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GUIDELINES

Preface: evidence-based practice guideline for the treatment of chronic kidney disease

The concept of chronic kidney disease (CKD) was proposed in the United States in 2002 and quickly spread, becoming widely accepted in Japan. This concept includes broader pathological conditions and symptoms than those of the classical renal diseases. The growing number of CKD patients who advance to end-stage renal disease and need dialysis treatment is a serious concern in government health economics. Moreover, CKD patients are shown to be highly susceptible to cardiovascular disease (CVD), and their morbidity and mortality due to CVD is high. There is an increasing need for measures and treatments to be undertaken against CKD, and the Japanese Society of Nephrology (JSN) has taken a leading role in establishing them. At the end of 2006, the Scientific Committee of JSN decided to create an evidence-based practice guideline for the treatment of CKD. A working group consisting of 35 physicians, nephrologists, and other specialists was set up, and the members undertook a number of difficult tasks that included setting out the problems to be discussed, searching through the evidence, evaluating each piece of that evidence, make abstract tables, and issuing policy statements. After more than 2 years of hard work, the "Evidence-Based Practice Guideline for the Treatment of CKD" was completed and was published in March 2009. The guideline is intended for medical personnel in general, but its primary target is nephrologists. The therapeutic objectives are CKD patients as a whole; however, we have not included dialysis patients (CKD stage 5D). We are pleased to have created this first evidence-based guideline, published by JSN. We hope that the guideline contributes to better management and treatment of CKD patients and, ultimately, to reducing the number of CKD patients.

Sei Sasaki MD
Chairman, Scientific Committee
Japanese Society of Nephrology

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List of Contributors

Scientific Committee, Japanese Society of Nephrology

Chairman

Sei Sasaki	Department of Nephrology, Tokyo Medical and Dental University, Tokyo, Japan

Co-Chairman

Norishige Yoshikawa	Department of Pediatrics, Wakayama Medical University, Wakayama, Japan

Members

Takashi Akiba	Department of Blood Purification, Tokyo Women's Medical University, Tokyo, Japan
Kenjiro Kimura	Nephrology and Hypertension, Internal Medicine, St. Marianna University School of Medicine, Kanagawa, Japan
Daisuke Koya	Endocrinology & Metabolism, Kanazawa Medical University, Ishikawa, Japan
Hirokazu Okada	Department of Nephrology, Faculty of Medicine, Saitama Medical University, Saitama, Japan
Yoshiharu Tsubakihara	Department of Kidney Disease and Hypertension, Osaka General Medical Center, Osaka, Japan

Secretariat

Naoki Kashihara	Department of Nephrology, Kawasaki Medical School
Toshiki Moriyama	Health Care Center, Osaka University, Osaka, Japan

Evidence-based Practice Guideline for the Treatment of CKD Production group

Koichi Asahi	Department of Nephrology, Hypertension, Diabetology, Endocrinology and Metabolism, Fukushima Medical University School of Medicine, Fukushima, Japan
Michio Fukuda	Department of Cardio-Renal Medicine and Hypertension, Nagoya City University Graduate School of Medical Sciences, Nagoya, Japan
Nobuhito Hirawa	Department of Hemodialysis and Apheresis, Division of Nephrology and Hypertension, Yokohama City University Medical Center, Yokohama City University, School of Medicine, Yokohama, Japan
Yohei Ikezumi	Department of Pediatrics, Niigata University Medical and Dental Hospital, Niigata, Japan
Tsutomu Inoue	Department of Nephrology, Faculty of Medicine, Saitama Medical University, Saitama, Japan
Eiji Ishikawa	Department of Cardiology and Nephrology, Mie University Graduate School of Medicine, Mie, Japan
Kenji Ishikura	Department of Pediatric Nephrology and Department of Clinical Research, Tokyo Metropolitan Kiyose Children's Hospital, Tokyo, Japan
Shuichi Ito	Division of Pediatric Nephrology, National Center for Child Health and Development, Tokyo, Japan
Takafumi Ito	Division of Nephrology, Department of Internal Medicine, Shimane University Faculty of Medicine, Shimane, Japan
Eiichiro Kanda	Department of Nephrology, Tokyo Medical and Dental University, Tokyo, Japan
Yoshihiko Kanno	Department of Nephrology, School of Medicine, Faculty of Medicine, Saitama Medical University, Saitama, Japan
Akihiko Kato	Division of Blood Purification, Hamamatsu University School of Medicine, Shizuoka, Japan

Shoji Kagami	Department of Pediatrics, The Institute of Health Bioscience, The University of Tokushima Graduate School, Tokushima, Japan
Tetsuya Kawamura	Division of Kidney and Hypertension, Department of Medicine, The Jikei University School of Medicine, Tokyo, Japan
Masaki Kobayashi	Department of Nephrology, Tokyo Medical University Ibaraki Medical Center, Ibaraki, Japan
Hiroo Kumagai	Department of Nephrology, National Defense Medical College, Saitama, Japan
Hirofumi Makino	Department of Medicine and Clinical Science Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan
Seiichi Matsuo	Department of Nephrology, Nagoya University Graduate School of Medicine, Nagoya, Japan
Tetsuya Mitarai	Department of Nephrology and Hypertension Saitama Medical Center, Saitama Medical University, Saitama, Japan
Sonoo Mizuiri	Department of Nephrology, Toho University School of Medicine, Tokyo, Japan
Toshiyuki Nakao	Department of Nephrology, Tokyo Medical University, Tokyo, Japan
Masaomi Nangaku	Division of Nephrology and Endocrinology, The University of Tokyo School of Medicine, Tokyo, Japan
Ichiei Narita	Division of Clinical Nephrology and Rheumatology, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan
Shinichi Nishi	Blood Purification Center, Niigata University Medical and Dental Hospital, Niigata, Japan
Kosaku Nitta	Department of Medicine, Kidney Center, Tokyo Women's Medical University, Tokyo, Japan
Satoshi Sugiyama	Division of Nephrology Department of Internal Medicine, Fujita Health University School of Medicine, Aichi, Japan
Hiromichi Suzuki	Department of Nephrology, Saitama Medical University, Saitama, Japan
Yoshiki Suzuki	Health Administration Center, Niigata University, Niigata, Japan
Kota Takahashi	Division of Urology, Department of Regenerative and Transplant Medicine, Graduate School of Medical and Dental Sciences, Niigata University, Niigata, Japan
Yusuke Tsukamoto	Division of Nephrology, Shuwa General Hospital, Saitama, Japan
Yoshifumi Ubara	Nephrology Center, Toranomon Hospital Kajigaya, Kanagawa, Japan
Nobuyuki Ura	Department of General Internal Medicine, Teine Keijinkai Hospital, Sapporo, Japan
Takashi Wada	Division of Nephrology, Department of Laboratory Medicine, Kanazawa University, Ishikawa, Japan
Tsuyoshi Watanabe	Department of Nephrology, Hypertension, Diabetology, Endocrinology and Metabolism, Division of Medicine, Fukushima Medical University School of Medicine, Fukushima, Japan
Kunihiro Yamagata	Department of Nephrology, Institute of Clinical Medicine, Graduate School of Comprehensive Human Sciences, University of Tsukuba, Ibaraki, Japan

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Evidence-based Practice Guideline for the Treatment of CKD

Chapter 1: Diagnosis of CKD

• Definition of CKD and staging

1. Diagnosis of CKD (Grade A, consensus)
CKD is defined as either of the conditions listed below lasting for more than 3 months [a].
a. Findings suggesting kidney damage, i.e. abnormal findings in blood or urine tests, imaging studies or pathological evaluations.
b. GFR < 60 mL/min/1.73 m^2

2. Evaluation of GFR (Grade A, consensus)
a. The gold standard for GFR is inulin clearance [b].
b. Estimated GFR (eGFR) is calculated using the following Japanese formula: [1, a]
eGFR(mL/min/1.73 m^2)
$= 194 \times Cr^{-1.094} \times Age^{-0.287}$ ($\times 0.739$ if women)

3. Staging and management of CKD (Grade A, consensus)
CKD is diagnosed and staged either by kidney function (GFR) or abnormal laboratory findings. CKD should be properly managed on the basis of its stage [a].

