Diabetic nephropathy patient with heavy proteinuria: 
A case report

Received October 08, 2020
Accepted January 24, 2022

Pei Yu1,2,3,4, Manman Lu1,2,3,4, Dongwei Liu1,2,3,4, Dan Gao1,2,3,4,*

1Department of Integrated Traditional and Western Nephrology, the First Affiliated Hospital of Zhengzhou University, Zhengzhou 450052, Henan Province, China
2Research Institute of Nephrology, Zhengzhou University, Zhengzhou 450052, Henan Province, China
3Henan Province Research Center For Kidney Disease, Zhengzhou 450052, Henan Province, China
4Key Laboratory of Precision Diagnosis and Treatment for Chronic Kidney Disease in Henan Province, Zhengzhou 450052, Henan Province, China

Abstract

We herein report a case of advanced stage rapidly progressive diabetic nephropathy that finally received long-term renal replacement therapy. A 53-year-old man with hypertension and heavy proteinuria suffered rapid progression of DKD [estimated glomerular filtration rate (eGFR): 18 mL/min/1.73m²; proteinuria: 12.21 g/d]. Renal biopsy revealed diabetic nephropathy (Class III) with severe interstitial lesion and tubular atrophy. Despite aggressive treatment, the proteinuria and blood pressure were poorly controlled. The patient finally became dialysis dependent. We hold the position that this is related to his proteinuria and hypertension.

Keywords

clinical prognosis • diabetic nephropathy • proteinuria

1. Introduction

Diabetic kidney disease (DKD) is the most common cause of end-stage renal disease (ESRD) worldwide, and the incidence of ESRD secondary to DKD is increasing rapidly over the years. The classical progression of DKD is deterioration of renal function over decades. Annual decline in glomerular filtration rate (GFR) in a person varies widely depending on various factors such as ethnicity, age, underlying medical problems, the etiology of chronic kidney disease (CKD), and the presence of comorbidities[1]. The results of our report allow us to infer that, apart from poor glycemic level, proteinuria and hypertension may be the leading risk factors for rapid progression of DKD.

2. Case Report

A 53-year-old man was diagnosed with diabetes mellitus 5 years ago. He was treated with subcutaneous insulin aspart 6IU three times per day (TID) and recombinant insulin glargine 12IU QN. He was admitted to our hospital because of a vitreous hemorrhage in March 2019. At that time, his diabetes mellitus control was not very good, with a glycated hemoglobin (HbA1) level of 6.9%. On physical examination, it was found that he had bilateral proliferative diabetic retinopathy with vitreous hemorrhage. Additionally, his renal function was impaired with estimated glomerular filtration rate (eGFR) of 34.6 mL/min/1.73m². He was discharged after fundus surgery, and no medical procedures were carried out to address his renal insufficiency.

In November 2019, he was referred to our department for treatment of nephrotic syndrome with abnormal renal function (eGFR: 18 mL/min/1.73m², proteinuria 12.21 g/d). He was diagnosed hypertension 5 months ago and treated with enalapril. At that time, he was experiencing bilateral lower limb swelling for the past 2 months. His blood pressure was 180/101 mmHg. Before consulting our department, his kidney function was declining. (eGFR change: −16.0 mL/min/1.73m², serum creatinine change: +130 µmol/L) (Figure 1). Except for diabetic nephropathy, no other pathogenic condition could be found to explain his renal disease. In addition, he had not been taking any medicines including traditional Chinese medicine, supplement, and analgesics that could induce acute kidney injury. Emission Computed Tomography (ECT) showed that eGFR in the left kidney was 24.77 mL/min/1.73m², and that in the right kidney was 21.89 mL/min/1.73m². Renal ultrasound was normal. Renal biopsy was performed, and it revealed diabetic nephropathy (Class III) with severe interstitial lesion and tubular atrophy (Figure 2). He was treated with subcutaneous premixed insulin 9IU TID, nifedipine 30 mg twice per day (BID), terazosin 2 mg BID, and ophiocordyceps sinensis (Bailing capsule) 2 g TID. He was advised fluid and diet control upon discharge.

