

Nutritional support in renal disease

Maryam Alinezhad-Namaghi MD, Ph.D.

Assistant Professor of Clinical Nutrition

Department of Nutrition, Mashhad University of Medical Sciences, Mashhad, Iran



BASICS IN CLINICAL NUTRITION

Fifth Edition

Editor in Chief
Luboš Sobotka

Associate Editors
Simon P. Allison
Alastair Forbes
Rémy F. Meier
Stéphane M. Schneider
Peter B. Soeters
Zeno Stanga
Andre Van Gossum

GALÉN

Learning objectives

- To understand the *metabolic abnormalities* in various groups of patients with renal disease
- To recognize the impact of *renal replacement therapy* on metabolism and nutrient balances
- To learn about the determinants of nutritional state and the *causes of malnutrition* in renal disease
- To understand the impact of *acute intermittent disease states* on nutrient requirements
- To learn about the *aims of nutritional support* as well as the type and composition of diets for patients with renal disease

Outline

1- Pathophysiology

2- Nutritional therapy for patients with renal disease

Non-catabolic patients with stable chronic kidney disease (CKD)

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

Patients with AKI and HD/CAPD patients with acute catabolic disease

Solutions for nutritional support

Complications and monitoring of nutritional support

3- Nutritional management of renal transplantation (RTX)

Pathophysiology

Protein-energy Wasting (PEW) in renal disease

Metabolic and nutritional consequences of renal replacement therapies

Pathophysiology

Patients with renal insufficiency

Comprise an extremely *heterogeneous* group of subjects

With *differing* and sometimes *contradictory*

Aims of nutritional support,

Nutritional requirements,

And consecutive composition of nutritional regimens

Pathophysiology

Renal failure is a

Pan-metabolic and *pan-endocrine* abnormality

Affecting virtually every metabolic pathway in the body

Despite **differences** in metabolic presentation and nutritional needs

In various forms of renal insufficiency and during the course of disease

In the individual patient,

There are *some common features in their metabolic changes*

Pathophysiology

Energy metabolism

Is not grossly affected by renal dysfunction

(which decreases rather than increases oxygen consumption)

And is more determined by **associated complications**

Pathophysiology

Common metabolic abnormalities in patients with renal failure

Peripheral insulin resistance

Impairment of lipolysis

Low-grade inflammatory state with activation of protein catabolism

Metabolic acidosis

Hyperparathyroidism, mineral and bone disorder MBD

Impairment of activation of vitamin D3

Augmented catabolic response to intermittent Disease

Impairment of potassium tolerance

Renal anaemia

Pathophysiology

Protein-energy Wasting (PEW) in renal disease

The term '*protein-energy wasting*' has been proposed for loss of body protein mass and fuel reserves in acute and chronic kidney disease (CKD).

Protein-energy wasting is diagnosed based on three characteristics:

- ✓ Low serum levels of *albumin, transthyretin (prealbumin), or cholesterol*
- ✓ Reduced body mass (low or reduced *body mass* or *fat mass*,
Or *weight loss* with reduced intake of protein and energy)
- ✓ Reduced muscle mass (*muscle* wasting or *sarcopenia*, reduced *circumference of mid-arm muscle*)

Pathophysiology

Metabolic and nutritional consequences of renal replacement therapies

Renal replacement therapies (RRTS)

Are associated with *multiple metabolic side effects*

Among are the *loss of nutritional substrates*

(e.g. Amino acids and water-soluble vitamins),

But also *systemic effects*

(e.g. Activation of *protein catabolism*

And *increase in ROS formation*

As a consequence of bio-incompatibility)

Causes of malnutrition in haemodialysis patients

- **Reduced oral intake of nutrients (anorexia, depression, restrictive diets, low social status)**
- **Gastrointestinal consequences of uraemia**
- **Uremic toxicity: Inadequate dialysis prescription**
- **Metabolic acidosis**
- **Endocrine factors (growth-factor abnormalities, deficiency in erythropoietin and testosterone, insulin resistance, hyperparathyroidism)**
- **Dialysis-associated factors (loss of nutrients, induction of protein catabolism)**
- **Diabetes mellitus**
- **Reduced physical activity**
- **Intermittent acute diseases (e.g. infections)**