• Albuminuria/Proteinuria

1. Collection and evaluation of urine samples (Grade A, Consensus)
Urinary protein/albumin is evaluated either by the dipstick for spot-urine or a quantitative evaluation for spot urine or a 24-h/timed collected urine. In the quantitative evaluation, the albumin/protein-to-creatinine (Cr) ratio is calculated (g per 1 g Cr) for a more precise evaluation [b]. Microalbuminuria is defined as an albumin-to-Cr ratio (ACR) of 30 to 299 mg/gCr [c, d]. To exclude orthostatic proteinuria, the first-morning urine sample should be evaluated for urinary protein at least once.

• Hematuria

1. Sampling and Evaluation
a. A mid-stream sample of either the first-void urine collected in the morning or spot-urine is evaluated by the dipstick test [e, f, g]. (Grade A, Consensus)
b. If occult blood yields a positive result with the dipstick test, the presence of red blood cells should be confirmed by microscopy of the urinary sediment. Deformity of red blood cells or cast formation suggests that the hematuria derives from glomerular lesions [h, i, j]. (Grade A, Consensus)

• Indication of renal biopsy

1. Biopsy for Isolated Proteinuria: Renal biopsy should be performed in patients with isolated and persistent proteinuria of more than 0.5 g/day or 0.5 g/gCr [k, l]. (Grade B, Consensus)
2. Biopsy for coexistence of both proteinuria and hematuria: If there is coexistence of both hematuria and proteinuria, a renal biopsy should be considered even if the amount of proteinuria is less than 0.5 g/day or 0.5 g/gCr [k, l]. (Grade A, Consensus)
3. Biopsy for isolated hematuria: When underlying progressive glomerulonephritis is suspected on the basis of abnormal urinary sediments, such as abnormal glomerular casts or a dysmorphic appearance of most red blood cells, a renal biopsy should be considered [k, l]. (Grade C, Consensus)

4. Biopsy for diabetic patients: Renal biopsy should be considered for diabetic patients, when coexistence with other glomerular diseases is suspected [2, m, n, o]. (Grade B, Consensus)

• Imaging studies in CKD

1. Imaging studies (i.e., abdominal computed tomography and ultrasonography) are recommended in patients with CKD to evaluate morphological abnormalities and complications (i.e., malignancy, urinary tract stones, infections, obstruction, vesico-ureteral reflux or polycystic kidney disease) [a]. (Grade A, Consensus)
2. To estimate GFR using radioisotope-labeled markers, a plasma/urinary sampling technique or a gamma camera positioned over the kidneys (quantitative renal imaging) is used. Quantitative renal imaging does not provide a precise determination of GFR, but has the advantage that renal imaging and the relative contribution of each kidney to total GFR can be obtained simultaneously [z]. (Grade B, Consensus)

Chapter 2: Significance of CKD

1. Decreased renal function and end-stage renal disease (ESRD)

a. Decreased renal function [1, 2] is a risk factor for ESRD [3–7]. (Grade A, evidence level 4)
b. In the Japanese population, the average rate of GFR decline is 0.36 mL/min/1.73 m^2/year. The rate is significantly higher in people aged 40–69 years with an initial GFR < 50 mL/min/1.73 m^2 and in people aged 70–79 years with an initial GFR < 40 mL/min/1.73 m^2 [8]. (Grade A, evidence level 4)

2. Proteinuria and ESRD

a. Proteinuria and albuminuria are risk factors for ESRD. Increased levels of proteinuria and albuminuria are associated with a higher risk of ESRD [9–16]. (Grade A, evidence level 1)
b. Reduction of proteinuria or albuminuria by treatment is associated with slowing the progression of renal disease. (Grade A, evidence level 2)

3. Hematuria and ESRD
A positive dipstick test result for hematuria is a risk factor for ESRD. The relation between the risk of ESRD and the degree of hematuria is weak compared with proteinuria. The presence of both proteinuria and hematuria is strongly associated with developing ESRD [10]. (Grade A, evidence level 4)

4. CKD and cardiovascular disease (CVD)

a. Reduced GFR is a risk factor for cardiovascular disease (CVD) [3, 17–34]. (Grade A, evidence level 1)
b. Proteinuria and albuminuria are risk factors for CVD [17–20, 22, 24, 28, 30–34]. (Grade A, evidence level 1)
c. Reduction of proteinuria or albuminuria by treatment is associated with reducing cardiovascular events [35–37]. (Grade A, evidence level 2)

5. Frequency of CKD in Japan
The frequency of CKD in Japanese adults is 0.6, 1.7, 10.4 and 0.2%, for stage I, II, III and IV plus V, respectively. The total number of patients is estimated to be approximately 10.97 million for stage III to V. Therefore, a comprehensive package of measures against CKD is required. (Grade A, evidence level 4)

Chapter 3: CKD and Life-style

Statements

1. Smoking (Grade A, Level 4)
Smoking is an independent risk factor for the development [1–10] and progression of CKD and also increases the risk of emergence of CVD [11–13]. Therefore, patients with CKD should quit smoking.

2. Alcohol consumption (Grade B, Level 4)
Moderate intake of alcohol, defined as 20–40 g of ethanol per day, correlates with a decreased risk of progression of CKD [2] and emergence of CVD [13]. On the other hand, patients with CKD should avoid heavy drinking, defined as the consumption of ethanol equal to or more than 60 g per day, which increases the risk of CKD and CVD [14].

3. Physical activity and exercise

a. Maintenance of physical activity (Grade A, Consensus)
Physical rest or restriction of physical activity is not recommended for all patients with CKD. Maintenance of adequate physical activity contributes to weight control, diabetes prevention, blood pressure control, and CVD prevention.
b. Exercise intensity (Grade B, Level 3)
There is no evidence that moderate intensity exercise (about 5 METs) leads to the progression of CKD. Regular exercise is recommended for CKD patients without severe complications [15, 16].

4. Vaccination (Grade B, Level 4)
Influenza vaccination is recommended for patients with CKD [17].

5. Cancer screening (Grade B, Consensus)
Cancer screening in patients with CKD is recommended to the same degree as in the general population. False-positive results should be ruled-out when tumor markers are evaluated.

Chapter 4: CKD and nutrition

Statements

1. Diet therapy (Grade A, Consensus)
   Continual diet therapy is recommended and should be customized to the life-style of each patient with CKD. The dietician should help patients to plan and improve their diet.

