

Surgical Management of Diffuse Premature Coronary Artery Disease and Giant Coronary Artery Aneurysm in Quiescent Systemic Lupus Erythematosus: A Case Report and Review of the Literature

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Abstract

Case Report

Coronary artery aneurysm (CAA) is an extremely rare cardiovascular manifestation of systemic lupus erythematosus (SLE) and may coexist with premature diffuse coronary artery disease (CAD), resulting in significant morbidity and mortality. We report a young female patient with quiescent SLE who presented with acute coronary syndrome secondary to giant CAA and diffuse triple-vessel CAD. This case highlights the importance of early recognition and multidisciplinary management of this uncommon but potentially life-threatening complication. **Case Presentation:** A 38-year-old, non-smoking woman with a 4-year history of quiescent SLE, maintained on hydroxychloroquine, presented with sudden-onset chest discomfort and dyspnoea. Physical examination revealed no significant cardiovascular abnormalities or clinical evidence of an acute SLE flare. Troponin I was elevated, while electrocardiography demonstrated T-wave inversion and ST-segment depression in the anteroseptal and lateral leads (V1–V5), consistent with non-ST-elevation acute coronary syndrome. Computed tomography coronary angiography and invasive coronary angiography demonstrated extensive coronary calcification, diffuse triple-vessel CAD, and multiple CAAs, including a giant aneurysm involving the distal left main coronary artery at the polygon of confluence. There was also chronic total occlusion of the mid-right coronary artery and distal left anterior descending artery. Transthoracic echocardiography demonstrated preserved left ventricular systolic function (left ventricular ejection fraction, LVEF 55%) with no significant valvular abnormalities. Laboratory investigations, including complete blood count, renal function, liver function, urinalysis, complement levels (C3 and C4), and lipid profile, were within normal limits. Following preoperative intravenous methylprednisolone (MTP) pulse therapy, the patient underwent coronary artery bypass grafting (CABG), coronary endarterectomy, and plication of the proximal LAD giant coronary artery aneurysm. Postoperatively, medical therapy was optimised with antiplatelet therapy, warfarin, a beta-blocker, a statin, and continued lupus-directed treatment. She recovered uneventfully and was discharged on postoperative day 14 after achieving a therapeutic international normalised ratio (INR). **Conclusion:** This case highlights an exceptionally rare but serious cardiovascular complication of SLE, demonstrating its potential to cause premature diffuse CAD and giant CAA in a young woman despite clinically quiescent disease. Successful management required close collaboration between cardiologist, rheumatologist, and cardiothoracic surgeon. Surgical coronary revascularisation combined with aneurysm repair, optimisation of lupus therapy, minimisation of glucocorticoid exposure, and aggressive modification of cardiovascular risk factors resulted in a favourable early outcome.

Keywords: systemic lupus erythematosus; coronary artery aneurysm; coronary artery disease; acute coronary syndrome; coronary artery bypass grafting; coronary endarterectomy; case report.

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INTRODUCTION

Coronary artery aneurysm (CAA) are extremely rare in systemic lupus erythematosus (SLE), where they typically arise from accelerated atherosclerosis disease [1,2] and autoimmune vasculitis. Manzi *et al.*, showed that women with SLE were 50 times more likely to develop coronary artery disease (CAD) in the

Framingham study [3]. Management strategies vary, often involving a combination of immunosuppressive therapy, surgical revascularization, aggressive cardiovascular risk reduction and anticoagulation to prevent life-threatening thrombotic complications. However, there are limited data available worldwide for the best management of SLE patients with diffuse CAD

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and CAA. In this case report, we aim to share our experience on a successful coronary artery bypass grafting (CABG), coronary endarterectomy and aneurysm plication on a patient with diffuse premature CAD with giant CAA.

CASE REPORT

We report a 38-year-old, non-smoker woman with SLE who presented with acute chest discomfort at remote hospital. She has a strong family history (mother and aunt) of SLE and was diagnosed with SLE at the age of 34. She was initially presented with cutaneous manifestation of photosensitivity and painful palmar nodular rash back in February 2022. Further investigation revealed a positive ANA and Anti-dsDNA with low level of C3 C4 count and negative ENA. Investigations revealed no systemic organs involvement; hence, she was started only tablet hydroxychloroquine (400 mg daily) with no prior exposure to steroid. Her lupus was in quiescent with no prior history of acute SLE flare under regular rheumatology follow up.

