

Acute Coronary Syndrome in FSGS: A Case Report

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ABSTRACT

Background: A 38-year-old male, who has been on treatment for nephrotic syndrome for 2 years, presented with sudden onset of chest pain, vomiting and excessive sweating. His ECG showed features of STEMI with elevated serum troponin. Coronary angiography was unavailable in our centre. He was treated with antiplatelets and anticoagulants and subsequently discharged on cyclophosphamide.

Keywords: Nephrotic syndrome, acute coronary syndrome

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Introduction

Nephrotic syndrome is a common disorder that manifest with massive proteinuria, hypoalbuminaemia, oedema, dyslipidaemia and/or lipiduria.¹ It is the archetype of glomerular disease. Nephrotic syndrome is generally classified into primary, when there is no obvious cause and secondary, when due to a systemic disorder which affects the kidneys. Several complications occur in patients who develop nephrotic syndrome such as infections, thromboembolic events, abnormalities of metabolism of protein and hormones, as well as acute or chronic kidney disease.² Thromboembolic events occur with frequency of 8 - 10% and predominantly affect the veins of the lower limbs. Arterial thromboses are rare in nephrotic syndrome.³ There are few case reports of coronary artery thrombosis in nephrotic syndrome; majority of which are among patients with membranous nephropathy.⁴ Although nephrotic syndrome patients have abnormalities of lipid, and atherosclerosis of coronary blood vessels has been demonstrated in them, most of the patients have an embolic blockage of their coronary vessels as the cause of acute myocardial infarction.⁵ Patients who have

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serum albumin levels <20g/dl, severe oedema, hypercholesterolaemia >12mmol/l and membranous nephropathy on histology are at higher risk of developing thromboembolic phenomena.⁶ The management of these patients will require a high index of suspicion since patients are usually children and young adults who present with typical symptoms of acute coronary syndrome.

Case Report

A 38-year-old male presented to the accident and emergency unit of the University of Maiduguri Teaching Hospital with complaints of sudden onset of retro-sternal chest pain, described as tightening of the chest. Pain radiates to the left arm and forearm. It was initially aggravated by walking, hours later it persisted even at rest. He had associated vomiting.

The patient had presented at the renal clinic 3 years previously with complaints of body swelling and was told he had nephrotic syndrome. His records showed proteinuria of 4.5g/24 hours, serum albumin 20g/l, creatinine 68 μ mol/l, urea 4.2mmol/l, total cholesterol 11.4mmol/l. His kidneys measured 10.83 x 4.77cm and 11.08 x 5.37cm on the left and right respectively. Antibodies to HIV and HCV were negative and HBsAg was also negative. He was given Atorvastatin 20mg daily, Lisinopril 10mg daily, Aspirin 75mg daily, Frusemide 40mg daily and Prednisolone 1mg/kg/day. His symptoms resolved gradually after starting the above treatment with the results of investigations done after 8 months showing albumin 40g/l, total cholesterol 3.9mmol/l, creatinine

90 μ mol/l, urea 4.1mmol/l, proteinuria 0.1g/24hours. He was subsequently lost to follow up.

On admission he was severely diaphoretic and anxious, not pale, BMI was 21.6Kg/m², Pulse rate was 122beats/minute with normal arterial wall, blood pressure 140/90mmHg in the right arm supine, jugular venous pressure was not raised and cardiac apex was localized to the 5th left intercostal space at the mid clavicular line. His SPO₂ was 97% on room air, lung fields were clear.

Investigation results showed ST segment elevation in anterior leads. Packed cell volume was 34%, white blood cell count was 7.45 x 10⁹/l, platelets were 250 x10⁹/l, Total cholesterol 4.1mmol/l, HDL 1.0mmol/l, FBG 6.1mmol/l, creatinine 107 μ mol/l, urea 5.7mmol/l, Albumin 27g/l, proteinuria 3+.

A diagnosis of ST segment elevation myocardial infarction on background nephrotic syndrome was made. And he was commenced on Aspirin 300mg stat, then continued on 150mg daily, Clopidogrel 75mg daily, Subcutaneous Clexane® (enoxaparin) 80mg twice daily, Isosorbide dinitrate 5mg twice daily, Atorvastatin 40mg daily, Carvedilol 6.25mg twice daily. Coronary angiography was not available in our centre and percutaneous coronary intervention was also not done due to non-availability.

He was subsequently discharged after 7 days of admission on carvedilol 6.25mg twice daily, rosuvastatin 10mg daily, Vasoprin® 75mg daily, and clopidogrel 75mg daily.

