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

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ABSTRACT

Cardiovascular (CV) disease is the leading cause of mortality in systemic lupus erythematosus (SLE). The risk of myocardial infarction (MI) in SLE is twice the incidence and ten years earlier in onset than in the general population. We present the first known case in the Philippines of acute MI from triple vessel coronary artery disease (CAD) in a young female patient with SLE. This aims to increase recognition and improve preventive strategies for this rare lupus complication.

A 31-year-old female with SLE for thirteen years, antiphopspholipid syndrome (APS) and controlled hypertension (HTN) presented with acute chest pain, diaphoresis, and dyspnea. She was a non-smoker with quiescent lupus and nephritis, maintained on low-dose aspirin, mycophenolate mofetil and hydroxychloroquine for the past four years. The physical examination revealed hypertension, bradycardia, normal heart sounds without murmurs, and no signs of lupus flare. The troponin level was elevated, and the electrocardiogram showed inferior wall ST-segment elevation myocardial infarction (STEMI). Coronary angiography revealed triple-vessel disease, with 80-90% stenosis of the left circumflex artery, and total occlusion of the left anterior descending and right coronary artery. There were segmental wall motion abnormalities and a low ejection fraction of 44% on echocardiography. The complete blood count, urinalysis, and serum C3 were within normal range. The anti-dsDNA was low and lipid levels were abnormal. The patient refused coronary artery bypass grafting (CABG). Medical management consisting of anti-platelets, beta-blockers, statin, and warfarin was maximized. The patient completed one year of follow-up without any lupus flares or cardiovascular events.

This case illustrates the complex interaction of disease-related and traditional cardiovascular risk factors leading to premature coronary artery disease in a young female with SLE. The case demonstrates favorable one-year outcomes after optimized post-MI medical management. Aside from optimized lupus control and reduced glucocorticoid use, proactive screening and aggressive management of modifiable CV risk factors and antiphospholipid antibodies (aPL), are necessary.

Keywords: systemic lupus erythematosus, coronary artery disease, myocardial infarction, antiphospholipid syndrome, case report

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INTRODUCTION

Advanced treatment modalities for systemic lupus erythematosus (SLE) have improved its overall prognosis. However, cardiovascular complications remain a substantial concern. Cardiovascular disease (CVD) is the leading cause of mortality, with 30% of related deaths from coronary artery disease (CAD).[1-3] The reported incidence of acute myocardial infarction (MI) in SLE is 9.6 events/1000 person-years. MI occurs ten years earlier than the general population, with an average age of 49 to 55 years.[4,5] Premenopausal women face a 50-fold increased MI risk compared to those without lupus.[6]

The accelerated development of CAD in SLE results from the interaction of disease-specific factors, such as chronic inflammation, antiphospholipid antibodies, glucocorticoid use, and traditional risk factors like hypertension (HTN) and dyslipidemia.[7] Severe manifestations, such as triple vessel disease in young patients, are uncommon, with only five reported cases in the literature. We present a case of acute MI secondary to triple vessel CAD in a young female with SLE in low disease activity. Her case shows the complex interaction between disease-related and traditional cardiovascular risks in SLE. The literature review provides updates on guidelines for CV risk assessment and treatment strategies. To our knowledge, this is the first reported case in the Philippines of triple vessel CAD in this patient population.

CASE PRESENTATION

The patient is a 31-year-old female with SLE who presented with acute chest pain at the Philippine General Hospital. She was diagnosed with SLE at the age of 16, following symptoms of oral ulcers, malar rash, edema, HTN, and frothy urine. The ANA test was positive and a kidney biopsy confirmed lupus nephritis ISN/RPS Class IV. Her initial treatment consisted of monthly cyclophosphamide using the National Institutes of Health (NIH) protocol, followed by long-term maintenance therapy with mycophenolate mofetil.