Pathophysiology

Metabolic and nutritional consequences of renal replacement therapies

In patients with **acute kidney injury (AKI)**,

Continuous renal replacement therapies (CRRTs)

Have become first-line treatment,

The metabolic side effects of which are relevant

Because of the *high turnover of fluids*

These effects of RRTs must be considered

In *designing a nutritional programme*

For a patient with *RRT-dependent renal insufficiency*

Pathophysiology

Nutrient requirements in non-catabolic patients with CKD

Energy	20–25	(max. 35)	kcal · kg ⁻¹ · day ⁻¹
Carbohydrates	2–3	(max. 4)	g · kg ⁻¹ · day ⁻¹
Lipids	0.8–1.2	(max. 1.5)	g · kg ⁻¹ · day ⁻¹
Amino acids / protein conservative therapy + RRT + hypercatabolism	0.6–1.2 1.2–1.5	(max. 1.7)	g · kg ⁻¹ · day ⁻¹ g · kg ⁻¹ · day ⁻¹ g · kg ⁻¹ · day ⁻¹
Vitamins (combination products containing RDA) Water soluble Lipid soluble		2 × RDA · day ⁻¹ (= 2 ampoules/ day) 1 × RDA · day ⁻¹ (= 1 ampoule/day)	(Cave: Vitamin C < 250 mg · day ⁻¹) (higher for vitamin D?)
Trace elements (combination products containing RDA)		1 × RDA · day ⁻¹ (= 1 ampoule/day)	(higher for selenium 200–500 µg · day ⁻¹ ?)
Electrolytes (must be adapted individually)	(Cave: refeeding hypophosphatemia)		

RRT = renal replacement therapy, RDA = recommended dietary allowances

* Please note: These are (and can only be) approximate values; requirements can fundamentally vary between patients but also within a patient during the course of disease!

Nutritional therapy for patients with renal disease

Non-catabolic patients with stable chronic kidney disease (CKD)

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

Patients with AKI and HD/CAPD patients with acute catabolic disease

Solutions for nutritional support

Complications and monitoring of nutritional support

Nutritional therapy for patients with renal disease

Nutritional support for patients with renal disease is focused on four main areas:

- *The non-catabolic patient with stable CKD(stages 3-5)*
- *The CKD patient on regular RRT (+/- malnutrition)*
- *The catabolic patient with AKI/ the CKD patient on chronic RRT and acute intermittent disease*
- *The patient undergoing renal transplantation*

Nutritional therapy for patients with renal disease

Non-catabolic patients with stable chronic kidney disease (CKD)

Aims of nutritional management

Nutritional state

Nutritional therapy in stable CKD patients

Aims of nutritional management

- Maintain *optimal nutritional status*
- Prevent *evolution of malnutrition*
- To reduce or control *accumulation of waste products*
- To alleviate *acidosis*,
- To prevent *cardiovascular disease*
- To manage *mineral bone disorder* of CKD (MBD) by treating hyperparathyroidism and vitamin-D deficiency,
- To treat *hyperlipidaemia*
- To retard progression of *renal dysfunction*

Nutritional state

CKD patients Are at high risk of malnutrition because of :