2. Decision of estimated energy requirement (Grade A, Consensus)
   The estimated energy requirement of patients with CKD should be continuously controlled, based on the Japanese average basal metabolic rate, daily physical activity, and the nutritional status of each patient [a].

3. Protein intake restriction (Grade B, Level 1)
   For patients with CKD stage 3 to 5, dietary protein restriction should be considered to control the progression of renal dysfunction [1–24].

4. Sodium intake restriction (Grade A, Level 2)
   For patients with CKD, daily salt intake should be less than 6 g of sodium chloride [25–28].

Chapter 5: Hypertension and CVD in CKD

Statements

1. Hypertension in CKD (Grade A, Level 1)
   Hypertension is both a cause and a complication of CKD. Hypertension is a risk factor for the progression of CKD and emergence of CVD [1, 2]. The goals of antihypertensive therapy in CKD are to slow the progression of CKD, and to reduce cardiovascular morbidity and mortality [3].

2. Measurement of blood pressure in patients with CKD (Grade A, Level 4)
   In addition to office blood pressure, self-measurement of blood pressure at home in the morning and before going to bed at night is preferable for the management of hypertension in CKD [4, 5].

3. Target blood pressure for patients with CKD (Grade A, Level 1)
   The target blood pressure for patients with CKD and proteinuria of less than 1 g/day is <130/80 mmHg, and <125/75 mmHg for patients with CKD and proteinuria equal to or more than 1 g/day [3, 6, 7].

4. Choice of antihypertensive drugs
   a. Blockers of the renin–angiotensin system (ACE inhibitor or angiotensin II receptor blocker) are preferred as the first-line of antihypertensive therapy [8, 9]. (Grade A, Level 1)
   b. Either diuretics or calcium channel blockers are encouraged as the agents of second choice to achieve the blood pressure goals [10, 11]. (Grade A, Level 2)

5. Association between CKD and CVD
   a. CKD independently increases the risk of CVD including stroke [12–18]. (Grade A, Level 2)
   b. Reduction of proteinuria to the lowest achievable level should be considered as a goal, since proteinuria, even at microalbuminuric levels, is a strong predictor of future cardiovascular events [13, 14, 19–22]. (Grade A, Level 2)

Chapter 6: Renal anemia

Statements

1. Concept of renal anemia (Grade A, Level 4)
   In the early stage of CKD, erythropoietin production in the kidney starts to deteriorate, and renal anemia develops [1]. Therefore, early diagnosis by means of periodic examinations is recommended.

2. Importance of treatment for renal anemia (Grade A, Level 2)
   Hypodermic injection with ESA should be started at an early stage, since treatment for renal anemia with ESA (erythropoiesis stimulating agent) is effective in preventing the emergence of various CKD-related complications [2–8, 11–14].

3. Target of treatment for renal anemia
   a. The target Hb level of CKD independent of renal replacement therapy is equal to or more than 11 g/dL. ESA administration should be initiated when the Hb levels are repeatedly found to be less than 11 g/dL [13–15]. (Grade B, Level 1)
   b. Excessive improvement of renal anemia is associated with deterioration of the prognosis for survival [13–15] possibly due to the adverse effects of high-dose ESA administration [16]. Therefore, ESA administration should be reduced or terminated when Hb levels reach a level of more than 13 g/dL. The upper limit of Hb levels should be 12 g/dL for CKD patients with CVD or those with other medical problems. (Grade A, Level 1)

4. Iron supplementation (Grade B, Consensus)
   For CKD patients, oral administration of iron is recommended at first, and if insufficient, intravenous injection of iron should be administered, independent of renal replacement therapy.

Chapter 7: Mineral and bone disorder in CKD

Statements

1. Concept of mineral and bone disorder in CKC (CKD-MBD) (Grade A, Consensus)
   CKD-MBD should be managed as a systemic disorder, which affects mortality due to CVD in addition to renal osteodystrophy [a].

2. Control of serum calcium and phosphorus levels (Grade C, Level 4)
   Elevated serum phosphorus levels are associated with the progression of renal dysfunction and mortality of patients with CKD [1, 2]. However, the optimal levels of serum calcium and phosphorus have not been definitively established for patients with CKD other than those with CKD stage 5D.

3. Treatment of secondary hyperparathyroidism in CKD-MBD (Grade C, Level 2)
   It has not been proved that treatment of secondary hyperparathyroidism with activated vitamin D compounds prevents the progression of renal dysfunction [3–8], but it might improve the mortality [9] of patients with CKD stage 3 to 5 (except for 5D).

Chapter 8: Diabetic Nephropathy

1. Diagnosis
   All patients with diabetes should be screened annually for diabetic kidney disease. Screening should include measurements of the urinary albumin–creatinine ratio (ACR) in a spot-urine sample, serum creatinine and eGFR. (Grade A, Consensus)

2. Treatment

a. Blood glucose control

- Intensive treatment of hyperglycemia prevents diabetic kidney disease and may slow the progression of established kidney disease. The target HbA1c for people with diabetes should be <6.5% [1–4]. (Grade A, Level 2)
- Concomitant with decreased kidney function (CKD stage 3 to 5), decreased clearance of the sulfonylureas or their active metabolites necessitates a decrease in drug dosing to avoid hypoglycemia. Patients with CKD stages 4 or 5 should be treated with insulin. (Grade B, Consensus)

b. Blood pressure control

- Blood pressure in diabetic patients should be strictly controlled to 130/80 mmHg or less using antihypertensive drugs, including ACEI or ARB as a first-line drug [5–27. (Grade A, Level 1)
- If urinary protein excretion is more than 1 g/day (or g/gcr), the target mean blood pressure should be 92 mmHg (125/75 mmHg) or less [16]. (Grade B, Level 2)
- In order to suppress the progression of nephropathy, preferably ACEI or ARB should be prescribed to diabetic patients even in cases with normal blood pressure [22, 23, 28–35]. (Grade B, Level 2)

c. Diet

- A low protein diet possibly retards the progression of overt diabetic nephropathy (CKD stage 3 to 5) [36–42]. (Grade C, Level 2)
- A low salt diet should be prescribed in patients with hypertension regardless of stages. (Grade B, Consensus)

d. Multidisciplinary approach

- In type 2 diabetic patients, early-stage diabetic nephropathy is retarded by a multidisciplinary approach, including strict blood glucose control, strict blood pressure control, administration of ACEI or ARB, management of dyslipidemia by using an HMG-CoA reductase inhibitor, low-dose aspirin, anti-oxidative drugs, physical exercise, a program to help patients stop smoking etc. [43, 44]. (Grade B, Level 2)

Chapter 9: IgA nephropathy

Statements

1. General principle (Grade A, Consensus)
   IgA nephropathy is a progressive renal disease, which in approximately 20% of cases finally leads to end-stage renal disease within 20 years [1]. Aggressive immunosuppressive treatment is recommended at an early stage among patients with one of the predictive risk factors for a poor prognosis, such as systolic hypertension, massive proteinuria, elevation of serum creatinine or advanced histological alterations observed in renal biopsies [2–4].

2. Renin–angiotensin system inhibitors (Grade A, Level 2)
   ACEI reduces proteinuria and suppresses the deterioration of renal function, therefore IgA nephropathy patients with hypertension should be treated with ACEI as the first-line of antihypertensive therapy [5, 6].