In the 6th and 7th years of her illness, she experienced two consecutive abortions at 26 and 22 weeks, respectively, along with positive lupus anticoagulant tests (prolonged Silica clotting time and activated partial thromboplastin time). A diagnosis of probable antiphospholipid syndrome (APS) was maintained, since repeat antibody tests after 12 weeks to fulfill the criteria for definite APS were not obtained. She had a family history of hypertension, but had never smoked. Her outpatient medications were hydroxychloroquine (200 mg daily) and mycophenolate mofetil (1 g daily) for the management of SLE; aspirin (80 mg daily) for APS; enalapril 10 mg daily, carvedilol (25 mg daily), and amlodipine (10 mg daily) for hypertension. Prednisone was successfully tapered and discontinued in the 11th year of illness. HTN was controlled, and lupus low disease activity was maintained for four years before her current hospitalization for chest pain.

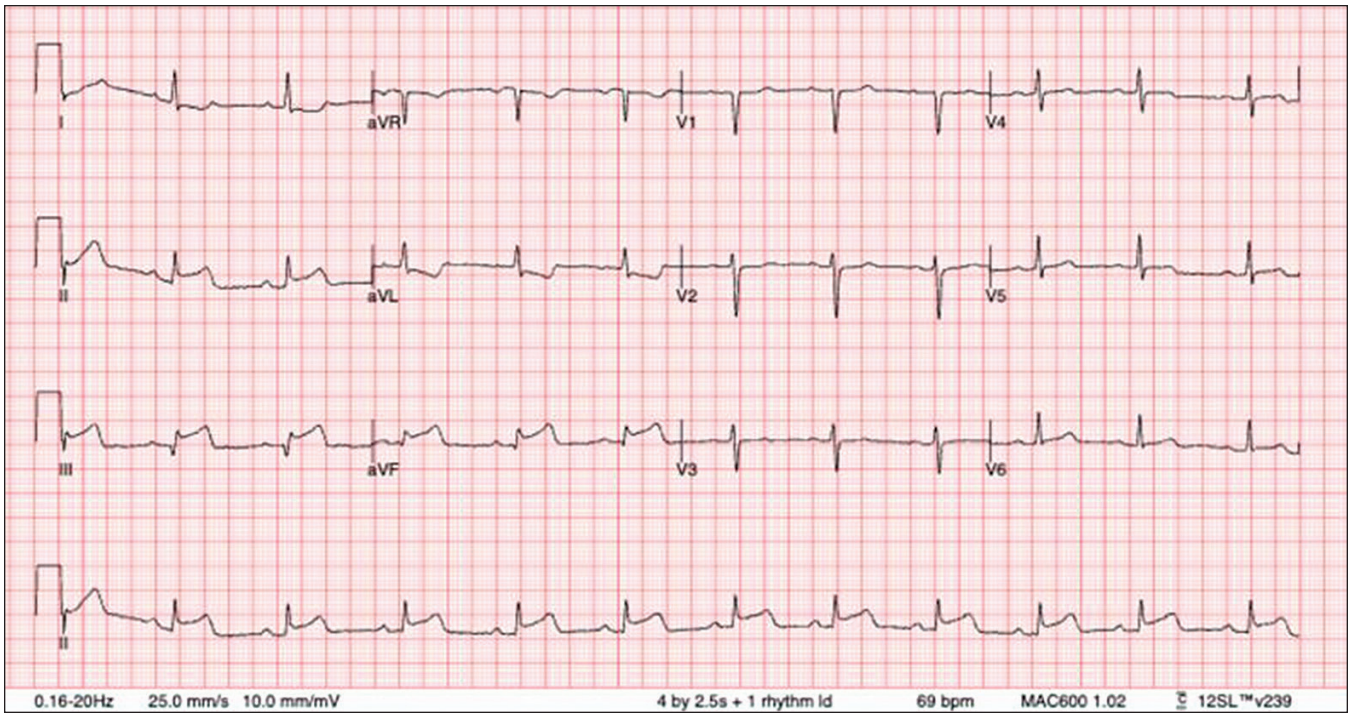

Figure 1. Electrocardiogram showing ST segment elevation in leads II, III, AVF, consistent with inferior wall STEMI.

