and the left main coronary artery is widely patent on control chest computed tomography.

Keywords: aortic root, endocarditis, aortic valve replacement, heart valve, allograft

Introduction

Although infrequent (1% to 4% in the first year), infective endocarditis may occur after aortic valve replacement.¹⁻³ When present, prosthetic valve endocarditis is a severe problem that is associated with considerable morbidity, especially related to peripheral embolism, perivalvular abscess, and sepsis.⁴⁻⁹

Acute coronary syndromes are uncommon in prosthetic valve endocarditis, with a prevalence of between 1% and 3%.¹⁰ The most likely mechanisms responsible for myocardial ischemia are the presence of preexisting coronary artery disease and coronary emboli from aortic vegetations. Other less frequent mechanisms have been described, such as obstruction of the coronary ostium due to large vegetation and severe aortic insufficiency.¹⁰⁻¹⁴ External coronary artery compression due to infective endocarditis is also a described mechanism, but is a rare occurrence with only few cases reported in medical literature.^{11,14-21}

We report a 46-year-old patient who presented with acute myocardial infarction caused by an aortic root abscess, which resulted in the external compression of the left main coronary artery.

Case description

A 46-year-old man presented at Instituto de Cardiologia do Distrito Federal, Brasília, Brazil with a history of dyspnea and fatigue exacerbated by a recent hospitalization related to pulmonary edema. Diagnosis was confirmed by echocardiography, which revealed the presence of a bicuspid aortic valve with mixed lesion, predominantly

Correspondence: Fernando A Atik
SQSW 102 Bl D Apto 508,
70670-204 Brasília, Distrito
Federal, Brazil
Tel +55 61 3222 5110
Email atikf@mac.com

stenotic. The left ventricle had moderate systolic dysfunction and severe concentric hypertrophy. Past medical history was consistent with hypertension. Surgery was indicated for symptomatic severe aortic stenosis. A preoperative coronary angiogram did not reveal any abnormalities. The patient was submitted to aortic valve replacement with a stented bioprosthesis. The operation and his postoperative recovery were uneventful, and the patient was discharged home on the sixth day after the procedure in good condition. He was prescribed the following medications to take at home: enalapril 5 mg twice daily, carvedilol 6.25 mg twice daily, furosemide 20 mg daily, and ferrous sulfate 300 mg daily (orally).

Approximately 1 month later, the patient returned, complaining of a high-grade fever and chills for a few days. He was admitted to the emergency department with acute onset chest pain, dyspnea, and vomiting. On physical exam, he was lethargic, febrile, pale, and hemodynamically unstable, with cold extremities and faint pulses. Admitting electrocardiogram revealed normal sinus rhythm, signs of left ventricle hypertrophy, and ST depression on posterior and lateral leads (Figure 1). Laboratory values on admission demonstrated remarkable elevation of cardiac enzymes (Table 1). The cardiologist on call interpreted the patient as having non-ST elevation acute coronary syndrome, and treated him with aspirin, clopidogrel, morphine, and heparin. An emergent coronary

Table 1 Admitting laboratory values

| Variable | Patient result |
|---|----------------|
| Hemoglobin (g/dL) | 12.5 |
| Hematocrit (%) | 38.9 |
| White blood cell count (cells/ μ L) | 16,100 |
| Platelet count (platelets/ μ L) | 145,000 |
| Glucose (mg/dL) | 131 |
| Urea (mg/dL) | 35 |
| Creatinine (mg/dL) | 1.1 |
| Sodium (mEq/L) | 134 |
| Troponin I (ng/mL) | 6.3 |
| CK-MB (ng/mL) | 5.8 |
| C-reactive protein (mg/dL) | 19.9 |
| Triglyceride (mg/dL) | 272 |
| HDL cholesterol (mg/dL) | 26 |
| LDL cholesterol (mg/dL) | 67.6 |

Abbreviations: CK-MB, creatinine kinase myocardial isoenzyme; HDL, high density lipoprotein; LDL, low density lipoprotein.