Uraemia-associated factors

Metabolic acidosis

Concurrent disease

Impaired appetite and oral intake of food

The gastrointestinal side effects of uraemia

And potentially, misdirected dietary regimens

Nutritional state

In the absence of concurrent disease

And

Adequate compensation for metabolic acidosis,

The patients usually are not (grossly) catabolic

Note: In patients with CKD, there is a delicate balance

Between the

Induction of toxic effects by giving excess nutrition

And inducing malnutrition by giving too little

Nutrient requirements in non-catabolic patients with CKD

	Conservative therapy	Haemodialysis	Peritoneal dialysis
Energy** (kcal · kg ⁻¹)	> 35	30–35	> 35***
Protein (g · kg ⁻¹)	0.6–0.8	1.1–1.4	1.2–1.5
Phosphorus (mg) (mmol)	600–1000 19–31	800–1000 25–32	800–1000 25–32
Potassium (mg) (mmol)	1500–2000** 38–40	2000–2500 40–63	2000–2500 40–63
Sodium (g) (mmol)	1.8–2.5** 77–106	1.8–2.5 77–106	1.8–2.5 77–106
Fluid (ml)	Not restricted	1000 ml + DO	1000 ml + UF + DO

**Individual requirements can differ considerably*

***for ambulatory but not bed-ridden patients*

****Includes energy (glucose) from the dialysate*

DO – daily (urine) output, UF – ultrafiltration

Nutritional therapy in stable CKD patients

Special attention must be given to

Protein,
Phosphorus,
Potassium,
Bicarbonate,
And vitamin-D, (or analogues)

Erythropoiesis-stimulating agents (and iron)

Should be applied, if necessary

Nutritional therapy in stable CKD patients

During dietary treatment,
the most controversial question is related to

Protein intake

There is some agreement that there should be (at least)

A *moderate restriction of protein* (0.7-0.8 g.kg . day⁻¹)

If protein intake is **reduced to < 0.6 g.kg .day,**

A supplement of keto-analogues of amino acids

Should be provided

Nutritional therapy in stable CKD patients

The extent of **potassium restriction**

Has to be adapted to the individual patient

Phosphate intake should be reduced,

As well as the **dietary acid load**,

And **bicarbonate**

Should be supplemented as required

The diet should be rich in

Fruits and **vegetables**, and in **fibre**,

Which can potentially

Reduce formation of uremic toxins and mitigate inflammatory status

Nutritional therapy in stable CKD patients

Specific "renal" oral nutritional supplements (ONS)
are available for this patient group

These are characterized by
low protein, potassium, and phosphate content

They may have *variable additions*, such as histidine and carnitine

Unfortunately, studies using these supplements are limited

Nutritional therapy in stable CKD patients

Enteral nutrition (EN)

or

parenteral nutrition (PN)

Is usually given only to patients

With *additional acute intermittent disease*,

Who often have *increased nutrient requirements*

Nutritional therapy for patients with renal disease

Non-catabolic CKD patients on chronic RRT
(with or without malnutrition)

Nutritional management In patients on renal replacement therapy

Nutritional therapy In stable HD patients (+/- malnutrition)

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

Patients on

Chronic RRT

Haemodialysis (HD);

Chronic ambulatory peritoneal dialysis (CAPD)

Are at a high risk of developing **malnutrition**

Malnutrition together with **inflammation**

Is a major determinant of **survival**

Causes of malnutrition in haemodialysis patients

- **Reduced oral intake of nutrients** (anorexia, depression, restrictive diets, low social status)
- Gastrointestinal consequences of uraemia
- Uremic toxicity: Inadequate dialysis prescription
- Metabolic acidosis
- Endocrine factors (growth-factor abnormalities, deficiency in erythropoietin and testosterone, insulin resistance, hyperparathyroidism)
- Dialysis-associated factors (loss of nutrients, induction of protein catabolism)
- **Diabetes mellitus**
- Reduced physical activity
- Intermittent acute diseases (e.g. infections)

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

HD per se

is A **catabolic** event,

Not only because of the

Loss of 8-12 g of amino acids

during one session

But also because of

An **induction of an inflammatory reaction**

Metabolic side effects of intermittent haemodialysis

Loss of water-soluble molecules

Amino acids

Water-soluble vitamins

L-carnitine

Electrolyte derangements

Induction of an inflammatory reaction / release of cytokines

IL-1 β , TNF- α

Activation of protein catabolism

Loss of amino acids

Loss of proteins and blood

Inflammatory stat

Increased formation of reactive oxygen species

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

During **CAPD**,

8-9 g of protein are lost

In the dialysate *every day*

In general, loss of *water-soluble substances*

Is lower

And loss of *protein bound substances*

(such as trace elements)

