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CASE REPORT

ACUTE ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION IN A YOUNG PATIENT WITH IGA NEPHROPATHY: A RARE CASE REPORT

Asraf Hussain[1,*], Shyam Raj Regmi[1], Bishnu Mani Dhital[1], Shovit Thapa[1], Tirth Dhungana[2], Saroj Shrestha[2]
[1]Department of Cardiology, Chitwan Medical College, Bharatpur Nepal
[2]Intern, Chitwan Medical College, Bharatpur Nepal

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***Correspondence to:** Asraf Hussain, Department of Cardiology, Chitwan Medical College, Bharatpur Nepal.
Email: asrafjeevanjyoti2060@gmail.com

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ABSTRACT

Cardiovascular events are less commonly described in glomerular disease. However, thromboembolic events are quite commonly reported in glomerular disease with nephrotic range proteinuria. Here, we report a case of a 29 years old male with IgA nephropathy present with central chest pain radiating to the jaw. He was under mycophenolate, prednisolone and losartan with remission of proteinuria for two months. ECG showed extensive ST segment elevation in V1-V6 leads. His cardiac troponins were elevated. Coronary angiography showed complete thrombotic occlusion of mid left anterior descending artery which was successfully recanalized after primary percutaneous intervention. Though less commonly reported in literature, such incidence of coronary events in IgA nephropathy can rarely present with acute myocardial infarction.

INTRODUCTION

Acute myocardial infarction is the ischemic necrosis of myocardium occurring after obstruction of the coronary artery. Atherosclerotic plaque changes and platelet activation are the major initial events for the pathogenesis of myocardial infarction. Hypercoagulable state in glomerular disease often tilts the hemodynamic balance towards thrombus formation.[1] Such thromboembolic phenomena are less common in the arterial side, coronary thrombosis is even rarely reported. This is the case of biopsy proven IgA nephropathy without known medical risk factors, later after two months presented with acute myocardial infarction.

CASE REPORT

A 29-year-old male patient presented to the Department of emergency medicine with complaints of central chest pain radiating to the jaw and left arm. He was diagnosed with IgA nephropathy two months ago and was under losartan, mycophenolate, and prednisolone therapy. He was a non-smoker, non-alcoholic without any history of illicit drug abuse. He had insignificant medical history in the past. On initial assessment, he looked anxious with pulse rate 120 beats per minute, blood pressure 140/100 mmHg, respiration rate 28 breath per min with saturation 98% under room air. He was tachypnoeic and tachycardic with elevated blood pressure at presentation while other systemic examination findings were within normal limits.

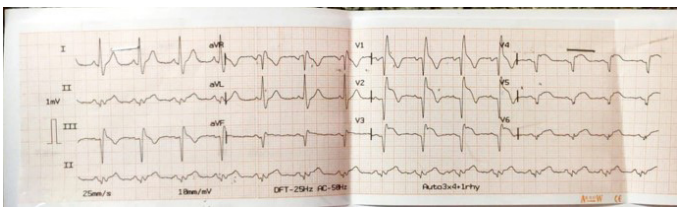

Figure 1: ECG showing ST-segment elevation in V1-V6 leads

On arrival, immediately ECG was done that showed ST segment elevation on V2-V6 leads (Fig 1). Quantitative cardiac troponin level (>50 ng/ml) was significantly elevated. Laboratory work up showed Hb (12.8 gm%), WBC count (20890 per/mm3), platelet count (302000 per/mm3). His recent coagulation profile was benign. Renal function tests were within normal limits with no proteinuria at presentation. Lipid profile showed minimal derangement with triglyceride (173 mg/dl), LDL cholesterol (73 mg/dl) HDL cholesterol (36 mg/dl). Coronary angiography

was performed that showed complete thrombotic occlusion (100%) of mid left anterior descending artery (Fig 2). Primary percutaneous intervention was done successfully to establish circulation distal to the occlusion. Echocardiography showed 40% ejection fraction with moderate systolic dysfunction. Patient improved symptomatically however on the following day creatinine level increased from (0.9 mg/dl) to (1.59 mg/dl). Other laboratory reports were benign. On the fourth day renal function improved and the patient was discharged with dual antiplatelets, beta blocker, ARB and statins therapy.