In the 4th year of her illness, in early May 2026, she presented to emergency department with the complaint of sudden onset of left sided chest discomfort and difficulty in breathing at rest. On admission, her vital signs showed blood pressure 109/69 mmHg, heart rate of 88 bpm, temperature 36.6-degree Celsius, respiratory rate of 20 and oxygen saturation 99% under room air. Her body mass index (BMI) was 34.6 kg/m² and body surface area (BSA) 1.96 m². Physical examination revealed normal cardiovascular findings without murmurs, no signs of pulmonary congestion, no legs edema and no evidence of active lupus flare, such as alopecia, malar rash, arthritis or serositis.

Initial investigation showed elevated troponin I was raised 3464 ng/L (normal value < 15.7 ng/L) and electrocardiography revealed T inversion and ST depression at antero-septallateral leads V1-V5, consistent with acute coronary syndrome (ACS). A transthoracic echocardiography displayed a preserved left ventricular ejection fraction (LVEF) of 55% and normal cardiac valves and chambers size, with no pericardial effusion.

She underwent an urgent computed tomography coronary angiogram (CTCA) and coronary angiography (COROS), revealing high calcium burden Agatston's score 772 and diffuse triple vessel disease with multiple coronary aneurysms (Figure 1) respectively. The largest coronary aneurysm located at distal left main (LM) polygon of confluence (POC). The POC aneurysm has wide neck measuring about 16.5mm and size measuring 20mm x 18mm) containing mural thrombus within and wall calcification. It trifurcates in left anterior descending artery (LAD), ramus and left circumflex artery (LCX). There was stenosis of LM 40%; LAD mild ostial disease with aneurysm in mid-segment, distal LAD was small and occluded; LCX ostial eccentric 50% to

70% disease after aneurysm, mid-segment had another aneurysm; OM2 and OM3 had aneurysm at the end and had likely thromboembolic occlusion; RCA ostial 90% disease with mid-segment total occlusion, distal RCA small calibre with PLV and small PDA receiving collateral flow from LAD and LCX.

The routine haematological, renal and liver function tests were within normal range. Lipid profile were normal with low density lipoprotein (LDL) 1.0 mmol/L; high density lipoprotein (HDL) 1.62 mmol/L; low non-HDL cholesterol 1.3 mmol/L; low total cholesterol (TC) 2.9 mmol/L; and low triglyceride (TG) 0.70 mmol/L. Immunologic studies revealed low C3 0.85 g/L (0.90 – 1.80), low C4 0.15 g/L (0.15 – 0.45) and positive anti-nuclear antibody (ANA) titre 1:1280.

Due to the complexity of the disease, a multidisciplinary discussion between rheumatologist, cardiologist and cardiac surgeon was held. Discussion concluded that she requires an aggressive preoperative medical optimization, lupus control, preoperative methylprednisolone (MTP) pulse therapy then for early CABG. CABG instead of percutaneous coronary intervention (PCI) was preferred because of patient's young age and anatomical factors including diffuse CAD with giant CAA, heavily calcified coronary wall and presence of thrombus within the CAA. She was then discharged and scheduled an elective early instead of urgent CABG to avoid the fragility of fresh infarcted, oedematous myocardial tissue which carries a high surgical mortality. Other than tablet hydroxychloroquine (400mg daily), she was started on regular tablet aspirin (150mg daily), Plavix (75mg daily), rivaroxaban (20mg daily), atorvastatin (40mg daily), bisoprolol (2.5mg daily), perindopril (2mg daily), vastarel MR (35mg daily). Anticoagulant was started due to presence of thrombus in the large giant left main CAA at POC.

One-month post-ACS, the patient was electively admitted and received three doses of intravenous (IV) MTP (500mg daily) for preoperative pulse therapy. One-week post-MTP, she underwent CABG, endarterectomy over PDA and proximal LAD coronary aneurysm plication (Figure 4).