3. Oral steroid therapy (Grade B, Level 1)
   Oral steroid therapy suppresses the deterioration of renal function in IgA nephropathy cases at CKD stage 1 or 2 and with proteinuria equal to or more than 1 g per day [7, 8].

4. Steroid pulse therapy (Grade B, Level 2)
   Among patients with IgA nephropathy at CKD stage 1 to 3 and with proteinuria ranging from 1 to 3.5 g per

day, steroid pulse therapy reduces proteinuria and suppresses the deterioration of renal function [9, 10]

5. Tonsilectomy plus steroid pulse therapy (Grade C, Level 3)
   In patients with IgA nephropathy, tonsilectomy plus steroid pulse therapy may be able to reduce proteinuria and suppress the deterioration of renal function [11–13].

6. Anti-platelet drugs (Grade C, Level 1)
   Although anti-platelet drugs reduce proteinuria in the short term, its long-term effect on renal function has not been elucidated [14].

Chapter 10: Nephrotic syndrome (Idiopathic membranous nephropathy, and primary focal segmental glomerulosclerosis)

Statements

1. Principle of therapy for idiopathic membranous nephropathy (Grade A, Consensus)
   Immunosuppressive therapy that aims for the induction of remission is recommended in patients who have risk factors for progression to renal failure including male sex, age of more than 60 years at onset, renal insufficiency at presentation, glomerular sclerotic lesion, tubulointerstitial lesion, and sustained nephrotic syndrome [a].

2. Immunosuppressive therapy for idiopathic membranous nephropathy
   a. Steroid monotherapy is used to induce remission in patients with idiopathic membranous nephropathy. (Grade C, Consensus)
   b. Treatment with cyclosporin in combination with low-dose steroid is used to induce remission. (Grade A, Consensus)
   c. Oral administration with cyclophosphamide in combination with steroid is effective for the induction of remission [1–8]. (Grade B, Level 1)
   d. In patients with cyclophosphamide-resistant idiopathic membranous nephropathy, treatment with cyclosporin in combination with low-dose steroid is effective for the induction of remission [9]. (Grade A, Level 2)

3. Steroid and immunosuppressive therapy for primary focal segmental glomerulosclerosis
   a. As the initial therapy, steroid therapy (using a prednisolone dose of 0.5–2 mg/kg/day) that aims at the induction of remission is used for primary focal segmental glomerulosclerosis. In steroid-responsive patients, prolonged therapy (of at least 6 months) is recommended [10–12]. (Grade B, Level 4)
   b. As initial therapy or second-line therapy for steroid-resistant patients, a prolonged course (for at least 6 months) of treatment with cyclosporin in combination with low-dose steroid is effective for the induction of remission and preserving the filtration function [13–16]. (Grade B, Level 1)

Chapter 11: Hypertensive nephrosclerosis

Statements

1. Effect of antihypertensive therapy (Grade A, Level 2)
   Strict management of blood pressure is important to inhibit the progression of renal dysfunction in patients with nephrosclerosis [1–5].

2. Significance of proteinuria (Grade A, Level 2)
   A decrease in urinary protein excretion resulting from antihypertensive therapy is related to a preventive effect against the progression of renal dysfunction in patients with nephrosclerosis [3].

3. Target of blood pressure control and choice of antihypertensive drugs (Grade A, Level 2)
   The target of blood pressure control should be less than 130/80 mmHg to inhibit the progression of renal dysfunction in patients with nephrosclerosis, regardless of the urinary protein level [1–5]. For patients with overt proteinuria, an ACEi ARB is the agent of first choice [1, 6–8]. However, the superiority of renin–angiotensin system inhibitors over other antihypertensive agents has not yet been proved for patients without overt proteinuria [1, 8].

Chapter 12: Atherosclerotic renal artery stenosis

Statements

1. Screening tests for renal artery stenosis (Grade A, Consensus)
   CKD may occur in combination with ischemic nephropathy, renovascular hypertension, or both. Clinical clues to the diagnosis of renal artery stenosis include severe or resistant hypertension and acute deterioration of renal function after the administration of a renin-angiotensin system inhibitor, which necessitate screening tests for renal artery stenosis.

2. Non-invasive examinations for renal artery stenosis (Grade A, Level 1)
   Renal duplex ultrasonography, magnetic resonance angiography, or computed tomographic angiography are recommended as screening tests to render the diagnosis of renal artery stenosis [1, 2].

3. Invasive examinations for renal artery stenosis (Grade A, Level 1)
   When the clinical index of suspicion is high and the results of noninvasive tests are inconclusive, catheter angiography (aortography or selective renal arteriography) is recommended as a diagnostic test to render the diagnosis of renal artery stenosis [3].

4. Antihypertensive therapy for renal artery stenosis (Grade A, Level 1)
   Antihypertensive therapy for hypertension associated with atherosclerotic renal artery stenosis is effective for inhibiting the progression of renal dysfunction [4–9].

5. Revascularization for renal artery stenosis (Grade A, Level 1)
   The antihypertensive effect of combination therapy using antihypertensive drugs with percutaneous transluminal renal angioplasty (PTRA) is better than that of antihypertensive drug therapy alone. However, evidence for a better kidney function-preserving effect of combination therapy using antihypertensive drugs with PTRA than that of antihypertensive drug therapy alone is inconclusive to date [4–9].

Chapter 13: Autosomal-dominant polycystic kidney disease (ADPKD)

1. Antihypertensive therapy (Grade A, Level 2)
   Hypertension is a risk factor for the progression of renal dysfunction in patients with ADPKD. Sufficient blood pressure control to less than 130/80 mmHg has been proved to inhibit such progression in patients with ADPKD equivalent to CKD stage 3 to 5 [1].

2. Protein intake restriction (Grade C, Level 2)
   Protein intake restriction has not been proved to inhibit the progression of renal dysfunction in patients with ADPKD [2, 3].

3. Cyst infection (Grade C, Consensus)
   For the initial empiric treatment for cyst infection, fluoroquinolones may be advantageous due to their effectiveness against gram-negative bacteria and better penetration into the renal cyst.

4. Care for other complications in patients with ADPKD (Grade C, Consensus)
   Screening for intracranial aneurysms (ICA) may be beneficial because ICA rupture in patients with ADPKD significantly influences their mortality. Fenestration of renal cysts or transcatheter arterial embolization of the renal arteries would shrink enlarged kidneys and may improve the quality of life of patients with ADPKD.

