Kidney Disease Associated With Anorexia Nervosa: A Case Series With Kidney Biopsies

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Rationale & Objective: Anorexia nervosa is often intractable and induces various physical disorders, including kidney disease and mineral disorders, occasionally progressing to kidney failure. No consensus-based clinical practice guidelines have been established for patients with anorexia nervosa referred to a nephrologist.

Study Design: Patients with anorexia nervosa–associated kidney disease diagnosed were analyzed retrospectively. Kidney outcomes were defined as doubling of serum creatinine level and/or progression to end-stage kidney disease.

Setting & Participants: Patients with a history of anorexia nervosa with kidney disease, including electrolyte abnormalities, who were referred to our hospital between 1992 and 2017 were included.

Results: 14 female patients were included. The time from anorexia nervosa onset to the initial visit with a nephrologist was 17.8 years. At the first visit, median body mass index was 13.4 kg/m^2, median serum creatinine level was 1.9 mg/dL, and median

serum potassium level was 2.7 mmol/L. All patients showed hypokalemia and addictive vomiting or diuretic/laxative abuse. During the median observation period of 3.1 years, kidney outcomes occurred in 9 patients, and 2 died due to their anorexia nervosa. 4 patients underwent kidney biopsy. The kidney biopsy findings of these patients included hypertrophy of the juxtaglomerular apparatus, advanced glomerular collapse, and interstitial fibrosis, consistent with ischemic kidney injury and hypokalemic nephropathy.

Limitations: The sample size was small, and kidney function was assessed based on serum creatinine levels in patients with anorexia nervosa with low muscle mass.

Conclusions: Most patients with anorexia nervosa referred to nephrologists had kidney disease at the time of the first visit. Improving kidney outcomes of patients with anorexia nervosa may require earlier collaboration between psychiatrists and nephrologists.

Eating disorders, including anorexia nervosa, are abnormal eating behaviors that occur mainly in young women and are characterized by complications such as higher-order dysfunction, mental distress, and other psychiatric disorders.[1] Although the prevalence of anorexia nervosa has remained stable in recent years, it has increased from 0.3% to 1.0% of the population compared with several decades ago.[2,3] Patients with anorexia nervosa have diverse physical complications, and the mortality rate is estimated to be >5%.[4] Thus, it is socioeconomically essential to recognize that patients with anorexia nervosa require much medical care because of their increased incidence of morbidity and various complications, as well as their high mortality rate.

Kidney disorders are among the most common complications of anorexia nervosa. The lifetime prevalence of kidney disorders, including electrolyte abnormalities, among patients with anorexia nervosa is approximately 70%.[5,6] Notably, a previous study reported that 5.2% of patients with anorexia nervosa develop end-stage kidney disease and undergo kidney replacement therapy.[7]

It has been postulated that kidney disease in patients with anorexia nervosa is caused by kidney tubulointerstitial inflammation and fibrosis due to the complex interplay of hypokalemia, kidney calcification, chronic dehydration, and rhabdomyolysis.[8,9] However, the precise

mechanisms underlying kidney disorders in patients with anorexia nervosa are unclear. This is probably because only a small fraction of the large number of patients with anorexia nervosa with kidney disease are referred to a nephrologist. In addition, most previous studies of kidney disease in patients with anorexia nervosa were conducted by psychiatrists; few have focused on the nephrologic aspects of anorexia nervosa.[10,11] In this study, we report a series of patients with anorexia nervosa who were treated by nephrologists, including some who underwent kidney biopsy.