The operation was conducted via median sternotomy approach. Left internal mammary artery (LIMA) was harvested in standard fashion. However, LIMA was not used and clipped because of poor flow as a result of severe heavy calcification and atherosclerosis disease (Figure 2). Hence, all conduits with reverse saphenous vein graft (RSVG) were used for coronary revascularization. Standard systemic heparinization of was given with activated clotting time (ACT) of 818 seconds. Cardiopulmonary bypass (CPB) was established with total CPB time of 137 minutes. A Sorin S5 oxygenator was used. Core cooling supplemented with topical intrapericardial cooling were used to lower

patient's temperature to 30 degrees Celsius. The aorta was cross-clamped for 111 minutes and antegrade blood cardioplegia was infused through the aortic root. The heart was vented via aortic root.

Intraoperatively, upon pericardiectomy, there was minimal pericardial effusion but no signs of adhesion or pericarditis. The heart was normal in size with good biventricular contractility. Assessment of native coronary artery revealed diffuse, heavily calcified triple vessel CAD with multiple segments of coronary aneurysm ranging 10-20mm, largest giant CAA located at proximal LAD measuring 20 mm by 20 mm in dimensions. Complete coronary revascularization was achieved with three good quality RSVG conduits (RSVG-LAD 1.25mm; RSVG-OM3 1.25mm; RSVG-PDA 1.25mm). The PDA was heavily calcified required an endarterectomy (2cm length). The distal veno-coronary anastomoses were performed using running 7/0 Prolene, while as the proximal aorto-coronary anastomoses were performed using running Prolene 6/0. Intraoperative flow-meter assessment showed good venocoronary flow with low resistance. The distal portion of the giant CAA at proximal LAD was clipped to prevent distal thromboembolic event (Figure 3). The body of giant CAA was plicated using two Teflon

patches and three interrupted suture with Prolene 4/0 (Figure 4). Given the close proximity of the giant CAA to the left main polygon of confluence (POC), aneurysm plication was performed rather than resection to avoid the risk of injury to the left main coronary and its major branches, namely left anterior descending artery (LAD), left circumflex artery (LCx), and ramus intermedius.

Following release of aortic cross clamp, the heart resumed to beat in sinus rhythm. The heart chambers were then actively deaired through aortic root vent. When rewarming was completed, cardiopulmonary bypass was terminated with minimal inotropes. Two precautionary epicardial pacing wire were placed at right atrium and right ventricle respectively. Two drains were placed at pericardial space (24 Fr) and mediastinal space (34 Fr). The chest was closed with standard fashion. The patient was then transferred to cardiac intensive care unit (CICU) with satisfactory hemodynamic stability.

The patient was extubated on postoperative day one and made an excellent postoperative recovery. She was discharged on post operative day 14 after optimization of INR target with concomitant medical therapy of antiplatelet therapy, warfarin, a beta-blocker, a statin, and continued lupus-directed treatment.

