中文題目：糖尿病病人的繼發性心腎症候群：個案報告

英文題目：Secondary Cardiorenal Syndrome in Diabetic Patients- A Case Report

作　　者：趙心毓[1]，邱怡文[1,2]，蔡宜純[1,2,3,]

服務單位：[1]高雄醫學大學附設醫院內科部，[2]腎臟內科，[3]一般醫學內科

***Introduction**:*

Complications of diabetes mellitus (DM), such as microvascular or macrovascular disease and immune dysfunction, are generally mentioned. We reported a case of a 47-year-old diabetic male who had both acute kidney injury with post streptococcus glomerulonephritis and infectious endocarditis with an initial presentation of left foot necrotizing fascilitis and osteomyelitis. Local infection might contribute to secondary cardiorenal syndrome and glomerulonephritis in diabetic patients.

***Case Report**:*

A 47-year-old male had significant past medical history for poor-controlled DM. He had recurrent left foot necrotizing fasilitis and osteomyelitis with multiple debridement in the past 2 years. This time, he presented fever, chills and foul odor noted from his left wound graft for 2 days. Upon arrival, his body temperature was 39℃, and had tachycardia (119bpm) with blood pressure of 155/114 mmHg. Laboratory findings showed elevated erythrocyte sedimentation rate (52 mm/h) and c-reactive protein level (48.54mg/L). Urine analysis showed hematuria and proteinuria. Blood culture showed Streptococcus mutans in two sets. Transesophageal echocardiography demonstrated vegetation at left anterior mitral leaflet and posterior mitral leaflet (Figure 1). We prescribed empirical antibiotic of ceftriaxone and metronidazole for streptococcus mutans bacteremia accompanied subacute infective endocarditis and bacteroides wound infection and osteomyelitis at left foot.

Progressive acute kidney injury was noted with serum creatinine level elevated from 1.11 mg/dL to 4.57 mg/dL within 2 weeks. Urine routine showed remarkable hematuria and proteinuria, with urine protein-creatinine ratio (9.33 mg/mg) in the range of nephrotic syndrome. Renal echo revealed bilateral enlarged kidney size (both 13cm) without hydronephrosis. Markedly lower Complement component 3 (C3) level (23.9 mg/dL), elevated serum Immunoglobulin G (IgG) level (1820 mg/dL) and positive of blood cryoglobulin level were shown. Tc-99m-DMSA renal cortical scan

showed no evidence of renal cortical necrosis, and renal infarction from septic emboli was not favored.

Therefore, we arranged ultrasound-guided renal biopsy for further pathological study of the etiologies of glomerulonephritis and nephritic syndrome. The renal biopsy specimen (Figure 2) revealed C3-predominant immune-complex deposits related glomerulonephritis with crescent formation with mesangial proliferation. Immunofluorescence in glomerular basement membrane demonstrated coarse granular pattern with positive Immunoglobulin M (IgM) and C3. Electron microscopy showed scattered electron dense deposits in mesangium and intramembranous area. The result of renal biopsy presented post-streptococcus glomerulonephritis. We kept antibiotic treatment for 4 weeks, and the renal function recovered gradually.

Conclusion*:*

Early detection and treatment of local infection of diabetes are critical to prevent systemic injury to vital organs. In our case, a 47-year-old male with poor diabetes control complicated with wound infection suffered from bacteremia, secondary cardiorenal syndrome including infectious endocarditis and acute kidney injury with poststreptococcal glomerulonephritis at the same time. Although diabetes directly induced glomerular sclerosis and fibrosis will be first suspected in a patient with poor glycemic control presenting proteinuria, hematuria and impaired renal function, other etiologies of acute kidney injury and glomerulonephritis should be considered if the clinical course of the patient presents acutely and rapidly.