Chapter 14: Management of Dyslipidemia in CKD

1. Dyslipidemia in CKD is an important risk factor for the development of CVD [12, 13] as well as for the progression of CKD [1–11]. (Grade A, Level 4)

2. The target LDL-C value is recommended to be less than 120 mg/dl (if possible, less than 100 mg/dl) when managing dyslipidemia in CKD. When the LDL-C value does not reach the target, despite appropriate lifestyle modifications, drug treatment should be started. (Grade A, Consensus)

3. Administration of HMG-CoA reductase inhibitor of CKD is expected to prevent the development of CVD [23–27] as well as the progression of CKD [14–22]. (Grade A, Level 4)

Chapter 15: Obesity and Metabolic syndrome

1. Obesity is a risk factor for proteinuria [1–7] and end-stage renal disease (ESRD) [8–14]. (Grade A, Level 4)

2. Abdominal adiposity is a risk factor for microalbuminuria [15–17]. (Grade B, Level 4)

3. Metabolic syndrome is a risk for microalbuminuria, overt proteinuria and kidney dysfunction [18–30]. (Grade A, Level 4)

4. Initial therapy for the control of metabolic syndrome is to correct life-style-related factors. Improvement of obesity by dietary and/or exercise interventions decreases proteinuria [31–34]. (Grade A, Level 2)

5. In cases of CKD with the metabolic syndrome, constitutive factors such as hypertension, dyslipidemia and diabetes mellitus should be managed according to the statements of each chapter. (Grade A, Consensus)

Chapter 16: Diagnosis of CKD in childhood

Statements
General remarks

(1) Diagnosis of CKD in childhood (Grade A, Consensus)
   The diagnosis of CKD in childhood is based on the presence of renal disease and evaluation of renal function [1, a–e]. Renal imaging and family history are also important for the diagnosis of CKD in childhood [d, 1].

(2) Classification of stages of childhood CKD (Grade B, Consensus)

In this first version, classification of stage in adult CKD is also adopted for children. However, it should be used for children at 2 years of age or older, when the renal function becomes approximately comparable to that of adults [1, d].

(3) Normal range of renal function in children (Grade A, Level 4)
The normal ranges of GFR and serum level of creatinine in children vary with age and gender [2–5].
It is recommended that the glomerular filtration rate (eGFR) in children is estimated using Schwartz's formula [6, 7].

(4) Diseases responsible for childhood CKD and epidemiology (Grade A, Level 4)
In comparison with adults, the incidence of childhood CKD is rare. Most childhood CKD cases are classified as stage 1. On the other hand, the most predominant cause of childhood CKD classified as stages 2–5 is congenital renal disease [d, 1, 8–12]. Furthermore, the most frequent causative diseases of end-stage renal failure (CKD stage 5) are congenital anomalies of the kidney and urinary tract. The second predominant cause of CKD stage 5 in childhood is glomerular disease, such as focal segmental glomerulosclerosis (FSGS), whereas life-style related diseases are very rare.

(5) Three-year-old and school urinary screening tests are opportunities to diagnose childhood CKD (Grade B, Level 4)
Many glomerular diseases are diagnosed as CKD stage 1 by 3-year-old/school urinary screening. Early detection and treatment of glomerular diseases have been demonstrated to contribute to decreasing the frequency and improving the prognosis of childhood CKD [13, 14].

Specific remarks
a. Examination

(1) Proteinuria (Grade A, Consensus)
Proteinuria is important in the diagnosis of CKD [a–e]. For the screening of children without diabetes for CKD, urinary protein should be measured using a first morning urine sample by either a urine dipstick or the urinary protein-to-creatinine ratio [15–17, a–e]. The urinary protein-to-creatinine ratio obtained from a first morning urine sample should be used to follow-up childhood CKD [15–17, a–e]. When a low level of proteinuria persists, low molecular proteinuria should be examined in order to exclude tubular proteinuria.

(2) Urinary protein-to-creatinine ratio (Grade A, Level 4)
The normal range of urinary protein-to-creatinine ratio is as follows [15].
Two years or older: less than 0.2 g/gCr
Under 2 years: less than 0.5 g/gCr

(3) Orthostatic (postural) proteinuria (Grade A, Consensus)
Orthostatic (postural) proteinuria must be excluded by measurement of a first morning urine sample [c]. The prognosis of orthostatic (postural) proteinuria is good [18]. For an accurate evaluation of a first morning urine sample, the patient should urinate just before going to bed, and urine sampling must be carried out just after getting up in the morning. With midstream urine sampling, it is essential for the patient to comply fully with these three points.

(4) Nephrotic syndrome (Grade A, Consensus)
Nephrotic syndrome is a clinical condition defined by heavy proteinuria and hypoalbuminemia. It is diagnosed using the criteria of the international study group for kidney disease in children (ISKDC) [f].

(5) Hematuria (Grade A, Consensus)
Hematuria is important in the diagnosis of CKD [c]. Red blood cells in the urine should be examined in children with CKD and those at risk for CKD [c, g]. Readers are recommended to refer to the adult section for the definition of hematuria.

(6) Renal biopsy (Grade A, Consensus)
Renal biopsy is important in the diagnosis of CKD. For the following cases, a renal biopsy is recommended [h].
1) Persistent proteinuria (urinary protein-to-creatinine ratio: ≥0.5 g/g, ≥3 months; aged 2 years or older)
2) Persistent hematuria + proteinuria (hematuria + urinary protein-to-creatinine ratio: ≥0.2 g/g, ≥3 months; aged 2 years or older)
3) Nephrotic syndrome: unlike adults, renal biopsy is not indicated in most children with nephrotic syndrome.
The following cases are exceptional in childhood nephrotic syndrome, and renal biopsy is recommended: cases in whom underlying diseases other than minimal change are suspected; cases who are suspected of congenital nephrotic syndrome; and cases with steroid-resistant nephriotic syndrome.
4) Rapidly progressive glomerulonephritis syndrome
5) Systemic lupus erythematosus (SLE)
6) Henoch-Schönlein purpura nephritis
Nephrotic syndrome, acute nephritic syndrome, rapidly progressive glomerulonephritis syndrome and cases with persistent proteinuria.

(7) Diagnosis of congenital anomalies of the kidney and urinary tract (Grade A, Consensus)
Hematuria and/or proteinuria are not common manifestations in congenital anomalies of the kidney and urinary tract. It is often accidentally diagnosed by

fetal/neonatal echography, and when urinary tract infection occurs [1, 19]. CKD stage 2–3 or more is often diagnosed by symptoms of chronic renal failure, such as failure to thrive, general fatigue or polyuria.

(8) Diagnostic imaging and renal function tests (Grade A, Consensus)
Diagnostic imaging (abdominal echography, several scintigraphic examinations, MRI and etc.) is important in the diagnosis of childhood CKD[1, 19] and is particularly useful in the following diseases.

1) Obstructive nephropathy, 2) reflux nephropathy, 3) dysplastic/hypoplastic kidney, 4) solitary kidney, horseshoe kidney, floating kidney, and 5) cystic kidney disease.

b. Childhood CKD as a risk factor of cardiovascular disease, failure to thrive or progression to CKD stage 5

(1) Childhood CKD as a risk factor of cardiovascular disease (CVD) (Grade A, Level 4)
Like adults, childhood CKD is a risk factor of cardiovascular disease (CVD) [12, 20–23]. Periodical evaluation of cardiac function and treatment of hypertension are important in the management of childhood CKD.

(2) Childhood CKD as a risk factor of failure to thrive (Grade A, Level 4)
Childhood CKD is a risk factor for failure to thrive. Short stature is a significant problem of childhood CKD [24, 25].

(3) Childhood CKD as a risk factor of progression to CKD stage 5 (Grade A, Level 4)
Like adults, childhood CKD is a risk factor of progression to CKD stage 5. In particular, proteinuria [26–38] and low GFR [9, 39–42] are risk factors of progression to CKD stage 5.