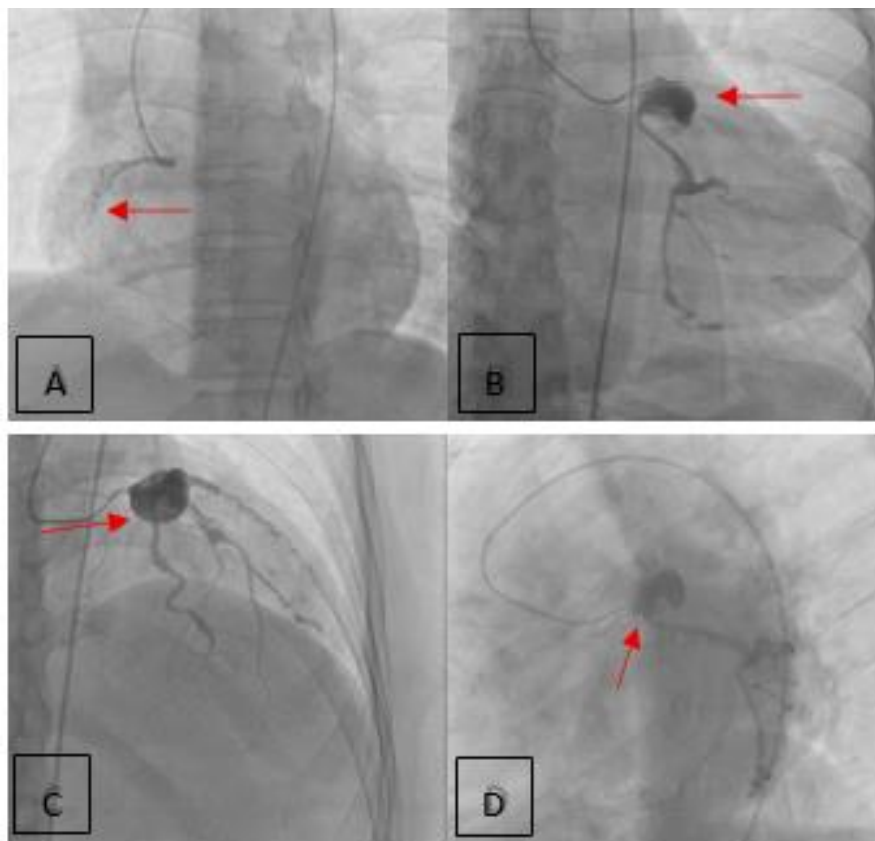


Figure 1: Coronary angiography reviewed (A) Right coronary artery (RCA) mid-segment total occlusion (red arrow); (B)(C)(D) Giant distal left main coronary aneurysm (red arrow) at polygon of confluence giving rise to left anterior descending artery (LAD), ramus and left circumflex artery (LCX)



Figure 2: Left internal mammary artery harvested. However, it was not used because of extensive atherosclerotic plaque and stenosis



Figure 3: Intraoperative view of giant CAA of proximal LAD and two haemoclip applied distal to the aneurysm to prevent distal thromboembolism



Figure 4: Proximal CAA was plicated with two Teflon patches to prevent rupture, or thrombus formation due to stagnant of blood

DISCUSSION

Coronary artery aneurysms (CAA) are an extremely rare cardiovascular complication of systemic lupus erythematosus (SLE) but carry significant morbidity and mortality if left unrecognized [4]. Although cardiac involvement in SLE commonly manifesting as pericarditis, myocarditis, or valvular disease, coronary abnormalities are generally categorized into three major categories: premature atherosclerosis, coronary arteritis, and the less frequently formation of coronary artery aneurysms [5]. In young patients, CAA are often diagnosed during investigations for acute coronary syndrome (ACS) or myocardial infarction (MI), and their clinical rarity necessitates a high index of suspicion to ensure early detection and timely intervention [4,5].

The formation of coronary aneurysms in SLE is multifactorial. Chronic systemic inflammatory accelerates premature atherosclerosis while immune-mediated coronary arteritis contributes directly to weakening of the arterial wall [5,6,7]. Histopathological examination of aneurysmal coronary walls has demonstrated significant damage to the media layer accompanied by inflammatory cell infiltration supporting an arteritic mechanism [8]. In addition antiphospholipid syndrome (APS) may coexist and further increase risk of thrombotic coronary occlusion [9]. The rapid development of aneurysms in young patients without traditional cardiovascular risk factors strongly implicates an active vasculitic process rather than isolated atherosclerosis [10].

Beyond their structural abnormalities, CAA serve as a critical nidus for thrombus formation, which significantly increases the risk of distal embolization and

subsequent myocardial infarction [6,11]. These aneurysms can cause myocardial ischemia even in the absence of significant atherosclerotic stenosis, as the stagnant blood flow within the dilated coronary segments predisposes patients to *in situ* thrombosis [11]. Clinicians must be vigilant for "missed" or atypical myocardial infarctions, as the clinical presentation may be masked by other SLE-related symptoms, such as pericarditis [11]. Rarely, rupture of a coronary aneurysm may produce a coronary steal phenomenon, resulting in acute myocardial infarction [12].

Despite these potentially life-threatening complications, SLE-associated CAAs remain uncommon, with only isolated case reports and small case series published to date [7,13,14,15,16]. Most reported CAA were clinically significant and complicated with stenosis, thrombosis, distal embolism, dissection or rupture ultimately leading to myocardial ischemia. Consequently, there are currently no evidence-based guidelines to direct the management of patients with concomitant SLE and CAA.

Management is therefore individualised and requires a multidisciplinary approach involving rheumatologists, cardiologists, and cardiothoracic surgeons. Medical therapy focuses on aggressive control of cardiovascular risk factors, optimisation of lipid profile and blood pressure, antiplatelet therapy, prevention of adverse left ventricular remodelling, and optimal suppression of lupus disease activity. High-dose corticosteroids are frequently administered in patients with active vasculitis, and additional immunosuppressive agents may be required for long-term disease control [5,9,10]. Because of the high risk of *in situ* thrombosis within aneurysmal coronary segments, anticoagulation is often combined with antiplatelet therapy to reduce the risk of distal embolisation [5,9,11].