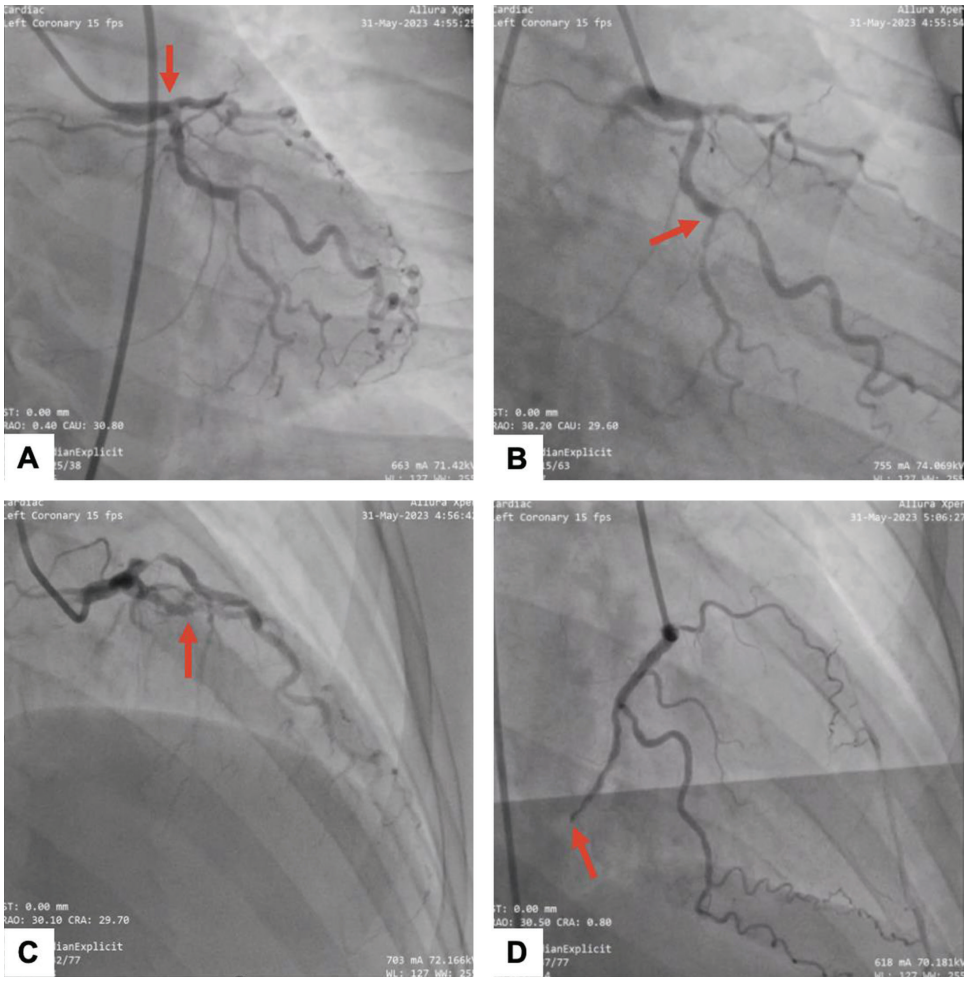

Figure 2. **(A)** 20-30% stenosis *(red arrow)* in left main coronary artery (LMCA), **(B)** 80-90% focal stenosis *(red arrow)* of the left circumflex artery (LCx), **(C)** total occlusion *(red arrow)* of the left anterior descending artery (LAD), and **(D)** total occlusion *(red arrow)* of the right coronary artery (RCA).

She was admitted to the emergency room after experiencing sudden onset substernal chest pain, diaphoresis, and dyspnea. Her vital signs on admission showed a blood pressure of 150/100 mmHg, heart rate of 57 bpm, respiratory rate of 21, and a temperature of 36.3 C. Her body mass index was 21.9 kg/m^2. Physical examination revealed normal heart sounds without murmurs, no signs of pulmonary congestion, and no evidence of active lupus flare, such as rash, alopecia, arthritis, or serositis.

Initial investigations revealed an elevated troponin I level [3744 ng/L, normal value (NV) <9 ng/L] and an electrocardiogram showing ST-segment elevation in the inferior leads (II, III, aVF), consistent with an acute inferior wall ST-segment elevation myocardial infarction (STEMI) (Figure 1). Immediate management included the administration of aspirin (320 mg), clopidogrel (300 mg), enoxaparin (60 mg), and atorvastatin (80 mg). She underwent an emergency angiography revealing triple vessel coronary artery disease. There was a 20-30% stenosis in the left main coronary artery (LMCA), 80-90% stenosis of the left circumflex artery (LCx), and total occlusion of the left anterior descending artery (LAD) and right coronary artery (RCA) (Figure 2).

