Recovery of Chronic Dialysis Hypotension After Kidney Transplantation: A Case Report

Böbrek Nakli Sonrası Kronik Diyaliz Hipotansiyonunun İyleşmesi: Olgu Sunumu

ABSTRACT

Chronic dialysis hypotension is described as low systolic blood pressure (<100 mmHg) during interdialytic period. The presence of low predialysis systolic blood pressure, typically <110 mmHg, is significantly associated with increased mortality. Kidney transplantation is the preferred model of renal replacement therapy in the treatment of end-stage renal disease (ESRD) as it improves quality of life and survival. In this article, a long-term hemodialysis (HD) patient with chronic hypotension improved after kidney transplantation is presented. A 39-year-old male patient received a deceased donor kidney transplant. The patient was on HD for 23 years. The patient had suffered from chronic persistent hypotension for the last 8 years. Blood pressure was 70/50 mmHg before dialysis and 60/40 mmHg after dialysis. In the post-transplant period, blood pressure was maintained above 110/70 mmHg by intermittent infusion of dopamine. Hypotension was improved after 24 days and dopamine was discontinued. Various etiologies may cause chronic hypotension in patients receiving long-term HD treatment. Kidney transplantation may improve survival and quality of life by correcting hypotension in these patients. Therefore kidney transplantation should not be avoided as renal replacement therapy in ESRD patients with hypotension.

KEY WORDS: Long-term hemodialysis, Hypotension, Autonomic dysfunction, Kidney transplantation, Blood pressure

ÖZ

Kronik diyaliz hipotansiyonu iki diyaliz arasındaki dönemde sistolik kan basınç düştüğü (<100 mmHg) olarak tanımlanmaktadır. Diyaliz öncesi düşük sistolik kan basınçlı (tipik olarak 110 mmHg’ın altı) artmış mortalite ile anlaşılmıştır. Böbrek nakli yaşam kalitesini ve sağınlığı iyileştirdiği için son dönemde yapılan tedavi (SDBY) tedavisinde tercih edilen böbrekštir. Hastanın diyaliz öncesi 70/50 mmHg, diyaliz sonrası ise 60/40 mm Hg civarında seyretmektediydi. Böbrek nakli sonrası erken dönemde aralıklı dopamin infüzyonu ile kan basınçının 110/70 mm Hg’nin üstünde olması sağlanmıştı. Hipotansiyon 24 gün sonra düzelmiş ve dopamin infüzyonu kesildi. Uzun dönemde HD tedavisi alınan hastalarda birçok farklı neden kronik hipotansiyona neden olabilir. Bu hastalarda böbrek nakli hipotansiyonu düzeltmek için hayat kalitesini düzeltebilir. Uzun dönem kronik hipotansiyonu olan SDBY hastalarında böbrek naklinin kaçınılmamalıdır.

ANAHTAR SÖZCÜKLER: Uzun dönem hemodiyaliz, Hipotansiyon, Otonomik disfonksiyon, Böbrek nakli, Kan basınç
INTRODUCTION

Chronic dialysis hypotension is described as low systolic blood pressure (<100 mmHg) during interdialytic period (1). The presence of low predialysis systolic blood pressure, typically <110 mmHg, is significantly associated with increased mortality (2). Several reasons such as heart failure, severe volume overload and autonomic dysfunction may cause chronic dialysis hypotension (2,3). Autonomic dysfunction is associated with increased cardiovascular mortality in patients with advanced renal failure (4-7). Kidney transplantation is the preferred model of renal replacement therapy in the treatment of end-stage renal disease (ESRD) as it improves quality of life and survival (8). Normalization of renal function after kidney transplantation is associated with partial recovery of autonomic dysfunction (9). This situation may contribute to normalization blood pressure and the patient’s survival. In this article, a long-term HD patient with chronic persistent hypotension who improved after kidney transplantation is presented.

CASE REPORT

A 39-year-old male patient underwent renal transplantation from a 4 HLA–mismatched, 36-year-old, deceased donor in February 2012. Panel-reactive antibody was 0 %. Cold ischemia time was 8 h. The patient was on chronic HD program for 23 years because of ESRD due to chronic glomerulonephritis (probably membranoproliferative glomerulonephritis). He had a history of parathyroidectomy operation due to parathyroid adenoma 5 years ago and tuberculosis arthritis. He suffered from chronic hypotension and severe intradialytic hypotension for the last 8 years. Therefore HD program was planned as four session per week for 6 hours. The predialysis and postdialysis blood pressure of the patient was 70/50 mmHg and 60/40 mmHg, respectively. The systolic and diastolic dysfunctions were not detected in transthoracic echocardiography. Ejection fraction was 70 %. The electrocardiography of patient revealed only prolongation of the PR interval with normally sinus rhythm before and after the operation (Figure 1, 2). The preoperative levels of serum urea, creatinine, glucose, sodium and potassium were 101 mg/dl, 6.3 mg/dl, 94 mg/dl, 142 meq/l and 4.7 meq/l, respectively. In his preoperative physical examination, blood pressure was 70/50 mmHg. Respiratory and cardiovascular system examination was normal. There was no pretibial edema. Anti-thymocyte immunoglobulin (ATG,Fresenius) was used for induction therapy. Cumulative dose of 1200 mg of ATG was given within 27 days after renal transplantation. The patient received methyl prednisolone I.V. 500 mg b.i.d in the pretransplant and posttransplant period at day 0. Steroid dose was gradually reduced to 30 mg/day by 9th day post-transplant. Mycophenolate sodium (MPS) was started at a dose of 1440 mg/day at the day of operation. Postoperatively, delayed graft function was observed. At follow up, the patient had adequate urine output. When the creatinine level fell below 2 mg/dl at the 30th day, tacrolimus was started at the dose of 0.1 mg/kg/day.