The role of surgical intervention has been described in only a limited number of reports. Coronary artery bypass grafting (CABG) is generally reserved for patients with diffuse coronary artery disease, significant coronary stenosis, large or giant aneurysms, or when there is a substantial risk of rupture or thromboembolic complications [6,7]. Surgical treatment may also include aneurysm ligation, plication, or resection in conjunction with myocardial revascularisation, depending on aneurysm location and anatomical feasibility [6,7]. In our patient, the giant aneurysm was located at the left main coronary artery polygon of confluence. Owing to its close proximity to the left main coronary artery and its major branches, aneurysm plication was preferred over resection to minimise the risk of injury to the left anterior descending artery, left circumflex artery, and ramus intermedius while preserving coronary perfusion.

Rinaldi *et al.*, reported two patients with SLE who successfully underwent CABG using the left internal mammary artery (LIMA) as the conduit [17].

Histopathological examination of both grafts demonstrated no evidence of lupus-related arteritis, suggesting that internal mammary artery grafts may remain suitable conduits despite the underlying autoimmune disease. Similarly, Ura *et al.*, reported satisfactory long-term graft patency of 83.7%, concluding that CABG can be performed with acceptable morbidity and mortality in patients with SLE despite the complexity of the underlying disease [18].

Conversely, evidence supporting percutaneous coronary intervention (PCI) remains limited. Wilson *et al.*, described two patients with SLE and acute myocardial infarction who underwent percutaneous transluminal coronary angioplasty (PTCA); both experienced restenosis of the treated vessel, and one patient subsequently died [19]. These findings suggest that PCI may have limited durability in selected patients with SLE and diffuse coronary involvement, although further experience is required before definitive conclusions can be drawn.

Our case contributes to the limited literature by demonstrating the successful multidisciplinary management of a young woman with quiescent SLE who developed diffuse premature triple-vessel coronary artery disease and a giant coronary artery aneurysm. Complete surgical revascularisation combined with aneurysm plication, optimisation of lupus-directed therapy, and comprehensive secondary cardiovascular prevention resulted in a favourable early postoperative outcome.

CONCLUSION

This case highlights an exceptionally rare but serious cardiovascular complication of SLE, demonstrating its potential to cause premature diffuse CAD and giant CAA in a young woman despite clinically quiescent disease. Successful management required close collaboration between cardiology, rheumatology, and cardiothoracic surgery. Surgical revascularisation combined with aneurysm repair, optimisation of lupus therapy, minimisation of glucocorticoid exposure, and aggressive modification of cardiovascular risk factors resulted in a favourable early outcome.

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Abbreviations: SLE, systemic lupus erythematosus; CAA, coronary artery aneurysm; CAD, coronary artery disease; ACS, acute coronary syndrome; CTCA, computed tomography coronary angiography; CABG, coronary artery bypass grafting; ECG, electrocardiogram; LVEF, left ventricular ejection fraction; CBC, complete blood count; eGFR, estimated glomerular filtration rate; INR, international normalised ratio; MTP, methylprednisolone.