However, his edema did not release. So, he went to our hospital in December 2019. During this current presentation, blood investigations noted creatinine of 310 µmol/L (eGFR 33 mL/
min/1.73m²) (Table 1), albumin 23.8 g/L, hemoglobin 96 g/L, and HbA1c 6.9%. His proteinuria was 13.92 g/d (Table 1). He was treated with intravenous diuresis. We also gave him iron treatment to improve his hemoglobin level. Unfortunately, his edema became worse. In April 2020, he was re-admitted due to advanced edema and chest distress. His urine volume decreased to 800 mL/d. His blood pressure was 200/100 mmHg. His admission creatinine was 571 µmol/L. CT examination showed he had bilateral pleural effusion, bilateral pneumonia, and pericardial effusion. His renal function did not recover despite antibiotic and dialysis support. The patient became dialysis dependent and currently is on hemodialysis as his mode of long-term renal replacement therapy.

3. Discussion

DKD is the most common cause of ESRD worldwide, accounting for almost 50% of patients on renal replacement treatment in the USA [1].

The development of DN is usually observed in patients with long-standing diabetes and is frequently accompanied by other microvascular complications, especially diabetic retinopathy. The Renal Pathology Society developed a system to classify diabetic nephropathy, which classified these biopsies into four categories: glomerular basement membrane thickening (class I), mesangial expansion (class IIa, mild; and class IIb, severe), until the formation of Kimmelstiel–Wilson nodules (class III), and, finally, advanced glomerulosclerosis, which was classified as class IV [2]. In this classification, glomerular lesions are considered the predominant histologic finding, while tubulointerstitial and vascular lesions are considered as complementary additions to clinical prognosis.

The clinical manifestations of DKD are common to those of other chronic kidney diseases, such as proteinuria, declining GFR, and hypertension, while the lesions underlying renal dysfunction are typical of this disease [3].

The failing kidney is characterized histologically by tubulointerstitial inflammation, tubular cell apoptosis, tubular atrophy, and fibrosis, and these changes correlate with the severity of proteinuria. The renal biopsy of our patient showed a nodular glomerulosclerosis pattern with 50% global sclerosis. It also showed the K–W nodules and severe mesangial cell and matrix proliferation (Figure 2). These are typical pathological changes associated with diabetic nephropathy. Our patient also had a heavy level of proteinuria, which had been

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine (µmol/L)</td>
<td>188</td>
<td>318</td>
<td>310</td>
<td>549</td>
<td>585</td>
</tr>
<tr>
<td>GFR (mL/min/1.73m²)</td>
<td>34.6</td>
<td>18.2</td>
<td>19</td>
<td>9.4</td>
<td>8.7</td>
</tr>
<tr>
<td>HbA1c%</td>
<td>6.90</td>
<td>7.90</td>
<td>6.9</td>
<td>6.00</td>
<td>6.10</td>
</tr>
<tr>
<td>Proteinuria (g/d)</td>
<td>–</td>
<td>12.21</td>
<td>13.92</td>
<td>14.25</td>
<td>–</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>27.9</td>
<td>28.4</td>
<td>23.8</td>
<td>22.7</td>
<td>22.8</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>–</td>
<td>83.0</td>
<td>–</td>
<td>90.0</td>
<td>99.8</td>
</tr>
</tbody>
</table>

GFR, glomerular filtration rate.
Table 1. Laboratory and clinical measurements
recognized as an independent risk factor for renal function loss. The presence and severity of proteinuria has been shown to be a reliable strategy in the identification of rapid renal decline in a community-based prospective cohort study [4].

Kidney Disease Improving Global Outcomes (KDIGO) guidelines define rapid progression as rate of eGFR decline that is >5 mL/min/1.73m² per year [5]. The kidney function of our patient continues to decline quickly, and this may be related to the massive proteinuria and renal insufficiency at baseline. The poor prognosis of our patient may also be attributable to the poor control over blood pressure. Current guidelines suggest a blood pressure goal of <140/90 mmHg in DKD patients. A sustained reduction in blood pressure is an important single intervention that can slow down the progression of diabetic nephropathy.

During our treatment, the glucose and glycated hemoglobin dropped to almost normal, but the proteinuria and blood pressure were poorly controlled. The kidney disease of our patient keeps progressing. We hold the position that this is related to his proteinuria and hypertension.

Source of Funding
Nil.

Ethics approval and consent to participate

Ethical issues are not involved in this paper. Statement of Human and Animal Rights: All procedures performed in this study was involving with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Conflict of interest

Dongwei Liu is a managing editor. The article was subject to the journal’s standard procedures, with peer review handled independently of this editor and his research groups.

REFERENCES