Is higher than during HD

Glucose uptake by using

Glucose containing hyperosmolar PD-solutions

May account for **up to 125 g day**

Nutritional management in patients on renal replacement therapy

The aim of nutritional management is to

Prevent, detect, or treat

Malnutrition,

To reduce accumulation of

Fluid and waste products, and of potassium, and phosphorus,

And to prevent complications of uraemia

(Cardiovascular disease,

Hypertension,

Mineral bone disorder (MBD))

Nutritional monitoring

Nutritional state

Should be monitored

Routinely

According to *standardized protocols*

In *all dialysis patients*

Nutrient requirements in non-catabolic patients with CKD

	Conservative therapy	Haemodialysis	Peritoneal dialysis
Energy** (kcal · kg ⁻¹)	> 35	30–35	> 35***
Protein (g · kg ⁻¹)	0.6–0.8	1.1–1.4	1.2–1.5
Phosphorus (mg) (mmol)	600–1000 19–31	800–1000 25–32	800–1000 25–32
Potassium (mg) (mmol)	1500–2000** 38–40	2000–2500 40–63	2000–2500 40–63
Sodium (g) (mmol)	1.8–2.5** 77–106	1.8–2.5 77–106	1.8–2.5 77–106
Fluid (ml)	Not restricted	1000 ml + DO	1000 ml + UF + DO

**Individual requirements can differ considerably*

***for ambulatory but not bed-ridden patients*

****Includes energy (glucose) from the dialysate*

DO – daily (urine) output, UF – ultrafiltration

Nutrient requirements

- Recommended **protein intakes**

With > 50% of high biological value proteins are 1.2-1.4 g.kg .d

- Recommendations for **energy intake**

Vary between 30 to 40 kcal.kg .d

According to patient age and physical activity

- Due to **dialysis-induced losses**,

Water-soluble vitamins should be supplied

Nutrient requirements

- **Vitamin D** should be given according to

Serum levels of calcium, phosphorus, and parathyroid hormone

- Regular HD does not induce significant trace element losses

However, in **depleted patients**, supplementation with

Zinc (15 mg/d) and **selenium (300-600 µg/d)** may be useful

- Intake of **fluid, potassium and phosphate** **should be reduced**

In addition, **oral phosphate binders** must be given in most HD patients

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

Nutritional management In patients on renal replacement therapy

Nutritional monitoring

Nutrient requirements

Nutritional therapy In stable HD patients (+/- malnutrition)

Prerequisite

Nutritional counselling

Oral nutritional supplements (ONS)

Intradialytic nutrition

Intradialytic oral/enteral nutrition (IDEN)

Intradialytic parenteral nutrition (IDPN)

Other options for malnutrition therapy

Nutritional therapy in stable HD patients (+/- malnutrition)

Prerequisite

Nutritional counselling

Oral nutritional supplements (ONS)

Intradialytic nutrition

Intradialytic oral/enteral nutrition (IDEN)

Intradialytic parenteral nutrition (IDPN)

Other options for malnutrition therapy

Nutritional therapy in stable HD patients (+/- malnutrition)

Prerequisite:

Treatable causes of protein-energy wasting

Such as inadequate dialysis prescription,

Intermittent diseases (especially infections),

Metabolic acidosis (midweek pre-dialysis bicarbonate ≥ 22 mmol/l)

should be eliminated

Nutritional therapy in stable HD patients (+/- malnutrition)

Dietary counselling:

Is the **first essential step**
To improving nutritional status
In ***depleted dialysis patients***

There is a need for
Regular supervision/encouragement by a **dietician** in *dialysis patients*
to

Assess spontaneous nutrient intake,
Monitor nutrition state,
And to ***adapt the diet***