METHODS

Patient Selection

This study included patients with a history of an eating disorder complicated with kidney disease including electrolyte abnormalities referred to Jikei University Hospital, Jikei University Kashiwa Hospital, or Kawaguchi Municipal Medical Center between January 1992 and December 2017. Patients with an eating disorder, such as anorexia nervosa and/or bulimia nervosa, who had been coded as *International Classification of Diseases, Ninth or Tenth Revision*, were retrospectively extracted, and a definitive diagnosis of anorexia nervosa was made by a psychiatrist. Exclusion criteria were 18 years or younger,

PLAIN-LANGUAGE SUMMARY

Anorexia nervosa is a type of abnormal eating behavior seen mainly in young women. This study was inspired by encountering a patient with anorexia nervosa with kidney failure undergoing dialysis therapy. This study was performed to clarify the characteristics of patients with anorexia nervosa who visited the hospital to obtain care for kidney disease. In patients with anorexia nervosa, habitual vomiting or diuretic/laxative abuse may cause irreversible damage to the kidneys. Therefore, patients with anorexia nervosa require both psychiatric and physical support.

missing results of blood or urine tests, and drop out within 2 months.

Consent was obtained by opting out for individual participants. All participants were provided the opportunity to read and review the presented document in the hospital and ask questions and discuss the study. This study was approved by the ethics review board of Jikei University School of Medicine [31-162(9661)].

Definition

We defined estimated glomerular filtration rate (eGFR) following the method of the Japanese Society of Nephrology: eGFR $(mL/min/1.73 m^2) = 194 \times creatinine^{-1.094} \times age^{-0.287} \times 0.739$ (if female).[12] We calculated body surface area (BSA) using the DuBois method: BSA $(m^2) = 0.007184 \times weight (kg)^{-0.425} \times height (cm)^{-0.725}$.[13] Body mass index (BMI) was calculated as weight (kg) ÷ [height (m)]^2. A lean state was defined as

BMI < 18.5 kg/m^2. We defined kidney disease as eGFR < 60 mL/min/1.73 m^2, hypokalemia as potassium level < 3.5 mmol/L, phosphorus disorder as phosphorus level > 4.5 or <2.5 mg/dL, and calcium disorder as corrected calcium level > 10.2 or <8.4 mg/dL. Kidney outcome was defined as a composite of doubling in serum creatinine level from baseline or progression to end-stage kidney disease (eGFR < 15 mL/min/1.73 m^2).[14] Initiation of kidney replacement therapy or death was applied as a hard outcome. All blood measurements were taken from serum samples. Patients who had a history of diuretic or laxative abuse were identified based on medical records. A history of vomiting was defined as past habitual vomiting.

Histopathologic Analyses

In 3 patients, kidney specimens were obtained by percutaneous needle biopsy using an 18-gauge biopsy needle. One patient underwent open biopsy, and the specimen was obtained by wedge resection. Tissues were embedded in paraffin, cut into 3- to 4-μm sections, and stained with hematoxylin and eosin, periodic acid–Schiff, Masson trichrome, and periodic acid–methenamine silver. Von Kossa staining was performed to visualize calcium deposits. The area of interstitial fibrosis and/or tubular atrophy was evaluated semi-quantitatively.[15] The number and percentage of glomeruli affected by global glomerulosclerosis were assessed. All biopsy specimens were examined with immunostaining and electron microscopy.

RESULTS

Baseline Characteristics and Kidney Outcomes

Baseline characteristics of patients are shown in Table 1. Median age at the time of anorexia nervosa diagnosis was