angiogram (Figure 2A) revealed a long, complex lesion on the left main coronary artery (80% lumen obstruction) with tapering contour, suggesting extrinsic compression. Due to ongoing myocardial ischemia and hemodynamic compromise, an intra-aortic balloon pump was inserted. The patient was further managed with intravenous fluids, inotropes, and mechanical ventilation. Blood cultures were drawn and identified as oxacillin-resistant *Staphylococcus epidermidis*. Before the blood cultures results were available, broad-spectrum anti-

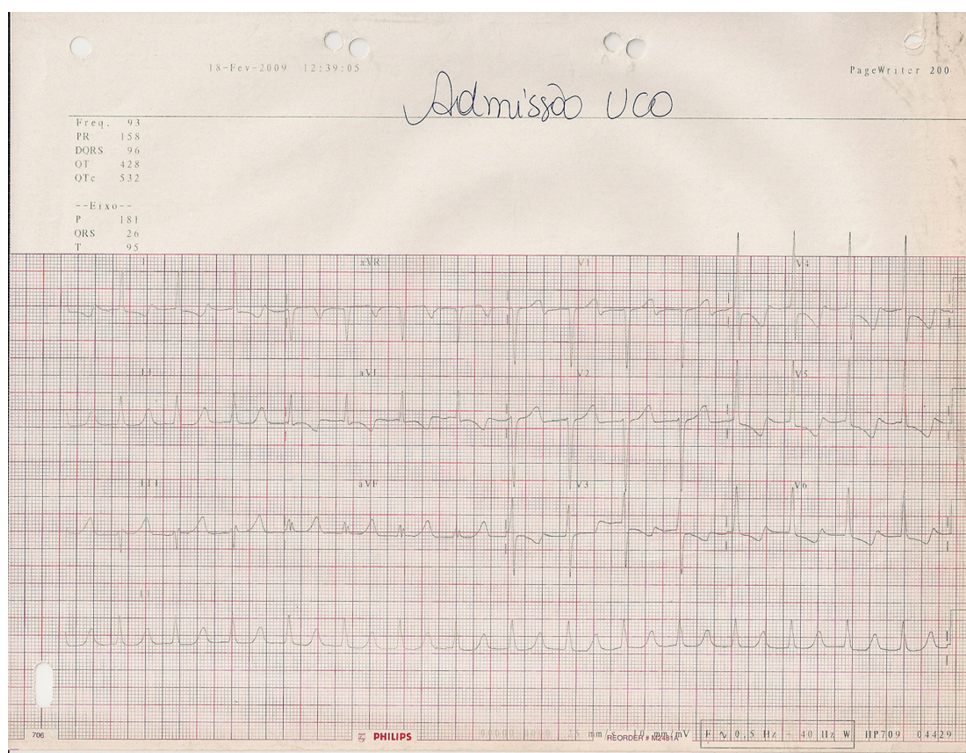


Figure 1 Admitting electrocardiogram.

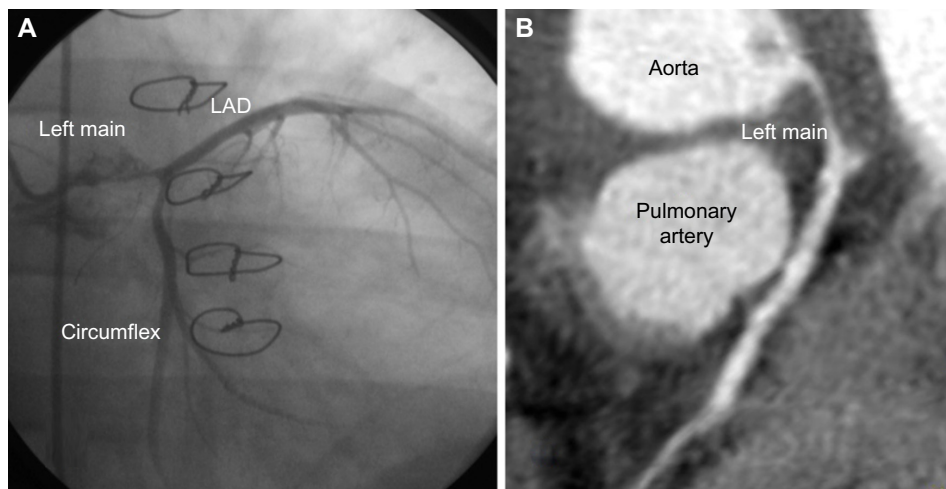


Figure 2 Coronary angiogram and chest computed tomography.