Two weeks after discharge, renal biopsy was done which showed focal segmental glomerulosclerosis.



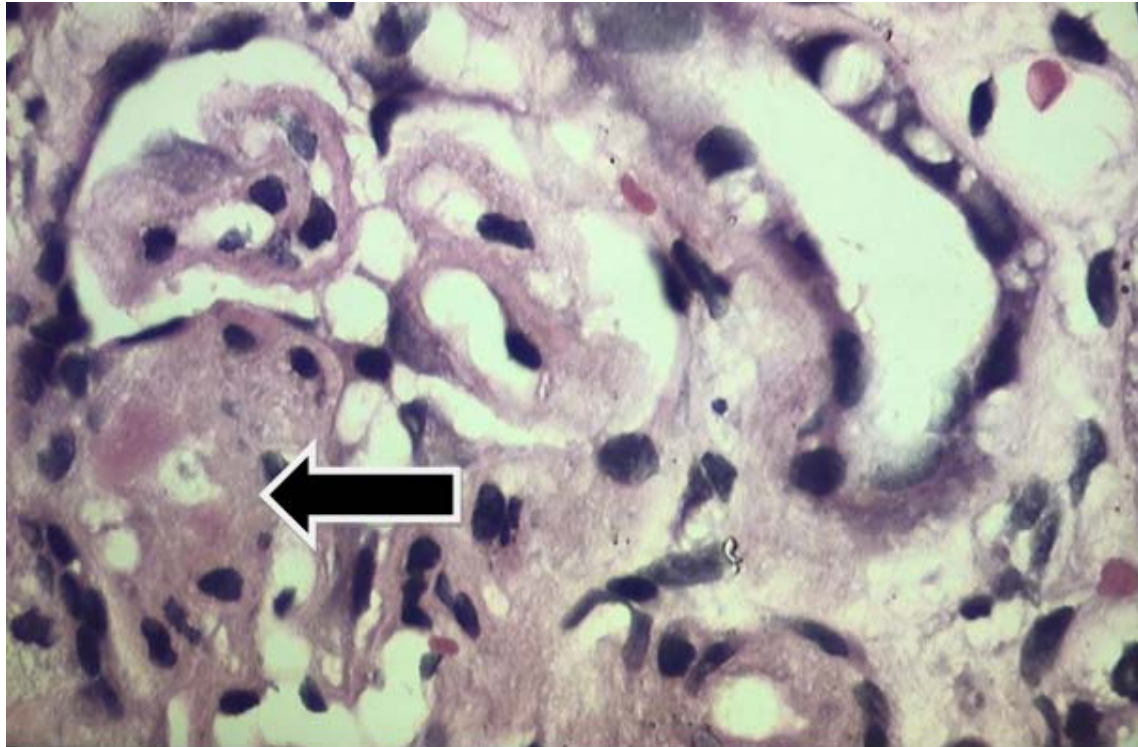


Figure 1: Glomerulus with segmental sclerotic lesion



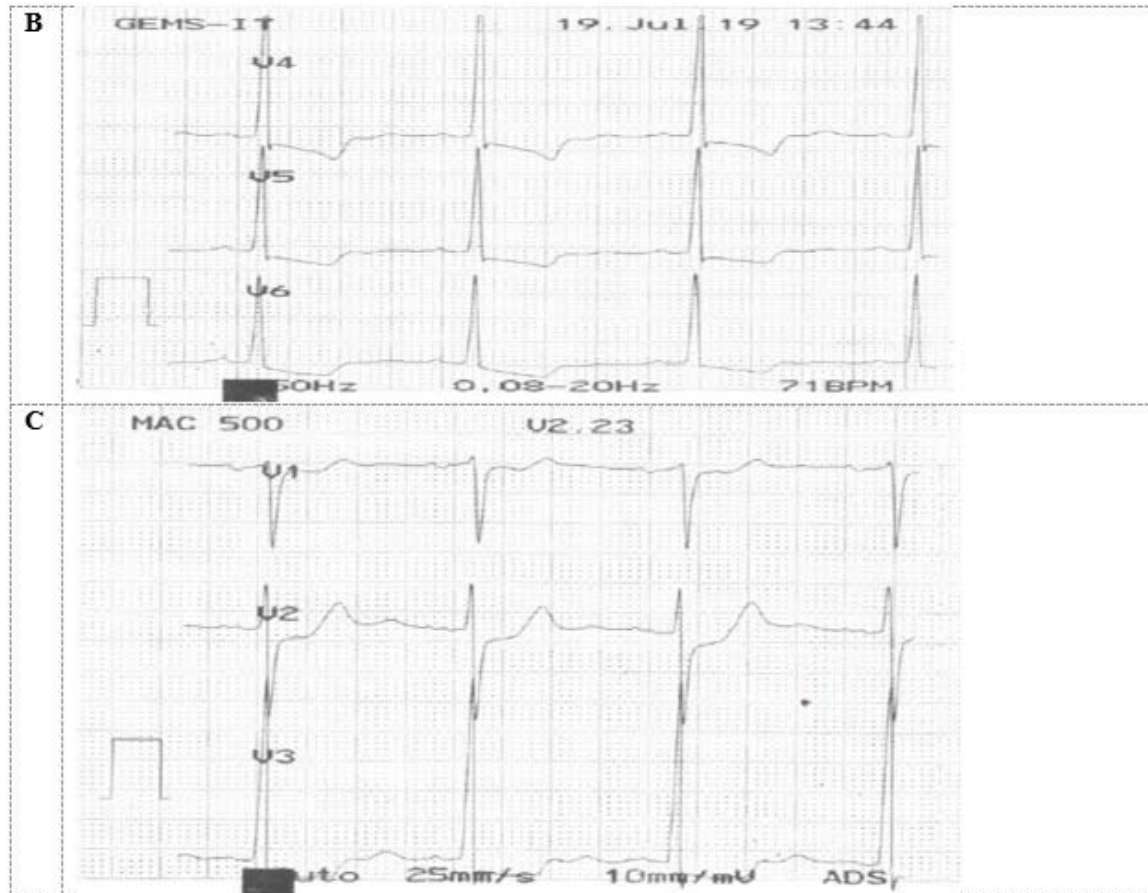


Figure 2: Electrocardiograph of the patient showing (A) ST-segment elevation in leads II, III and aVF, (B and C) reciprocal changes in leads V4-6.

Discussion

Acute myocardial infarction is an infrequent complication of nephrotic syndrome. In patients with secondary nephrotic syndrome such as lupus nephritis, myocardial infarction may develop from the secondary condition.⁷ Patients with primary nephrotic syndrome develop arterial and venous thrombosis. Patients are usually younger than patients who develop myocardial infarction from atherosclerosis.

Our patient is a 38-year-old male. Symptoms are typical of myocardial infarction presenting with chest pain that is characterized as either tightness or heaviness. Electrocardiographic findings include both ST segment elevation and non-ST segment elevation MI.

In nephrotic patients, hypercoagulability results from changes in the levels of coagulation proteins, enhanced platelets aggregability and hyperlipidaemia.⁸ Most reported cases of myocardial infarction associated with nephrotic syndrome showed normal coronary arteries. However, there are few reported patients with atherosclerotic changes in coronary and cerebral vessels. Prolonged immobility and co-infection can have an additive effect on thrombus formation. It is, however, not clear why thrombosis occurs in the coronary arteries in some patients whereas it occurs in other vessels in others.⁹