Table 1. Patient Characteristics

Variables	All Subjects (n = 14)	Variables	All Subjects (n = 14)
Female sex	14 (100%)	Aldosterone to renin activity ratio	16.9 [10.3, 38.1]
Time to the first visit, y	17.5 [10.8, 23.5]	Plasma aldosterone concentration, pg/mL	128 [77, 465]
Age at the first visit, y	39.0 [33.3, 44.0]	Plasma renin activity, ng/mL/h	11.4 [2.3, 19.8]
Age at eating disorder onset, y	19.5 [17.3, 26.0]	pH	7.4 [7.4, 7.5]
BMI, kg/m²	13.4 [12.4, 15.1]	Base excess	3.2 [−6.3, 13.9]
BSA, m²	1.21 [1.19, 1.33]	P_{CO_2}, mm Hg	44 [40, 51]
Follow-up period, y	3.1 [0.7, 5.9]	HCO_3^-, mmol/L	27.4 [19.2, 39.6]
		Urinary protein excretion, g/g Cr	0.14 [0.04, 0.44]
Serum creatinine, mg/dL	1.9 [1.2, 3.5]	Urinary β2-MG, μg/g Cr	4,000 [540, 54,986]
eGFR, mL/min/1.73 m²	20.9 [8.5, 32.2]	Urinary NAG, U/g Cr	18.3 [8.9, 39.0]
Serum urea nitrogen, mg/dL	29.7 [17.0, 55.6]	FE_{Na}, %	0.28 [0.08, 1.38]
Serum sodium, mmol/L	138 [136, 141]		
Serum potassium, mmol/L	2.7 [2.2, 3.1]	Use of diuretics or laxatives	7 (50%)
Serum chloride, mmol/L	95 [87, 100]	Habit of vomiting	13 (93%)
Serum phosphorus, mg/dL	3.85 [3.20, 5.17]	Hypertension	0 (0%)
Serum calcium, mg/dL	9.15 [8.62, 9.57]	Use of RAAS inhibitors	0 (0%)
Serum magnesium, g/dL	2.30 [2.02, 2.70]	Death	2 (14%)

Note: Values for categorical variables are given as number (percent); values for continuous variables are given as median [interquartile range]. Conversion factors for units: creatinine in mg/dL to μmol/L, ×88.4; serum urea nitrogen in mg/dL to mmol/L, ×0.357; serum calcium in mg/dL to mmol/L, ×0.2495.
Abbreviations: BMI, body mass index; BSA, body surface area; Cr, creatinine; eGFR, estimated glomerular filtration rate; FE$_{Na}$, fractional excretion of sodium; MG, macroglobulin; NAG, N-acetyl-β-ᴅ-glucosaminidase; RAAS, renin-angiotensin-aldosterone system.

Table 2. Comparison Between 2 Groups With or Without Kidney Outcomes and Kidney Biopsy During Follow-up

Variables	Kidney Outcomes	
	Without Kidney Outcomes (n = 5)	With Kidney Outcomes (n = 9)
At diagnosis		
Years to first visit to nephrologist (years)	14 [6, 18]	19 [13, 24]
Age at first visit to a nephrologist, y	34 [33, 35]	42 [37, 48]
Age at eating disorder onset, y	20 [17, 27]	19 [18, 23]
Body weight, kg	39 [35, 40]	31 [27, 33]
BMI, kg/m²	15.2 [14.6, 15.6]	12.8 [11.0, 13.9]
BSA, m²	1.34 [1.26, 1.38]	1.19 [1.12, 1.22]
Serum albumin, g/dL	3.9 [3.2, 4.6]	3.5 [3.4, 4.0]
eGFR, mL/min/1.73 m²	34.7 [27.0, 57.5]	13.8 [10.7, 40.4]
Serum urea nitrogen, mg/dL	24 [11, 32]	46 [20, 63]
Serum sodium, mmol/L	136 [135, 138]	139 [137, 141]
Serum potassium, mmol/L	2.6 [2.4, 2.8]	2.8 [2.1, 3.1]
Serum chloride, mmol/L	95 [95, 99]	91 [85, 100]
Serum calcium, mg/dL	9.2 [8.6, 9.5]	9.1 [8.7, 9.6]
Serum magnesium, g/dL	1.95 [1.85, 2.02]	2.60 [2.32, 4.38]
pH	7.44 [7.43, 7.45]	7.44 [7.25, 7.47]
Base excess, mmol/L	2.25 [-2.75, 8.90]	3.20 [-12.05, 14.03]
PCO_2, mm Hg	43.6 [39.7, 50.9]	43.7 [41.4, 51.5]
HCO_3^-, mmol/L	26.7 [20.8, 34.8]	27.4 [14.6, 39.6]
ACTH, pg/mL	17.9 [15.8, 20.1]	26.8 [20.9, 28.8]
Cortisol, μg/dL	15.0 [11.3, 19.7]	16.5 [13.0, 17.7]
Aldosterone to renin activity ratio	24.1 [16.2, 34.9]	14.7 [10.3, 34.5]
Plasma aldosterone concentration, pg/mL	128 [93, 252]	130 [77, 779]
Plasma renin activity, ng/mL/h	9.9 [2.6, 18]	13 [2.7, 37]
During follow-up		
Follow-up, y	1.8 [0.3, 3.3]	4.8 [2.0, 6.0]
Serum creatinine at last observation, mg/dL	1.04 [0.93, 1.71]	3.65 [3.04, 4.84]
eGFR at last observation, mL/min/1.73 m²	51.6 [27.9, 58.8]	12.9 [9.57, 16.2]
Patient reached doubling of creatinine	0	6
Patient reached ESKD	0	7