Notes: Coronary angiogram (A) in right anterior oblique showing a complex obstruction of the left main coronary artery. Chest computed tomography (B) showing a long obstruction of the left main coronary artery due to extrinsic compression.

Abbreviation: LAD, left anterior descending.

biotics (vancomycin, rifampicin, and imipenem) and also an anti-fungal drug (amphotericin B) were initiated empirically. Renal function was impaired. Transesophageal echocardiography showed vegetations attached to the prosthesis, aortic root abscess, and an aortic-to-right-atrium fistula. Left ventricle ejection fraction was 36%, and there were wall motion abnormalities on lateral and anterior walls. Chest computed tomography (Figure 2B) suggested that left main compression was due to aortic root abscess or hematoma.

Urgent reoperative aortic valve replacement was indicated. The operation was performed with the aid of hypothermic cardiopulmonary bypass with aortic and bicaval cannulation. Myocardial protection was achieved with cold blood cardioplegia delivered through the coronary ostium and coronary sinus. An oblique aortic arteriotomy revealed a large anterior aortic root abscess, which had invaded the left and right coronary cusps. The bioprosthesis was removed, and the abscess was evacuated; complete debridement of all infected material was also performed. Both coronary buttons were carefully mobilized and cleaned of all infected tissue. The aortic annulus was severely destroyed by the infection. Aortic root replacement with cryopreserved homograft was performed using interrupted monofilament sutures. The coronary buttons were directly re-implanted on the homograft with continuous sutures. The aortic-to-right atrium fistula was closed with an autologous pericardium patch. Additionally, a coronary artery bypass grafting with a reversed saphenous vein graft to left anterior descending artery was performed. Prolonged cardiopulmonary bypass and mediastinal bleeding were problems in the operating room, requiring vigorous

transfusion and delayed chest closure on the next day. Cultures drawn in the operating room were negative.

Postoperatively, the patient had acute renal failure requiring hemodialysis, sepsis, transient liver failure, prolonged mechanical ventilation, and complete atrioventricular block requiring permanent pacemaker. Antibiotic therapy, as previously mentioned, was initiated empirically with vancomycin, rifampicin, imipenem, and amphotericin

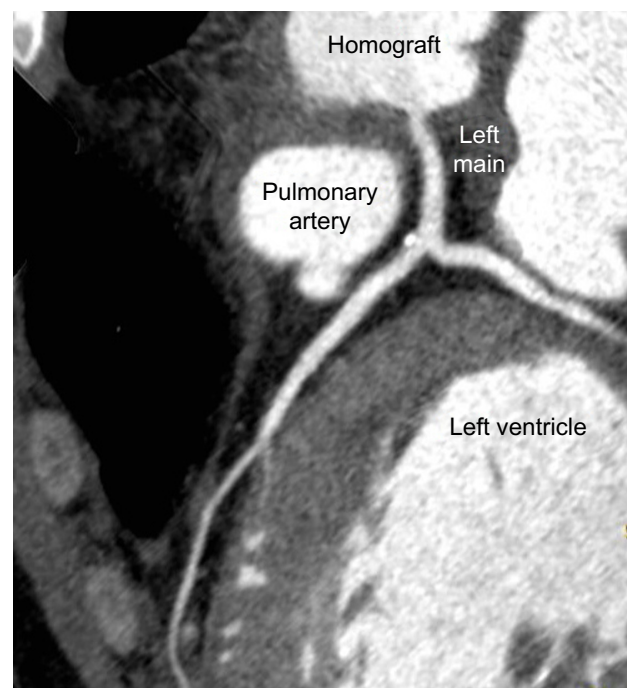


Figure 3 Late postoperative chest computed tomography revealed a patent left coronary artery after homograft root replacement.