The complete blood count, transaminases, and serum electrolytes, were all within normal ranges. She had trace proteinuria and renal insufficiency (serum creatinine 102 umol/L, NV 46 to 92 mmol/L with estimated glomerular filtration rate of 63 ml/min). She had low high-density lipoprotein (HDL) of 39.38 mg/dL (NV 40-60 mg/dL), elevated low-density lipoprotein (LDL) of 176.33 mg/dL (NV 100 -127 mg/dL), total cholesterol of 235.38 mg/dL (NV <200 mg/dL), and triglycerides of 100.8 mg/dL (NV <150 mg/dL). Her baseline coagulation showed a prolonged prothrombin time (PT) of 15.1 seconds with 76% activity and international normalized ratio (INR) of 1.21. Immunologic studies revealed normal C3 at 1.047g/L (NV

0.811-1.570 g/L), and low anti-dsDNA at 11.9 IU/mL (NV <10 IU/mL negative).

Chest x-ray showed left-sided predominant cardiomegaly. Echocardiography displayed an ejection fraction (EF) of 44%, concentric remodeling and segmental wall motion abnormalities of the left ventricle, with mildly depressed overall systolic function. Cardiac valves and other ventricles were structurally normal, with no pericardial effusion.

Due to multivessel involvement, coronary artery bypass grafting (CABG) was recommended. However, the patient declined surgery after expressing a clear understanding of its benefits, citing personal concerns, and opting instead for medical management. She received enoxaparin (0.6 mg twice a day), aspirin (80 mg daily), clopidogrel (75 mg daily), carvedilol (12.5 mg twice a day), sacubitril/valsartan (50 mg twice a day), atorvastatin (80 mg daily), hydroxychloroquine (200 mg daily) and mycophenolate mofetil (1 g daily). Enoxaparin was bridged to warfarin (2.5mg daily) prior to discharge.

Subsequently, she received follow-up care at the rheumatology and cardiology outpatient clinics, where she was closely monitored. This included clinical risk assessment, regular blood pressure, lipid profile, and coagulation monitoring, and medication adjustment to meet therapeutic targets. After one year of observation and adherence to therapy, she had no recurrence of angina, with good functional capacity, controlled HTN and lupus in low disease activity state.

DISCUSSION

Cardiovascular disease is a well-recognized complication of SLE; however, acute myocardial infarction with triple vessel disease is uncommon in young patients. This case highlights the need for awareness of premature and severe coronary artery disease that may occur even among young females with SLE.

Both traditional and disease-specific risk factors play critical roles in the premature development of coronary artery disease in SLE. The role of traditional risk factors, including HTN and dyslipidemia, is well-established but fails to fully explain the excess cardiovascular risk.[7,8] This suggests disease-related mechanisms contributing significantly to cardiovascular pathology.

Immune-mediated inflammation is the primary mechanism of accelerated atherosclerosis, with dysregulated innate and adaptive immune responses, microvascular damage, and endothelial dysfunction playing key roles in plaque formation. Impaired clearance of apoptotic cells contributes to sustained inflammation, creating a cycle that exacerbates atherosclerosis progression.[9,10] The presence of antiphospholipid antibodies (aPL) also increases CAD risk by inducing a prothrombotic state through direct effects on endothelial cells, platelets, and the coagulation pathway.[11,12]

Other disease-related factors, including glucocorticoid use and lupus nephritis, also play contributory roles. Proteinuria in lupus nephritis exacerbates atherogenesis by promoting dyslipidemia and endothelial dysfunction. These interrelated mechanisms demonstrate how SLE increases CAD risk through a dual pathway of inflammation-driven atherosclerosis and thrombosis.

In our patient, a history of lupus nephritis, history of prolonged corticosteroid use, hypertension, and presence of aPL created a synergistic risk profile leading to severe CAD. While she did not fully satisfy the Sapporo criteria for definite APS due to the absence of repeat confirmatory testing, the presence of recurrent obstetric morbidity, positive lupus anticoagulant, and underlying SLE warranted presumptive treatment for probable APS with low- dose aspirin. Unfortunately, this lapse in diagnostic follow-through may have limited the opportunity for timely risk stratification and preventive management.