Note: Values for categorical variables are given as number; values for continuous variables are given as median [interquartile range]. Conversion factors for units: creatinine in mg/dL to μmol/L, ×88.4; serum urea nitrogen in mg/dL to mmol/L, ×0.357; serum calcium in mg/dL to mmol/L, ×0.2495.
Abbreviations: ACTH, adrenocorticotropic hormone; BMI, body mass index; BSA, body surface area; eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease.

19.5 years, and that at the time of the first visit to a nephrologist was 39.0 years. At the time of the first visit, all patients were lean (BMI < 18.5 kg/m²). Median creatinine level was 1.9 [interquartile range, 1.2, 3.5] mg/dL, median eGFR was 20.9 [interquartile range, 8.5, 32.2] mL/min/1.73 m². Thirteen patients (93%) had hypokalemia, 6 (43%) had a phosphorus concentration disorder, and 3 (21%) had a calcium concentration disorder.

Patients' Characteristics According to Kidney Outcomes

Clinical characteristics and outcomes of patients according to kidney outcomes are presented separately (Table 2). Of the 14 cases, 9 reached a kidney outcome; 6 had a doubling of creatinine level and 7 had end-stage kidney disease. Kidney replacement therapy was initiated in 3 of the 9 patients; however, 2 of them died due to anorexia nervosa.

Case Presentations of the 4 Patients Who Underwent Kidney Biopsy

Four of the 14 patients underwent kidney biopsy; their characteristics and histopathology results are shown in Table 3 and Figure 1. One (patient 1) had normal kidney function, whereas the remaining 3 (patients 2, 3, and 4) had decreased kidney function at the time of biopsy. Kidney biopsy was performed in patient 1 soon after the anorexia nervosa diagnosis, and in the remaining 3 patients, it was performed after an interval of 9 to 14 years. These 4 patients were lean and had various degrees of hypokalemia. The biopsy specimen of patient 1 showed hyperplasia of the juxtaglomerular apparatus and only mild tubulointerstitial damage (Fig 1A and B). Patient 2 had approximately 50% interstitial fibrosis and/or tubular atrophy and massive infiltration of lymphocytes into the interstitium (Fig 1C and D). Based on a diagnosis of diffuse tubulointerstitial nephritis of unknown origin, oral corticosteroid therapy was administered for 6 months.

Table 3. Clinical and Histopathologic Findings at Biopsy

	Case 1	Case 2	Case 3	Case 4
Clinical findings				
Age at biopsy, y	30s	30s	30s	20s
Reasons for renal biopsy indication	Elevated tubulointerstitial marker, hypokalemia	Unknown renal dysfunction	Unknown renal dysfunction	Rapidly progressing renal dysfunction, hypokalemia
Duration from development of an eating disorder to biopsy, y	0	9	14	10
BMI, kg/m²	17.6	13.9	14.6	14.9
BSA, m²	1.44	1.30	1.26	1.19
Serum creatinine, mg/dL	0.59	3.83	2.99	1.4
eGFR, mL/min/1.73 m²	94	13	16	40
Serum potassium, mmol/L	2.8	2.9	3.3	2.1
Serum magnesium, g/dL	2.1	2.3	1.8	5.2
pH	7.478	7.471	7.403	7.436
HCO_3^-, mmol/L	32.4	39.4	20.6	26.9
Histopathologic findings				
IF/TA, %	5	50	60	30
Calcification	NA	NA	NA	+
Cortex medulla ratio	9:1	9:1	8:2	10:0
Glomerular number	47	35	37	146
GGS, %	2	23	43	3
Hypertrophy of JGA	+	±	−	−

Note: Conversion factors for units: creatinine in mg/dL to μmol/L, ×88.4.