Increased urinary loss of low molecular proteins leads to deficiency of coagulation

factors IX, XI and XII. In contrast levels of high molecular factors II, V, VII, X, XIII and fibrinogen are raised due to increased hepatic synthesis. Although thromboembolism complicates up to 10-15% of nephrotic syndrome, membranous nephropathy has been found to confer a higher risk of thromboembolism. This risk is associated with severity of hypoalbuminaemia (<20g/d) and severity of oedema which perhaps promotes immobilization.¹⁰

Patients who are evaluated with coronary angiography show thrombosis most of the time rather than atherosclerosis.

The right coronary artery (RCA) and left anterior descending (LAD) are most commonly involved with evidence of slow blood flow.

Treatment may require extraction of blood clots from the affected blood vessel or placement of stents in few patients.¹¹

Prevention of thrombosis in patients with nephrotic syndrome requires identification of factors associated with increased risk of thrombosis such as hypoalbuminaemia <20mg/dl, thrombocytosis, and increased platelets aggregation and adhesiveness, elevated serum cholesterol >7mmol/l and membranous nephropathy on histology. Many of these abnormalities are present in our patient and may have contributed to the development of coronary thrombosis. However, our patient has FSGS on histology.¹¹

Careful risk factor assessment is essential in patients with nephrotic syndrome so that prophylactic antiplatelets and anticoagulation can be given.

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