Chapter 17: Management of CKD in children

Statements
General remarks

The patients included in these management guidelines are children under 15 years who are still growing and with stage 1 to 4 chronic kidney disease (CKD). Many factors, including the issue of carry-over, the patients' age and build, medication compliance, and appropriate timing of the transfer to adult CKD guidelines, should always be considered in the management of such patients.

(1) Basic considerations (Grade A, Consensus)
The objectives of CKD management are to decrease the progression of CKD and to prevent cardiovascular events and lifestyle-related diseases. In children with CKD, the maintenance of normal growth and the prevention of growth failure are other essential objectives. Moreover, it should be feasible to carry out the management process in children.
It is desirable that the management of children with CKD should be conducted by experienced pediatric nephrologists to ensure that the parents of CKD patients are provided with sufficient information related to the treatment and pathophysiology of CKD.
Cooperation with a pediatric urologist is essential for the management of children with CKD because of the high prevalence of congenital urinary tract anomalies in these patients [1, 2].

(2) Educational guidance for the management of lifestyle factors and nutrition (Grade B, Consensus)

1) Exercise (Grade B-level 4)
The level of exercise should be limited in patients with circulatory disturbances, those treated with anticoagulant agents, or those whose renal function is aggravated by exercise [3, a, b].
For children with CKD who have been undertaking long-term medication and recuperation, the physical strength of the patient and their exercise intentions should be considered.

2) Management of obesity (Grade A, level 2)
For school children with CKD, careful attention should be paid to avoiding excessive salt and lipid intake to prevent obesity and hypertension [4, 5].
For children with CKD who are already showing signs of obesity or hypertension, consumption of snacks and high-salt processed foods should be limited, and regular physical activity that can be achieved over the long-term, such as swimming, walking, jogging or cycling, is recommended [6].

3) Caloric intake (Grade A, level 4)
For infants with CKD that is attributed to a congenital urinary tract disorder, the target calorie intake should be equivalent to the estimated energy requirement of Japanese infants [7–9].
For children with CKD that is complicated with nutritional disturbances, tube-feeding should be considered [9].
For children with CKD of school age and with signs of obesity, the calorie intake should be restricted to prevent the exacerbation of obesity and hypertension [4, 5].

4) Dietary protein (Grade B, level 1)
For children with CKD, reduced dietary protein intake is not currently recommended [10]. However, excessive intake of a high protein diet should be avoided to prevent hyperphosphatemia.

5) Dietary salt (sodium) (Grade A, level 4)
For school children and adolescents who are also obese or show signs of hypertension, it is strongly

recommended that their dietary salt intake be restricted to below 6 g/day, as for adult patients.

For patients with advanced-stage disease or those with hypertension, edema or cardiac hypertrophy caused by fluid overload, the dietary salt intake should be restricted more strictly.

Meanwhile, children with CKD attributed to a congenital anomaly of the urinary tract frequently present with salt-losing nephropathies, and therefore require salt repletion. For these patients, the urinary volume and sodium excretion should be monitored carefully and adequate supplementation of salt and fluid should be achieved [11].

6) Lipids

For children with CKD, there is currently no evidence that restricting lipid intake slows renal failure progression, and is, therefore, currently not recommended. (Grade B, Consensus)

For adolescent patients at the early stage of CKD accompanied by obesity or hypertension, excessive intake of lipids should be avoided to improve the patients' general conditions [4, 5, 12]. (Grade B, level 4)

7) Vaccination (Grade A, level 4)

CKD children should actively receive vaccinations to avoid infections [13–16]. (Grade A, level 4)

CKD children who are at an advanced stage or patients taking a large dose of glucocorticoids (≥2 mg/kg/day) should avoid vaccination [14–16, c]. (Grade A, level 4)

Indications for vaccination should be assessed in individual patients according to the patient's condition, the prevalence of infectious diseases at the time, and the type of vaccine (live or inactivated) [17, c]. (Grade B, level 4)

(3) Management of complications

1) Cardiovascular hypertension (Grade A, level 3)

The definition of hypertension in children with CKD is summarized in Table 2. Hypertension or edema due to a fluid overload should be treated with loop diuretics (furosemide 0.5–2.0 mg/kg/day) and the salt intake should be restricted [18].

Hypertension in children with early-stage CKD with accompanying obesity should be managed with exercise and dietary modifications. Children with an inadequate response to lifestyle modification should be administered pharmacologic therapy [18–23].

2) Mineral and bone disorders (Grade B, level 4)

Children with CKD at stage 2 or greater should be managed to maintain levels of serum calcium (Ca) and phosphorous (P) within the normal range to prevent renal osteodystrophy. The Ca·P product should be maintained at <60 mg^2/dl^2 in children and <65 mg^2/dl^2 in infants, and the intact PTH level should be ≤150 pg/ml [24–26, d].

For infants, the use of phosphorus binding agents and/or low-phosphorous milk should be considered [27].

3) Management of anemia (Grade B, level 4)

Children with CKD with a serum hemoglobin (Hb) level of 80% or lower than the normal range for their age should start treatment with iron agents and erythropoiesis-stimulating agents (recombinant human erythropoietin [rhEPO]) to achieve an Hb ≥ 11 g/dl [27, 28.

Oral iron therapy should be managed using doses of elemental iron ranging from 2 to 3 mg/kg/day, with a maximum dose of 6 mg/kg/day. Adequate iron levels are indicated by a ferritin level ≥100 ng/ml and transferrin saturation ≥20% [29–31, e, f].

A starting dose of rhEPO of 50 IU/kg/week administered subcutaneously is appropriate for children with non-dialysis dependent CKD. The target is to increase the Hb level at a rate of 1 g/dl every 4 weeks by increasing the dose of rhEPO by 50 IU/kg/week. Because rhEPO can increase the blood pressure, blood pressure should be monitored in patients treated with rhEPO to avoid hypertension [29–34].

4) Growth failure (Grade B, level 1)

Prepubertal children with CKD at stage 3 or higher with accompanying growth retardation should start treatment using recombinant human growth hormone (rhGH) [35–37].

Specific remarks

(1) Treatment for CKD stage 1

a. Nephrotic Syndrome (including FSGS)

1) Background (Grade A, Consensus)

Steroid therapy should be initiated without histological confirmation by renal biopsy because most idiopathic pediatric nephrotic syndrome (NS) patients respond well to steroids. However, a kidney biopsy and histological diagnosis are recommended before starting steroid therapy in the following cases: 1) Age younger than 1 year; 2) Having the following findings: apparent hematuria, hypertension or elevated serum creatinine levels; 3) Having extra-renal symptoms, such as rash or purpura [38, 39, g].

2) Initial treatment for NS (Grade A, level 2)

Start treatment with the standard initial treatment regimen (International Study of Kidney Diseases in Children; ISKDC method) or a longer initial regimen. Most patients are responsive to these therapies.

Standard regimen: (Grade A, level 2)

Oral prednisone at the dose of 60 mg/m^2 per day for the first 4 weeks followed by 4 weeks of prednisone of 40 mg/m^2 on alternate days [38–40, h].

Longer regimen: (Grade A, level 2)

Oral prednisone at the dose of 60 mg/m^2 per day for the first 4 weeks followed by alternate day treatment lasting for 2–6 months [41–43].