Abbreviations: BMI, body mass index; BSA, body surface area; eGFR, estimated glomerular filtration rate; GGS, global glomerular sclerosis; IF/TA, interstitial fibrosis and/or tubular atrophy; JGA, juxtaglomerular apparatus; NA, not available.

However, the therapeutic effect was not evident and creatinine level remained high (4-5 mg/dL) during the follow-up period. Patient 3 showed approximately 60% interstitial fibrosis and/or tubular atrophy (Fig 1E and F). Patient 4 underwent open kidney biopsy because the material obtained by percutaneous needle biopsy was insufficient to make a diagnosis. Light microscopy showed that 4 of 146 glomeruli were globally sclerotic and most glomeruli on the surface layer of the cortex were collapsed (Fig 1G and H). Interstitial fibrosis was observed predominantly in the subcapsular region. During the follow-up period, her decreased kidney function progressed slowly. This patient underwent a final examination in our department in her 40s, at which time her creatinine level was 3.4 mg/dL. In all 4 cases, no evidence of vascular or glomerular disease was identified.

DISCUSSION

We report clinical characteristics, kidney prognosis, and histopathologic findings of patients with anorexia nervosa treated by nephrologists. Median age at diagnosis with anorexia nervosa was 19.5 years, and that at the first visit to a nephrologist was 39.0 years, an interval of 20 or more years. At the first visit to a nephrologist, most patients showed various degrees of kidney disease, a small BSA, and hypokalemia. In patients with anorexia nervosa, eGFR and BMI have been reported to be significantly correlated,[10] implying that being extremely underweight promotes decreased kidney function.

Serum potassium levels were low in most patients included in this study. Hypokalemia is one of the most frequent and serious consequences of anorexia nervosa, with an estimated prevalence of approximately 20%.[16] In general, serum potassium levels increase as chronic kidney disease progresses. However, serum potassium levels are paradoxically decreased in patients with anorexia nervosa, even those with advanced kidney disease. Hypokalemia in anorexia nervosa may be due to a deficit in potassium intake, habitual vomiting, and abuse of diuretics or laxatives. The renin-angiotensin-aldosterone system is upregulated in response to a persistent decrease in serum potassium levels, which may further contribute to the maintenance of hypokalemia in anorexia nervosa. Magnesium levels were high in some patients with anorexia nervosa included in this study, especially patients with advanced kidney disease. In the presence of kidney disease, the use of magnesium-containing laxatives may be associated with a risk for hypermagnesemia.[17]