REFERENCES

1. Zeller CB, Appenzeller S. Cardiovascular disease in systemic lupus erythematosus: the role of traditional and lupus related risk factors. *Curr Cardiol Rev* 2018; 4(2): 116-22
2. Gaba, Vaishali, Anant Munde, Kalyan S. Munde, Khalil Shaikh, Anagh T.S, and Prasad Jain. 2025. "Diffuse Coronary Artery Disease in a Young Systemic Lupus Erythematosus Patient: A Case Report". *Cardiology and Angiology: An International Journal* 14 (3):72-77. <https://doi.org/10.9734/ca/2025/v14i3501>.
3. Manzi S, Meilahn EN, Rairie JE, Conte CG, Medsget TA Jr, Jansen-McWilliams L, *et al.*, Agespecific incidence rates of myocardial infarction and angina in women with systemic lupus erythematosus: comparison with the Framingham Study. *Am J Epidemiol* 1997; 145(5): 40815.
4. A. Hind, S. Saousan, E. G. Benouna, A. Drighil, L. Azzouzi, and R. Habbal, "Coronary Artery Aneurysm Revealed by an Acute Coronary Syndrome in a Patient with Systemic Lupus Erythematosus:A Case Report," vol. 1, no. 1, pp. 17–21, Aug. 2020, doi: 10.46619/JOH.2020.11005.
5. A. J, R. A, S. A, L. R, and P. A. M, "An unusual presentation of chest pain.," *Boletín de la Asociación Médica de Puerto Rico*, vol. 98, no. 4, p. 252, Oct. 2006.
6. H. Kurokawa *et al.*, "Myocardial infarction due to thrombi in coronary aneurysms in a young woman with systemic lupus erythematosus," *Journal of cardiology*, vol. 16, no. 1, pp. 249–258, Mar. 1986.
7. H. Suzuki *et al.*, "Giant Coronary Aneurysm in a Patient with Systemic Lupus Erythematosus," *Internal Medicine*, vol. 48, no. 16, pp. 1407–1412, Jan. 2009, doi: 10.2169/INTERNALMEDICINE.48.2079.
8. K. Hk *et al.*, "Coexistence of coronary aneurysms and total occlusion of coronary arteries in systemic lupus erythematosus.," *Clinical and Experimental Rheumatology*, vol. 16, no. 6, pp. 739–742, Nov. 1998.
9. T. P. Nobrega, E. Klodas, J. F. Breen, S. P. Liggett, S. T. Higano, and G. S. Reeder, "Giant coronary artery aneurysms and myocardial infarction in a patient with systemic lupus erythematosus.," *Catheterization and Cardiovascular Diagnosis*, vol. 39, no. 1, pp. 75–79, Sept. 1996, doi: 10.1002/(SICI)1097-0304(199609)39:1<75::AID-CCD16>3.0.CO;2-N.
10. Yao, Z., Long, Y., Zong, Z. *et al.*, Giant left anterior descending artery aneurysm in a patient with active systemic lupus erythematosus: a case report. *J Cardiothorac Surg* 16, 339 (2021). <https://doi.org/10.1186/s13019-021-01725-2>
11. E. A. Caracciolo, C. B. Marcu, A. Ghantous, T. J. Donohue, and G. Hutchinson, "Coronary vasculitis with acute myocardial infarction in a young woman with systemic lupus erythematosus.," *Jcr-journal of Clinical Rheumatology*, vol. 10, no. 2, pp. 66–68, Apr. 2004, doi: 10.1097/01.RHU.0000111317.80408.16.
12. K. Hirata *et al.*, "Coronary steal due to ruptured right coronary aneurysm causing myocardial infarction in a patient with systemic lupus erythematosus," *Cardiovascular diagnosis and therapy*, vol. 4, no. 4, pp. 333–336, Aug. 2014, doi: 10.3978/J.ISSN.22233652.2014.07.02.
13. Mulles AFC, Gonzales JRM, Uy MNAR, Fermin AMA, Osio-Salido E. Acute Myocardial Infarction Secondary to Triple Vessel Coronary Artery Disease in a 31-year-old Female with Systemic Lupus Erythematosus: Case Report and Review of Literature. *Acta Med Philipp*. 2026 Feb 13;60(3):88-94. doi: 10.47895/amp.vi0.12952. PMID: 41846702; PMCID: PMC12990927.
14. Matayoshi A, Dhond M, Laslett L; Multiple Coronary Aneurysms in a Case of Systemic Lupus Erythematosus; *CHEST*, 116, 1116-1118
15. Famularo G, Vallone A, Compagnucci M ...Tiny Bubbles, Coronary Aneurysms and Lupus, *The American Journal of Medicine*, 2014; 127, 933-935
16. Nagao T, Ito K, Tsuboi H, Ide Y. Rare complications of systemic lupus erythematosus. *Intern Med*. 2012; 51:3101-3102.
17. Rinaldi RG, Carballido J, Betancourt B, Sartori M, Almodovar EA. Coronary artery bypass grafting in patients with systemic lupus erythematosus. Report of 2 cases. *Tex Heart Inst J* 1995; 22(2): 185-8
18. Ura M, Sakata R, Nakayama Y, Ohtsuka Y, Saito T. Coronary artery bypass grafting in patients with systemic lupus erythematosus. *Eur J Cardiothorac Surg* 1999; 15(5): 697-701
19. Wilson VE, Eck SL, Bates ER. Evaluation and treatment of acute myocardial infarction complicating systemic lupus erythematosus. *Chest* 1992; 101:420-4.