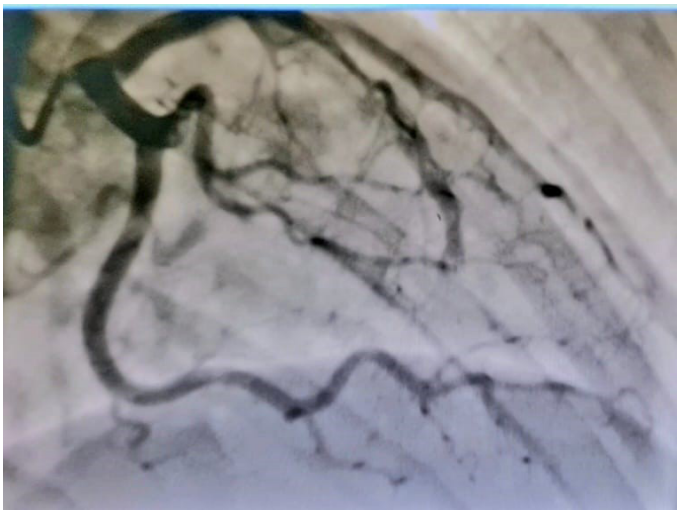

Figure 2: Angiography showing complete thromboembolic occlusion of mid LAD

DISCUSSION

Acute myocardial infarction in a young individual is usually considered below 45 years of age.[2] Though it is relatively an uncommon entity, it could be broadly classified into two categories: those with coronary artery disease and with normal coronaries. Conventional risk factors for coronary artery disease include smoking, diabetes mellitus, lipid abnormality and family history. On the other hand, thromboembolic phenomena, coronary spasm and arteritis are associated with myocardial infarction in normal coronaries.[3] Thromboembolic manifestations are not uncommon in glomerular diseases with nephrotic range proteinuria. Several factors are involved in the pathogenesis of such events; namely thrombocytosis, decreased levels of antithrombin III, plasminogen and free protein S, increased amounts of factor V, VIII and fibrinogen, increased platelet activation and hemoconcentration.[4]

In our index case, the patient presented with acute myocardial infarction two months after diagnosis of IgA nephropathy. His proteinuria was under control with ARB, immunomodulator, and steroid therapy. A similar case of IgA nephropathy with pulmonary thromboembolism and renal artery infarction was also reported in the literature who was proteinuria at the time of presentation.[5] In fact, it was a case of both venous and arterial side thromboembolism reported on the same individual, similar episodes of arterial side thrombosis: namely femoral, popliteal, brachial arteries along with coronary and cerebrovascular arteries in glomerular disease have been reported in the studies.[6,7] On literature review, the risk of thromboembolic events in glomerular disease is found to be higher in membranous nephropathy (7.85%) than in focal segmental glomerulosclerosis (2.97%) and IgA nephropathy (0.36%).[8] To our best knowledge this is the novel case of IgA nephropathy reported who presented with acute myocardial infarction.

Persistent proteinuria is one of the independent risk factors for cardiovascular events however no distinct threshold has been defined that confers the increased risk. Hypertension with presence of proteinuria and hypoalbuminemia increases a four-time greater risk of coronary event than in absence of proteinuria.[9] This effect appears to be independent of conventional atherosclerotic risk factors. In literature, proteinuria suggests subclinical inflammation with generalized dysfunction of the vascular endothelium. Von Willebrand factors, soluble vascular cell adhesion molecule, fibrinogen, and tissue plasminogen activator along with elevated CRP level have been implicated to be potential thrombogenic factors for cardiovascular events.[10] Immunosuppressive agents are also known to cause increased cardiovascular risk factors such as high blood pressure, high lipids, and high blood sugar. However, in recent study mycophenolate is found to be beneficial which confers protective effects against ischemia/reperfusion injury, through its immunosuppressive and anti-inflammatory actions.[11] Enough evidence against immune mediated glomerular disease is still lacking, however few ongoing trials are there on reducing cardiovascular risk in renal transplant patients which will definitely provide some better evidence in near future.

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