2) Treatment for relapsing NS (Grade B, level 4)
Oral prednisone at the dose of 60 mg/m^2 per day is continued for 3 days after the proteinuria disappears followed by 4 weeks of alternate day prednisone of 40 mg/m^2 according to the ISKDC method [38, g, h].

4) Treatment for frequent relapsing/steroid-dependent NS
Cyclosporine or cyclophosphamide is effective in maintaining remission in patients with frequent relapsing or steroid-dependent NS, although cyclophosphamide for steroid-dependent NS is still controversial.

1. Cyclosporine (Grade A, level 1)
2. Cyclophosphamide (Grade A, level 2)

5) Steroid-resistant NS
The efficacy of cyclosporine and/or steroid pulse therapy has been confirmed in a proportion of steroid-resistant NS cases. Patients who respond to these therapies have a relatively good prognosis.

1. Cyclosporine [68–71, h] (Grade A, level 2)
2. Steroid pulse therapy [44, 45, g] (Grade A, level 4)

b. IgA nephropathy

1) Background (Grade A, level 5)
Severe IgA nephropathy is histologically defined as showing diffuse mesangial proliferation, and mild IgA nephropathy is defined as focal mesangial proliferation [46]. The optimal approach to the treatment of IgA nephropathy should be determined by the histological findings.

2) Treatment for mild IgA nephropathy (Grade A, level 4)
ACE inhibitor and/or ARB treatment are recommended. These pharmacologic therapies reduce urinary protein excretion and slow the progression of IgA nephropathy [47–49, i].

2) Treatment for severe IgA nephropathy (Grade A, level 2)
Combined therapy with prednisone, azathioprine, heparin–warfarin and dipyridamole for 2 years is recommended. Both an antiproteinuric effect and pathological improvement can be observed in patients treated with this regimen [50–52, i, j].

(2) Treatment for CKD stage 2–4 (Grade A, level 4)
ACE inhibitor and/or ARB treatment for pediatric patients with CKD stage 2–4 due to chronic nephritis show antihypertensive and antiproteinuric effects. This therapy also can be expected to slow the progression of kidney damage as in adults. However, this treatment for CKD derived from congenital dysplastic or hypoplastic kidney is not recommended, because one report has shown no evidence of the effectiveness of this therapy [53–56, k].

(3) Treatment for CKD stage 5 (Dialysis and Transplantation) (Grade B, level 4)

1) Basic considerations

1. Children with CKD stage 4–5 should be treated by a multidisciplinary team consisting of pediatric nephrologists, pediatric urologists, and other professionals. The team should be acquainted with hemodialysis, peritoneal dialysis, and renal transplantation [l]. (Grade A, Consensus)
2. Mental support should be provided to patients, as well as their parents and siblings. (Grade B, Consensus)

2) Indications for renal replacement therapy

1. Preparations for renal replacement therapy should be made for children with CKD stage 4. (Grade B, Consensus)
2. Commencement of renal replacement therapy is recommended for children with CKD stage 5 and for children with CKD stage 4 who have retarded growth and development, malnutrition, and uremia that is not well controlled by conservative therapy. (Grade A, Consensus)

3) Selection of renal replacement therapy (Grade B, Level 4)
Hemodialysis, peritoneal dialysis, and renal transplantation can be performed in children, similar to the treatment of adults. However, dialysis should be a temporary solution, and renal transplantation should be the ultimate goal in terms of the patient's survival [57], growth, development, and QOL [m].

4) Dialysis

1. As maintenance dialysis, peritoneal dialysis is the first-line of treatment for children. Peritoneal dialysis is superior to hemodialysis with respect to vascular access and fluid and nutritional management, particularly in very young children. (Grade B, Consensus)
2. Swan-neck and double-cuffed Tenckhoff catheters are recommended for maintenance peritoneal dialysis [58]. (Grade B, Level 4)
3. Exumbilication, gastrorrhexis, exstrophy of the bladder, diaphragmatocele, intestinal damage, and extensive peritoneal adhesion are indisputable contraindications for the commencement of peritoneal dialysis. Polycystic kidney disease with huge cysts, other intraabdominal lesions, pleuroperitoneal communications, colostomy, spinal disease, impaired ventilation, and similar conditions are relatively strong contraindications. A lack of appropriate caregivers for children is also a relatively strong contraindication. (Grade A, Consensus)
4. The right internal jugular vein is the site of choice for placement of a catheter for hemodialysis. Catheter placement in the inferior vena cava should be avoided whenever possible [59]. (Grade A, Level 5)

5) Renal transplantation

1. Both living-donor transplantation and deceased-donor renal transplantation are options for children with CKD. For infants, however, living-donor renal transplantation is recommended. (Grade B, Consensus)
2. Preemptive transplantation (PET) can be performed even in children [60–62]. (Grade B, Level 4)
3. Body size is an important factor in renal transplantation. Children who are approximately 1 year or older or who have a height of 75–80 cm or more can be candidate recipients. (Grade B, Consensus)
4. For children approximately 5 years or older, ABO-incompatible renal transplantation can be performed after plasma exchange and splenectomy. Outcomes (graft survival) of these patients are equivalent to those of recipients of ABO-compatible renal transplants [63]. (Grade B, Level 4)
5. Active infections, malignant tumors (except for cases with complete recovery), and severe cardiac or hepatic dysfunction are indisputable contraindications to renal transplantation. Aplasia and obstruction of the inferior vena cava are relatively strong contraindications. FSGS is not a contraindication to renal transplantation, but is associated with a high recurrence rate after renal transplantation. Therefore, steroid pulse therapy [64] and plasma exchange [65] should be considered for the prevention and management of relapse. (Grade A, Consensus)
6. Potential urinary tract anomalies associated with CKD must be assessed before renal transplantation. (Grade A, Consensus)

Chapter 18: Initiation of dialysis

Statements

1. Patient education and care preceding the initiation of dialysis

a. All patients with stage 4 or 5 CKD should periodically consult a nephrologist [1, 2]. (Grade A, Level 3)

b. Patients with stage 4 or 5 CKD and their families should have sufficient information on renal replacement therapy (hemodialysis, peritoneal dialysis and kidney transplantation) [3, 4]. (Grade A, Level 3)

c. Ambulatory care and educational programs for outpatients with the participation of different professionals (including physicians as well as other health professionals) are effective for pre-dialysis education and related care [5, 6]. (Grade B, Level 3)

2. Initiation of dialysis

a. For patients with stage 5 CKD, appropriate timing for the initiation of dialysis is determined, considering its effects on their prognosis for survival, possible avoidance of complications associated with end-stage kidney failure, QOL of individual patients, as well as the risks associated with the dialysis and economic burden related to the procedures [7, 8]. (Grade A, Level 4)

b. Even for patients with stage 4 CKD, dialysis is initiated regardless of their serum Cr levels, if body fluid retention, an abnormal electrolyte content, or exacerbation of the patients' nutritional status is recognized. For high-risk patients with a diabetic complication in particular, early initiation of dialysis may be desirable in view of their clinical condition, even when their serum Cr levels are relatively low [7, 8]. (Grade A, Level 4)

3. Selection of dialysis modalities (Grade B, Level 4)
There is no difference in the prognosis for survival between hemodialysis and peritoneal dialysis for at least a few years following the initiation of the procedure [9–14]. Preservation of the residual renal functions (for the immediate period) and maintenance of a sufficient quantity of dialysis and the control of body fluid volume (for a sustained period) are cited as the preliminary requirements. One of the modalities should be selected to satisfy these requirements in accordance with each patient's preference and life style, medical contraindication, geographical conditions and the status of dialysis facilities.