Triple vessel disease is a severe form of CAD, defined by significant stenosis of at least 50% in the three major epicardial coronary arteries: the left anterior descending artery, the circumflex artery, and the right coronary artery.[13] Compared to single-vessel disease, triple vessel disease carries a higher mortality rate and a significantly reduced 12-year survival rate of 50%, in contrast to 74% for single-vessel disease.[14,15] Its prevalence in the general population varies but is observed in 40 to 50% of patients with ST-segment elevation myocardial infarction.[16] While CAD occurs in 6 to 10% of patients with SLE, the prevalence of triple vessel disease in this population remains unknown.[17] Likewise, its occurrence in young patients with SLE has been documented only in case reports. In the Framingham cohort, myocardial infarction was observed in all age groups with SLE but none was documented in women below 34 years old.[12] Similarly, Korkmaz reviewed myocardial infarction occurrences in patients with SLE younger than 35 years; however, none of the 50 cases demonstrated significant triple vessel disease as per the defined criteria.[18]

Given the limited data among young patients, we conducted a literature search to identify case reports, case series, and reviews of patients with SLE under 35 years old presenting with acute myocardial infarction and triple vessel CAD. We searched PubMed and Google Scholar using the terms “systemic lupus erythematosus”, “triple vessel coronary artery disease” or “three vessel coronary artery disease”, and “myocardial infarction”. No time frame limit was applied to capture all reported cases. In addition, reference lists of the retrieved studies were screened manually for relevant articles. A total of 98 records were identified. After removing duplicates and excluding abstracts, posters and studies without clear documentation of coronary artery disease, five case reports were included in the final review. Inclusion criteria were: (1) confirmed diagnosis of SLE, (2) age below 35 years, (3) clinical presentation of myocardial infarction, and (4) documented evidence of triple vessel coronary artery disease based on coronary angiogram or autopsy. The summary of the reported cases is shown in Table 1.

Table 1. Reported Cases of Myocardial Infarction and Triple Vessel Coronary Artery Disease in Female Patients with SLE below 35 Years Old

Authors (Year published)	Age in years	Age at diagnosis of SLE	Coronary Pathology	SLE activity	Risk factors	Therapy	Outcome
Tsakraklides et al.[19] (1974)	29	16	LAD, RCA + LCX athero- sclerosis, thrombosis [a]	NR	HTN, nephrotic syndrome, CS use	None	Died
Homcy et al.[20] (1982)	30	27	LAD and RCA occlusion, LCX stenosis [a]	Nephritis, arthritis, pleuro-pericarditis	HTN, CS use	NR	Died after five years
Rinaldi et al. [21] (1995)	26	18	Critical stenosis LAD, LCX, RCA	NR	NR	CABG	Alive
Meyringer et al.[22] (2005)	17	6	Occlusion at LCX and RCA, stenosis LCA	No SLE activity	CKD from lupus nephritis, HTN, dyslipidemia	PTCA	Alive
Zafar et al.[23] (2019)	32	19	Critical stenosis at LAD, ramus intermedius and RCA, moderate stenosis at LMCA, LCX	NR	HTN, APS	CABG	Alive
Present case	31	16	LCX focal stenosis, total occlusion at LAD and RCA	No SLE activity	HTN, lupus nephritis, APS, CS use	Medical only	Alive

LAD – left anterior descending artery, RCA – right coronary artery, LCX – left circumflex artery, LCA – left coronary artery, LMCA – left main coronary artery, NR – not reported, HTN – hypertension, CKD – chronic kidney disease, CABG – coronary artery bypass grafting, PTCA – percutaneous transluminal coronary angioplasty, APS – antiphospholipid antibody syndrome, CS – corticosteroids, [a] – autopsy finding

All patients were females, with ages ranging from 17 to 32 years old. The average disease duration of SLE prior to the myocardial infarction is 9.6 ± 4.2 years, ranging from 3 to 13 years. Similar to our patient, most cases had both disease-related and traditional cardiovascular risk factors, such as APS, hypertension, chronic kidney disease, and prolonged corticosteroid use. A past or current active lupus nephritis was seen in our patient and in three of the cases.