Hypotension continued in the early period after transplant operation. Blood pressure was maintained above 110/70 mmHg by intermittent infusion of dopamine at a dose of 5 to 10 micrograms per kg per minute. In addition, low molecular weight heparin (enoxaparin sodium 40 mg per day subcutaneously) was given for prophylaxis of vascular thrombosis. Postoperatively, ten sessions of HD were performed due to the delayed graft

![Figure 1](image1.png): The electrocardiograph of the patient before the operation.  
![Figure 2](image2.png): The electrocardiograph of the patient after the operation.
function within 24 days. Dopamine infusion dose was increased to 10 micrograms per kg per minute to prevent the development of hypotension on HD sessions. Hypotension improved after 24 days follow-up period. Dopamine was not required after that time. Four months after transplantation the patient was prescribed 5 mg of amlodipine due to high blood pressure. This drug was stopped when blood pressure was 110/70 mmHg. The patient has just been at 11th months of kidney transplantation. Blood pressure was maintained at 120-130/70-80 mmHg without any antihypertensive drugs and the last serum creatinine level decreased to 1.37 mg/dl. The patient received prednisolone 5 mg per day, MPS 1080 mg per day, tacrolimus 1 mg per day as maintenance immunosuppressive treatment.

DISCUSSION

Our case was on chronic HD program for 23 years and he had a history of chronic persistent hypotension and severe intradialytic hypotension for the last 8 years. In such long-term HD patients, cardiac causes should be considered in the differential diagnosis of chronic hypotension. Respiratory and cardiovascular system examination of the patient was normal; any pathology responsible for cardiac hypotension was not detected by echocardiography and electrocardiography. The patient did not have any signs or findings such as electrolyte imbalance or abdominal pain or low blood glucose that support adrenal insufficiency except hypotension. In addition, the survival of 8 years in patients with adrenal insufficiency without treatment is not expected. If the diagnosis were adrenal insufficiency, hypotension would improve within a few days after steroid therapy and fluid replacement. The hypotension resolved after 24 days. Therefore adrenal insufficiency was not considered. However, we could not perform tests for autonomic dysfunction in our routine practice. We did not detect any cause for chronic hypotension in our patient. Therefore we thought that chronic hypotension in our case could be due to autonomic dysfunction. Nevertheless, the diagnosis of autonomic dysfunction of our patient was speculative as we could not identify it definitely.

Chronic hypotension due to autonomic dysfunction is one of the long-term complications of chronic kidney disease. Uremic toxins are thought to be responsible for autonomic neuropathy and hypotension in these patients. This situation increases with duration of ESRD (2). Improvement of all autonomic dysfunction parameters such as heart rate variability, blood pressure variability and baroreflex sensitivity after successful kidney transplant has been shown (10). Improvement of hypotension in the early postoperative period suggested the recovery of autonomic dysfunction in our case.

The patient had used prednisolone, MPS, and tacrolimus as immunosuppressive therapy. Tacrolimus was started when the creatinine level was 2 mg/dL and after the blood pressure increased to a normal level. Therefore, this drug was not considered to contribute to the increase in blood pressure.

Chronic hypotension is associated with increased mortality such as chronic hypertension in HD patients (11). It has been reported that relationship between mortality and predialytic systolic and diastolic blood pressure was similar to “U” shape, and 3-year mortality rate was the lowest in patients with predialytic systolic blood pressure 160-179 mmHg, mortality was 3 times higher in patients with systolic blood pressure lower than 120 mmHg and mortality was 1.2 to 1.5 higher in patients with systolic blood pressure higher than 200 mmHg (12). Higher risk of mortality in chronic hypotension has been described as a component reversed epidemiology (13). In our patient systolic blood pressure was lower. Although he was on an HD program of four sessions per week for 6 hours, he still suffered from chronic hypotension and severe intradialytic hypotension. The risk of increased mortality could be mentioned before kidney transplantation for our patient. The rise in patient’s blood pressure by kidney transplantation would probably reduce the risk of increased mortality.

Clinicians can be unwilling to perform kidney transplantation due to early postoperative vascular complications such as renal artery or vein thrombosis, especially in long-term HD patients with hypotension. We wanted to emphasize that clinicians should be more willing to transplant such patients by presenting that case.

In conclusion, chronic persistent hypotension may occur because of various etiologies in patients receiving long-term HD treatment. Kidney transplantation may improve survival and quality of life by correcting hypotension in these patients. Therefore we thought that kidney transplantation should not be avoided as renal replacement therapy in ESRD patients with chronic hypotension.

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