B. Rifampicin was discontinued on the second day of use due to mild liver failure. Imipenem was used until the 14th postoperative day. Vancomycin was replaced by teicoplanin shortly after the operation due to acute renal failure, and it was used until the 30th postoperative day. Amphotericin B was used until the 34th postoperative day. Then, it was replaced by fluconazole, which was continued for a year thereafter. A month after the operation, the left ventricular function had improved (ejection fraction of 58%) with a mildly dilated ventricle and a normal aortic valve function.

The patient was discharged home 6 weeks after surgery in good condition, with no signs of active infection. At 36-month follow-up, the patient is currently in New York Heart Association functional class I with no recurrent infection, and the ventricular function is normal. The left main is widely patent and the saphenous vein graft is occluded on control chest computed tomography (Figure 3).

Discussion

The present case describes a rare complication of prosthetic valve endocarditis, an aortic root abscess causing external coronary artery compression and acute myocardial infarction. This mechanism of myocardial infarction has already been described.^{11,14–21} The most likely mechanisms responsible for myocardial infarction during infective endocarditis are preexisting coronary artery disease and coronary artery emboli from vegetations.

Prosthetic valve endocarditis is associated with elevated hospital mortality (approximately 40%)²² and morbidity dependent on the infecting pathogen, duration of illness prior to therapy, and underlying comorbidities.²³ Mansur et al²⁴ presented a total of 386 complications in 223 episodes of endocarditis (74% complication rate), being 100 occurrences of cardiac complications (severe heart failure, valve injury, pericarditis, acute myocardial infarction, conduction abnormalities, fistulous communication, perivalvular abscess, and others), 72 neurologic complications (cerebral embolism, mycotic aneurysms, meningitis, stroke, intracranial hemorrhage, cerebral abscess, and others), 46 septic complications (infection unresponsive to treatment, persistent fever, disseminated intravascular coagulation, and others), 27 renal complications (renal failure, nephrotic syndrome), 16 extracranial systemic arterial complications, 26 septic pulmonary embolisms, three splenic infarctions or abscesses, and some other complications associated with infective endocarditis.

Clinical presentation of acute coronary syndromes in patients with endocarditis is similar to those with coronary

artery disease. As systems attempt to meet stringent door-to-balloon initiatives, it becomes imperative that a detailed history and physical exam be performed in this narrow time window in order to avoid unnecessary tests and/or therapeutic regimens. For instance, the diagnostic work-up of a patient with a clinical history of high-grade fever and chills within a month after a straightforward aortic valve replacement with known absence of coronary artery disease includes, firstly, a transesophageal echocardiography.¹⁷ It has a sensitivity and a specificity for abscess detection of 87% and 95%, respectively.²⁵ Coronary angiogram in this particular patient was indicated based on a misdiagnosis of coronary artery disease. Although the angiographic appearance is typical (complex and long lesion that often disappears on diastole), coronary angiogram should be avoided because it may cause dislodgment of septic fragments, which fortunately did not occur in this particular case.

The isolation of the causative organism of prosthetic valve endocarditis is essential, and antibiotic therapy should be delayed pending the blood culture results in cases of patients who are hemodynamically stable with an indolent clinical course. However, patients presenting hemodynamic instability or acute disease should receive empiric broad-spectrum antibiotic therapy promptly.²⁶ Therapy should be subsequently adjusted according to the culture results. The American Heart Association (AHA) and the European Society of Cardiology (ESC) guidelines recommend that prosthetic valve endocarditis should be treated for at least 6 weeks with adequate bactericidal agent.