Four of the patients underwent kidney biopsy. Among them, a patient with preserved kidney function showed hypertrophy of the juxtaglomerular apparatus, but 3 patients with moderate to advanced kidney disease did not. Hypertrophy of the juxtaglomerular apparatus may reflect hyper-reninemia due to a long-term decrease in chloride concentration in the macula densa, suggesting that this patient had a condition involving loss of salt from the kidney.[18] Our findings indicate that over the long term such a state blunts the feedback mechanism that mediates histopathologic renormalization of the juxtaglomerular
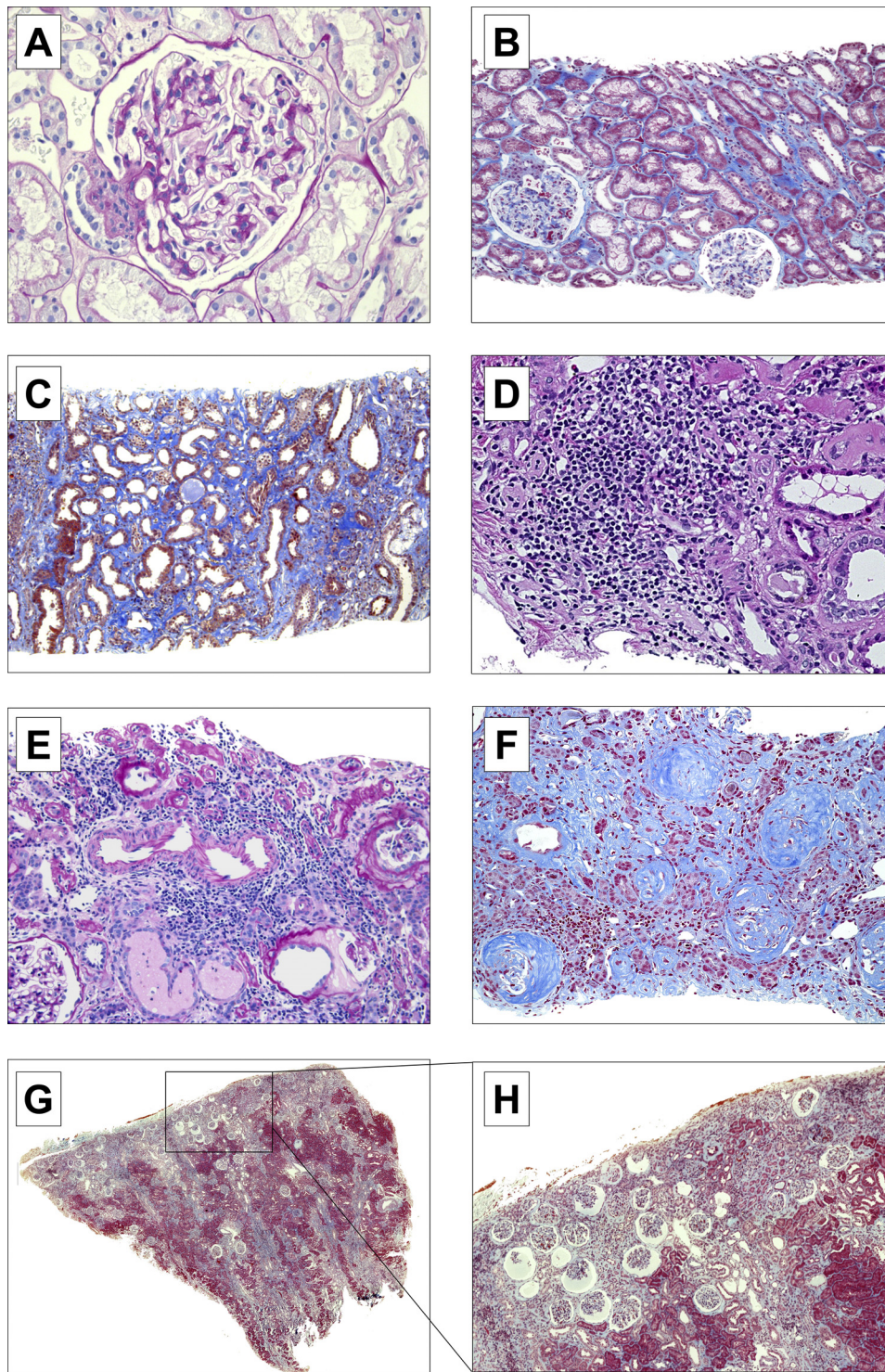
Figure 1. Kidney histopathologic findings. (A) Case 1: a woman in her 30s with normal kidney function exhibited hyperplasia of the juxtaglomerular apparatus (periodic acid–Schiff staining; original magnification, ×400). (B) Case 1: tubulointerstitium damage was mild (interstitial fibrosis and/or tubular atrophy < 5%). (C) Case 2: a woman in her 30s with severely decreased kidney function; interstitial expansion and inflammation were found (B, C: Masson trichrome staining; original magnification, ×100). (D) Case 2: most infiltrating cells in the tubulointerstitium were mononuclear cells (hematoxylin and eosin staining; original magnification, ×200). (E) Case 3: a woman in her 30s with severely decreased kidney function showed marked tubulointerstitial damage with cast formation and global glomerulosclerosis (periodic acid–Schiff staining; original magnification, ×100). (F) Most glomeruli showed global glomerulosclerosis of the obsolescence type (Masson trichrome staining; original magnification, ×100). (G) Case 4: a woman in her 20s with moderately decreased kidney function. Analyses of kidney biopsy specimens showed that most glomeruli on the surface layer of the renal cortex were collapsed (Masson trichrome staining; original magnification, ×50). (H) Case 4: enlargement of the boxed area in G (Masson trichrome staining; original magnification, ×100).