Chapter 19: Kidney transplantation

Statements

1. Kidney transplantation as a treatment option for end-stage kidney disease (Grade A, Level 4)
Since kidney transplantation generally confers a survival benefit, this treatment option for end-stage kidney disease should be explained to all patients with CKD stage 4 and 5, and also to their family [1].

2. Significance of pre-emptive kidney transplantation (Grade B, Level 4)
Living donor kidney transplantation can be performed prior to the initiation of dialysis, which is called "pre-emptive kidney transplantation (PET)". PET is reported to be superior in terms of patient and graft survival compared to kidney transplantation after the initiation of dialysis [2, 3].

3. Importance of management of CKD in kidney transplant recipients and donors (Grade A, Level 4)
A number of kidney transplant recipients and donors have developed into CKD stage 3 to 5 after kidney

transplantation and donor nephrectomy, respectively. Thus, long-term and periodical follow-up and management of CKD in both recipients and donors are important [4].

Chapter 20: CKD care for the elderly

Statements

1. Characteristics of CKD in the elderly
a. It is recommended that the elderly regularly have their kidney function checked, since kidney function progressively deteriorates in parallel with ageing [1–3]. (Grade A, Level 2)
b. A higher chance of malignancy warrants medical checks such as urine cytology, echography, and cystoscopy in the elderly with hematuria [4, 5]. (Grade A, Level 1)

2. Life-style and nutrition for the elderly with CKD
a. It is recommended that the elderly with CKD quit smoking since smoking is a risk factor for CKD progression [6]. (Grade B, Level 2)
b. It is recommended that the elderly with CKD receive influenza and pneumococcal vaccinations [7]. (Grade B, Level 4)
c. A low protein diet is recommended for the elderly with CKD stage 3 to 5 [8–16]. (Grade B, Level 1)
d. Salt restriction (<6 g/24 h) is recommended for the elderly with CKD because of their salt-sensitive hypertension [17, 18]. However, attention should be paid to kidney functional deterioration due to volume depletion under a low salt diet. (Grade A, Consensus)

3. Hypertension and CVD in the elderly with CKD
a. It is suggested that antihypertensive therapy for the elderly with CKD suppresses their CKD progression and CVD emergence [19–26]. (Grade A, Level 1)
b. Optimal target blood pressure is not established for the elderly with CKD. Lowering blood pressure to less than 120/80 mmHg worsens their prognosis [27–29]. (Grade B, Level 1)
c. Monotherapy or combined therapy with ACEi/ARB, diuretics or CCB is recommended as optimal antihypertensive medication [19, 30–39]. (Grade A, Level 2)
d. Five to 22% of the elderly with CKD are complicated with atherosclerotic renal artery stenosis [40–43]. (Grade B, Level 4)

4. Metabolic disorders in the elderly with CKD
a. Hyperglycemia is considered a risk factor for diabetic macroangiopathy and nephropathy in the elderly with CKD [44]. Therefore, optimal control of blood glucose, blood pressure and lipid is recommended for the elderly with diabetic nephropathy. (Grade B, Level 4)
b. Therapy with statins against dyslipidemia may slow CKD progression in the elderly with CKD [45]. (Grade B, Level 1)
c. While an overweight condition (BMI:25–29.9) is not associated with prognosis in the elderly with CKD, obesity (BMI $\geq$ 30) increases the relative risk of all-cause mortality 1.1-fold [46]. (Grade B, Level 1)
d. In the elderly, the waist-to-hip ratio predicts the emergence of CKD and CVD better than BMI [47–49]. (Grade B, Level 4)
e. An association between metabolic syndrome and the emergence of CKD and CVD remains to be established in the elderly [50–53]. (Grade B, Level 4)
f. Therapy using bisphosphonates against osteoporosis should be taken care within the elderly with CKD stage 1 to 3 and avoided in those with CKD stage 4 and 5. (Grade B, Consensus)
g. Care should be taken with the administration of an active form of vitamin D to the elderly with CKD because of the emergence of hypercalcemia. (Grade A, Consensus)

5. Kidney transplantation in the elderly
a. Kidney transplantation may extend the life expectancy of the elderly with CKD [54]. (Grade A, Level 4)
b. Kidney donation from the elderly is not contraindicated if informed consent for postoperational kidney function and optimal care after kidney donation are ensured [55–59]. (Grade A, Level 4)

6. Drug administration for the elderly with CKD
a. Percutaneus transluminal coronary intervention is a high-grade risk factor for contrast nephropathy in the elderly over 75 years of age with CKD [60]. (Grade A, Level 4)
b. Dose and duration of medication with COX-2 selective and non-selective anti-inflammatory drugs should be minimized for the elderly because both may deteriorate kidney function to an identical degree [61–63]. (Grade A, Level 2)

Chapter 21: Drug administration

Statements

1. Contrast media
a. The frequency of use and dose of contrast media is positively associated with an increase in the relative risk for kidney functional deterioration [1]. (Grade A, Level 4)
b. To prevent contrast-induced nephropathy, the dose of contrast medium should be minimized [1]. (Grade A, Level 4)

c. To prevent contrast-induced nephropathy, peri-procedural hydration is recommended [2–5]. (Grade A, Level 2)
d. Post-procedural blood purification does not prevent contrast-induced nephropathy [6, 7]. (Grade A, Level 1)
e. The use of gadolinium-based MRI contrast media in patients with CKD is a risk factor for the emergence of nephrogenic systemic fibrosis. As a general rule, patients with CKD stage 4 and 5 should avoid the use of gadolinium-based MRI contrast media [a]. (Grade A, Consensus)

2. Anti-inflammatory drugs (Grade B, Consensus)
There have been no anti-inflammatory drugs devoid of acute deterioration of renal function in patients with CKD. Therefore, the duration of use and dose of any anti-inflammatory drugs should be minimized.

3. Anti-biotics (Grade A, Consensus)
The dose of antibiotics should be reduced according to the residual renal function in patients with CKD. Therapeutic drug monitoring should be performed when administering aminoglycosides and glycopeptides to patients with CKD.

4. Anti-uremic drugs (Grade C, Level 2)
An oral adsorbent AST-120 may delay the initiation of dialysis in patients with CKD [8–14].

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Chapter 1: Diagnosis of CKD

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Chapter 2: Significance of CKD

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Chapter 3: CKD and Life-style

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Chapter 6: Renal anemia

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Chapter 7: Mineral and bone disorder in CKD

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Chapter 9: IgA nephropathy

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Chapter 10: Nephrotic syndrome (Idiopathic membranous nephropathy, and primary focal segmental glomerulosclerosis)

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Chapter 12: Atherosclerotic renal artery stenosis

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Chapter 13: Autosomal-dominant polycystic kidney disease (ADPKD)

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Chapter 14: Management of Dyslipidemia in CKD

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Chapter 15: Obesity and Metabolic syndrome

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Chapter 18: Initiation of dialysis

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Chapter 20: CKD care for the elderly

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Chapter 21: Drug administration

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