Presence of acute coronary syndrome in the setting of prosthetic valve endocarditis carries an elevated morbidity and mortality rates.²⁷ Although uncommon, external compression of coronary artery should be considered in the differential diagnosis of these patients, and adequate diagnostic work-up and treatment should be indicated.¹¹ Treatment with anti-platelet agents increased the risk of cerebral bleeding and certainly complicated the operative treatment in this patient.²⁸ Although the patient was a very high-risk surgical candidate, the urgent operation was justified due to patient's age and treatable heart problems, despite the presence of active infection, aortic root invasion, and abscess. Operative findings confirmed the severity of infection. The root abscess perforated the membranous septum, causing aortic to right atrium fistula, and there was a huge invasion of the left coronary cusp around the left main coronary artery, which caused its extrinsic compression and myocardial ischemia. The treatment of prosthetic endocarditis was standard: extensive debridement, patch closure of fistula, and aortic root replacement with cryopreserved homograft.²⁹

Coronary artery bypass to left anterior descending was also performed in order to guarantee an adequate myocardial blood flow shortly after the operation. The adequate coronary button re-implant and obstruction relief established good coronary perfusion, and the saphenous vein graft has occluded due to competitive flow.

Conclusion

We experienced a rare complication of prosthetic valve endocarditis: external coronary artery compression due to aortic root abscess. Although uncommon, this mechanism should be considered in the differential diagnosis of patients presenting acute coronary syndrome in the setting of prosthetic valve endocarditis, and adequate diagnostic work-up and treatment should be indicated.