apparatus. A patient with kidney disease exhibited glomerular collapse or global glomerulosclerosis on the surface layer of the renal cortex together with the tubulointerstitial injuries. These findings are consistent with the notion that the kidney disease was caused by chronic ischemia and/or electrolyte disorders.

Persistence of chronic hypokalemia is hypothesized to cause tubulointerstitial damage, renal tubular fibrosis, atrophy, and cyst formation, but the causal relationship between chronic hypokalemia and anorexia nervosa has not been established. In an experimental model, chronic hypokalemia results in kidney ischemia and subsequent tubular injury due to accumulation of ammonium from urea.[19]

Global glomerulosclerosis is classified as the solidification or obsolescence type.[20] The former is caused by expansion of the mesangial matrices and relative condensation of the glomerular capillaries, while the latter reflects glomerular shrinkage and collapse due to ischemia. In this study, 3 of 4 patients showed marked global glomerulosclerosis, all of the obsolescence type. Patients with anorexia nervosa tend to repeatedly self-induce vomiting and abuse diuretics and laxatives due to extreme body dysmorphia. This can induce kidney ischemia, followed by chronic kidney injury and irreversible sclerotic and fibrotic changes in the kidneys.

Our findings have implications for daily clinical practice in nephrology. First, patients with anorexia nervosa should be referred to nephrologists at an earlier stage of kidney disease; that is, before irreversible kidney injury occurs. To this end, close collaboration between psychiatrists and nephrologists may be required. Second, education is required for patients with anorexia nervosa regarding the risks of abusing diuretics or laxatives and the need for adequate hydration to protect the kidneys from ischemic and/or hypokalemic injury. Third, a kidney biopsy should be considered in some patients with anorexia nervosa with kidney disease to confirm histopathologically that the kidney injury is induced by anorexia nervosa.

The strength of this study is that it is one of a few to focus on the clinical and histopathologic features of patients with anorexia nervosa from the viewpoint of nephrologists.[10,11] In addition, the long-term follow-up enabled us to compare patient characteristics according to kidney outcomes.

However, this study also had several limitations. First, this was a retrospective observational study in limited facilities and may have included various biases. Therefore, it is difficult to generalize our observations to all patients with anorexia nervosa. Large-scale studies of patients with anorexia nervosa, including those in the early stages of disease, are required.

Second, kidney function was assessed based on creatinine level, which may have led to overestimation of kidney function, because patients with anorexia nervosa have less muscle mass than healthy people. In future studies, kidney function should be evaluated using metrics not influenced by muscle mass, such as measured GFR or eGFR by cystatin C level. Although measured GFR is the gold standard for assessing kidney function, we could not obtain these data in most of our patients. Estimating GFR based on cystatin C level may be helpful as an alternative method because cystatin C is less affected by muscle mass than creatinine.[21]

Third, anorexia nervosa was diagnosed based on the medical records of psychiatrists, not using criteria in the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition*. Characteristics of all patients were consistent with those typical of anorexia nervosa, but we were unable to determine their anorexia nervosa subtypes (eg, the casual eating–excretion and restriction subtypes).

In conclusion, the 14 patients with anorexia nervosa in this study showed relatively severe and irreversible kidney disease at the first visit to a nephrologist. Therefore, improving kidney outcomes of patients with anorexia nervosa–associated kidney disease may require earlier intervention by nephrologists.

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