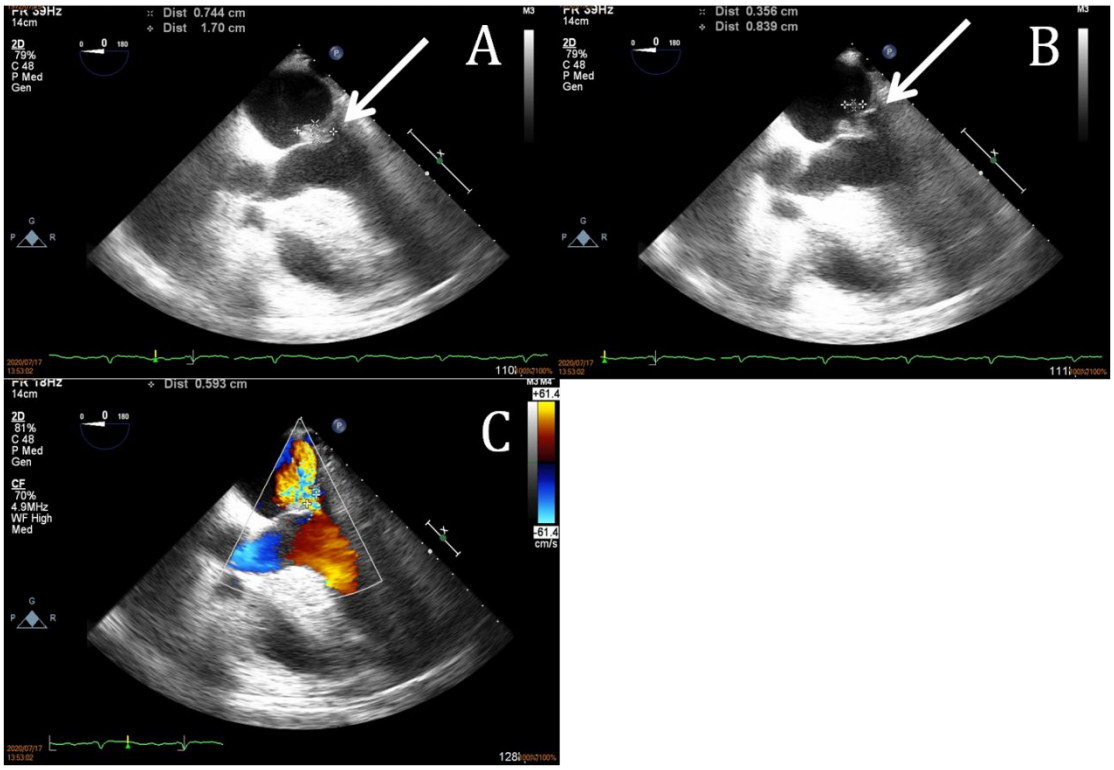

Figure 1. Transesophageal echocardiography images of patient.

Oscillation mass over (A) anterior mitral leaflet (1.6*1.2 cm2) (white arrow) and (B) posterior mitral leaflet (0.84*0.36 cm2) (white arrow). (C) Eccentric severe mitral regurgitation with vena contracta of 0.73mm.

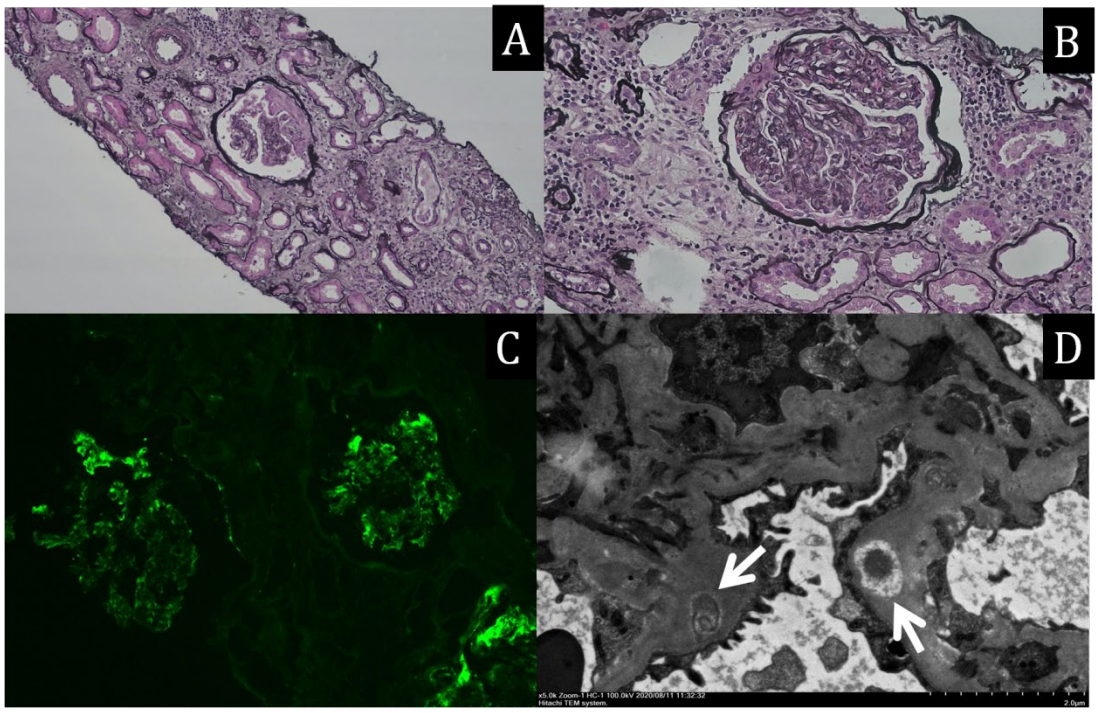

Figure 2. Pathology from patient's renal biopsy.

(A) Light microscopy showing interstitial fibrosis and tubular atrophy. (PASM, 10x). (B) Light microscopy showing a proliferative (exudative) glomerulonephritis. Note numerous neutrophils within glomerular capillaries and prominent endocapillary proliferation (Periodic Schiff-Methenamine Silver (PASM, 20x). (C) Immunofluorescence microscopy showing bright granular capillary wall staining for C3 (20x). (D) Electron microscopy showing scattered electron dense deposits (white arrow) in mesangium and intramembranous area (5000x).