Antiplatelets, anticoagulants, and statin therapy were standard management for CAD in the majority of the cases. Clinical outcomes varied: three patients underwent interventional procedures and survived, while Tsakraklides' case resulted in death within one day of symptom and no intervention was performed. Similarly, Homcy's patient died of renal failure five years after the myocardial infarction, although details of therapeutic management were not provided. Notably, revascularization procedures showed improved survival as seen in these cases. While revascularization remains the recommended treatment for myocardial infarction from triple vessel disease, this case is distinct among previous reports in demonstrating a favorable short-term outcome with medical therapy.

CABG is the recommended treatment for three vessel disease. Evidence suggests that CABG for triple vessel disease improves survival compared to medical therapy or percutaneous coronary intervention (PCI).[24,25] Among patients with connective tissue diseases, including SLE, CABG also resulted in good outcomes, although there may be a risk of repeat revascularization.[26,27] Despite these benefits, our patient declined surgical intervention and opted for optimized medical therapy including long-term anticoagulation in view of her prothrombotic state from APS. The potential consequences of non-revascularization were clearly discussed with the patient, who made an informed decision to pursue conservative management.

Outcomes from medical management alone in young SLE patients with myocardial infarction remain underreported, and revascularization continues to be the standard of care regardless of underlying disease. However, in a cohort study of 29 APS patients with acute MI, 62% were managed conservatively. Over four years, four deaths occurred regardless of treatment, with none attributed to coronary events.[28] Our patient, who was observed for one year without cardiovascular events or lupus flares, adds to the body of clinical experience on the use of medical management in selected SLE patients with APS. Her outcome may reflect the benefit of strict adherence to pharmacologic treatment, long-term anticoagulation, and effective control of lupus activity in mitigating post-MI complications.

In 2021, the European Alliance of Associations for Rheumatology (EULAR) updated the recommendations for cardiovascular risk management in rheumatic and muscular diseases.[29] Eight recommendations were specific for managing cardiovascular risk in SLE and APS. The recommendations emphasize thorough assessment of traditional risk factors and disease-related risk factors to guide risk modification. With regard to disease-related factors, the guidelines recommend achieving low disease activity and using the lowest possible glucocorticoid dose to mitigate cardiovascular risk. They also recommended preventive strategies for blood pressure control and treatment of hyperlipidemia that follow that of the general population. In our patient, many of these recommendations were followed: blood pressure was managed with anti-hypertensives, low-

dose aspirin was given for APS, and lupus was in low disease activity with hydroxychloroquine and mycophenolate mofetil in the last four years. However, lipid profile monitoring was not routinely done, dyslipidemia was only recognized during the hospitalization, and she had not been on lipid-lowering therapy—a limitation in guideline adherence that may have contributed to the event. Consistent application of these recommendations in long-term management is therefore necessary in routine clinical practice.

Challenges persist in implementing these recommendations. One barrier to comprehensive screening and assessment of risk factors is the lack of validation for many CV risk prediction models for patients with SLE. Specific screening strategies in the younger age group are also lacking. Ongoing and future studies are essential for the validation of these strategies. Nevertheless, clinicians should adopt a proactive approach, maintaining a low threshold for initiating cardiac evaluations for patients with SLE.

CONCLUSION

This report presents a case of acute myocardial infarction due to triple vessel CAD in a 31-year-old female with SLE diagnosed at age 16. Despite quiescent nephritis and controlled HTN, she developed premature CAD, illustrating the complex interplay of disease-related and traditional cardiovascular risk factors in SLE. Her favorable one-year outcome adds to the body of clinical experience of optimized medical management in selected patients with SLE and APS. Current recommendations for mitigation of CV risk in persons with rheumatic diseases are similar with those for the general population. However, comprehensive CV risk assessment, including guideline-based aPL testing and routine lipid monitoring, must be practiced even in young patients with clinically stable disease. Likewise, clinicians should prioritize control of inflammatory disease activity and modifiable risk factors while minimizing glucocorticoids exposure, to reduce cardiovascular complications in SLE.

Informed Consent

Written consent was obtained prior to writing this case report.

Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

All authors declared no conflicts of interest.

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