Author contributions

All authors contributed toward data analysis, drafting and revising the paper and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Bayer AS. Infective endocarditis. *Clin Infect Dis*. 1993;17(3): 313–320.
2. Grover FL, Cohen DJ, Oprian C, Henderson WG, Sethi G, Hammermeister KE. Determinants of the occurrence of and survival from prosthetic valve endocarditis. Experience of the Veterans Affairs Cooperative Study on Valvular Heart Disease. *J Thorac Cardiovasc Surg*. 1994;108(2):207–214.
3. Millaire A, Van Belle E, de Groot P, Leroy O, Ducloux G. Obstruction of the left main coronary ostium due to an aortic vegetation: survival after early surgery. *Clin Infect Dis*. 1996;22(1):192–193.
4. Steckelberg JM, Murphy JG, Ballard D, et al. Emboli in infective endocarditis: the prognostic value of echocardiography. *Ann Intern Med*. 1991;114(8):635–640.
5. De Castro S, Magni G, Beni S, et al. Role of transthoracic and transesophageal echocardiography in predicting embolic events in patients with active infective endocarditis involving native cardiac valves. *Am J Cardiol*. 1997;80(8):1030–1034.
6. Snygg-Martin U, Gustafsson L, Rosengren L, et al. Cerebrovascular complications in patients with left-sided infective endocarditis are common: a prospective study using magnetic resonance imaging and neurochemical brain damage markers. *Clin Infect Dis*. 2008;47(1):23–30.
7. Arnett EN, Roberts WC. Valve ring abscess in active infective endocarditis. Frequency, location, and clues to clinical diagnosis from the study of 95 necropsy patients. *Circulation*. 1976;54(1):140–145.
8. Omari B, Shapiro S, Ginzton L, et al. Predictive risk factors for periannular extension of native valve endocarditis. Clinical and echocardiographic analyses. *Chest*. 1989;96(6):1273–1279.
9. Daniel WG, Mügge A, Martin RP, et al. Improvement in the diagnosis of abscesses associated with endocarditis by transesophageal echocardiography. *N Engl J Med*. 1991;324(12):795–800.
10. Attias D, Messika-Zeitoun D, Wolf M, Lepage L, Vahanian A. Acute coronary syndrome in aortic infective endocarditis. *Eur J Echocardiogr*. 2008;9(6):727–728.
11. Harinstein ME, Marroquin OC. External coronary artery compression due to prosthetic valve bacterial endocarditis. *Catheter Cardiovasc Interv*. 2014;83(3):E168–E170.
12. Manzano MC, Vilacosta I, San Román JA, et al. Síndrome coronario agudo en la endocarditis infecciosa. [Acute coronary syndrome in infective endocarditis]. *Rev Esp Cardiol*. 2007;60(1):24–31. Spanish.
13. Khan JA, Panwar Z, Mujtaba F, Shah K. Infective endocarditis: a rare cause of acute coronary syndrome. *J Coll Physicians Surg Pak*. 2012;22(4):248–249.
14. Cripps T, Guvendik L. Coronary artery compression caused by abscess formation in infective endocarditis. *Int J Cardiol*. 1987;14(1): 99–102.
15. Schlaifer JD, Martin TD, Hill JA, Kerensky RA. Coronary artery obstruction caused by perivalvular abscess in aortic valve endocarditis. *Am Heart J*. 1996;131(2):413–416.
16. Zoffoli G, Gherli T. Periaortic valve abscess presenting as unstable angina. *Circulation*. 2005;112(13):e240–e241.
17. Horton CJ Jr, Nanda NC, Nekkanti R, Mukhtar O, McGiffin D. Prosthetic aortic valve abscess producing total right coronary artery occlusion: diagnosis by transesophageal three-dimensional echocardiography. *Echocardiography*. 2002;19(5):395–398.
18. Dean JW, Kuo J, Wood AJ. Myocardial infarction due to coronary artery compression by aortic root abscess. *Int J Cardiol*. 1993;41(2): 165–167.
19. Cowan SW, Fiser SM, Albrecht M, Fifer M, Vlahakes GJ, Madsen JC. Management of coronary artery compression caused by recurrent aortic root abscess. *J Card Surg*. 2008;23(3):195–197.
20. Wickline CL, Goli VD, Buell JC. Coronary artery narrowing due to extrinsic compression by myocardial abscess. *Cathet Cardiovasc Diagn*. 1991;23(2):121–123.
21. Parashara DK, Jacobs LE, Kotler MN, et al. Angina caused by systolic compression of the left coronary artery as a result of pseudoaneurysm of the mitral-aortic intervalvular fibrosa. *Am Heart J*. 1995;129(2): 417–421.
22. Hoen B, Duval X. Infective endocarditis. *N Engl J Med*. 2013; 369(8):785.
23. Baddour LM, Wilson WR, Bayer AS, et al. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on rheumatic fever, endocarditis and Kawasaki disease. *Circulation*. 2005;111(23): e394–e434.
24. Mansur AJ, Grinberg M, da Luz PL, Bellotti G. The complications of infective endocarditis. A reappraisal in the 1980s. *Arch Intern Med*. 1992;152(12):2428–2432.
25. Daniel WG, Mügge A, Martin RP, et al. Improvement in the diagnosis of abscesses associated with endocarditis by transesophageal echocardiography. *N Engl J Med*. 1991;324(12):795–800.
26. Manzano MC, Vilacosta I, San Román JA, et al. Síndrome coronario agudo en la endocarditis infecciosa [Acute coronary syndrome in infective endocarditis]. *Rev Esp Cardiol*. 2007;60(1):24–31. Spanish.
27. Nishimura RA, Carabello BA, Faxon MD et al. ACC/AHA 2008 Guideline update on valvular heart disease: focused update on infective endocarditis: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol*. 2008;52(8):676–685.
28. Hunter AJ, Girard DE. Thrombolytics in infectious endocarditis associated myocardial infarction. *J Emerg Med*. 2001;21(4):401–406.
29. Lytle BW, Sabik JF, Blackstone EH, Svensson LG, Pettersson GB, Cosgrove DM 3rd. Reoperative cryopreserved root and ascending aorta replacement for acute aortic prosthetic valve endocarditis. *Ann Thorac Surg*. 2002;74(5):S1754–S1757; discussion S1792–S1799.

International Medical Case Reports Journal

Dovepress

Publish your work in this journal

The International Medical Case Reports Journal is an international, peer-reviewed open-access journal publishing original case reports from all medical specialties. Previously unpublished medical posters are also accepted relating to any area of clinical or preclinical science. Submissions should not normally exceed 2,000 words or

4 published pages including figures, diagrams and references. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit <http://www.dovepress.com/testimonials.php> to read real quotes from published authors.

Submit your manuscript here: <http://www.dovepress.com/international-medical-case-reports-journal-journal>