Nutritional therapy in stable HD patients (+/- malnutrition)

Oral nutritional supplements (ONS):

Specific ONS adapted to the nutritional needs of dialysis patients
Are available

These diets have a

Higher protein content

And are reduced in potassium and phosphorus

Some have additions such as *Histidine* or *Carnitine*

Nutritional therapy in stable HD patients (+/- malnutrition)

Numerous studies (including several controlled trials) have reported

A positive effect of ONS on nutritional parameters

In HD patients

ONS should be taken

1 hour after usual meals

And/ or during HD sessions

Intra dialytic nutrition

Note: Take advantage of the intradialytic interval!

In contrast to earlier recommendations,

The **intradialytic time** presents

An optimal period for dietary counselling

And

For *nutritional interventions*

(Oral/ ONS/IDEN/ IDPN)

Intra dialytic nutrition

Intradialytic oral/enteral nutrition (IDEN):

HD patients *should be encouraged to eat during dialysis*

In patients at high risk of malnutrition, ONS can be given as

An **alternative**

Or in **addition**

To usual meals

Several controlled studies have documented the

Beneficial effect of intradialytic ONS on

Nutritional indices and even ***outcomes***

Intradialytic parenteral nutrition (IDPN)

In selected patients

in whom other interventions fail,

IDPN should be considered

IDPN can provide the equivalent of

7-8 kcal/kg d

and 0.3-0.4g protein/kg/d

And, thus, can supplement only the oral/enteral intake.

Usually, a spontaneous intake of

< 0.8 g/kg protein and of 20 kcal/kg .D

Is required to ensure recommended intakes in patients on IDPN

Intradialytic parenteral nutrition (IDPN)

IDPN

Is a **cyclic PN**
Infused into the

Complete PN mixtures,

Including **amino acids, lipids, and glucose,**

Are usually given

The solution is infused at a constant rate

Up to a maximum of **250 ml/h** during the **whole 4-h dialysis session**

The associated volume must be eliminated by ultrafiltration of an
Equivalent volume.

Other options for malnutrition therapy

Alternative - several of them experimental - interventions to
Improve nutritional state include

The use of orexigenic compounds to improve appetite,

Special nutrients to *decrease protein breakdown*

And/or to *promote protein synthesis*

(*essential/ branched-chain amino acid formulas, keto acids*),

Anabolic agents,

Nutrients to reduce inflammation (fish-oil, antioxidants),

Anti-inflammatory agents,

And, in particular, physical exercise

Other options for malnutrition therapy

Among these approaches,

Physical exercise (*with/without anabolic steroids*)

Is of **predominant importance**

*It is **difficult/impossible***

*To **improve nutritional state***

Without exercise

Other options for malnutrition therapy

In addition to

Improving nutritional state

And reducing obesity,

Physical activity can **improve**

Subjective well-being

And quality of life,

Reduce depression,

Retard cognitive decline,

And slow progression of renal dysfunction

In general,

Nutritional therapy cannot be presented as an isolated intervention

But must be part of a multimodal approach

Outline

1- Pathophysiology

2- Nutritional therapy for patients with renal disease

Non-catabolic patients with stable chronic kidney disease (CKD)

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

Patients with AKI and HD/CAPD patients with acute catabolic disease

Solutions for nutritional support

Complications and monitoring of nutritional support

3- Nutritional management of renal transplantation (RTX)

Nutritional therapy for patients with renal disease

Patients with AKI and HD/CAPD patients with acute catabolic disease

Aims of nutritional therapy in AKI

Metabolic aspects and nutritional requirements

Nutrient requirements

Energy metabolism and energy requirements

Requirements of amino acids and proteins

Carbohydrate metabolism

Lipid metabolism

Micronutrients

Electrolytes

Patients with AKI and HD/CAPD patients with acute catabolic disease

The practice of nutritional therapy in

Acutely ill patients with AKI/CKD 5 requiring RRT

Follows the same principles

As in those of other **critically ill patient** groups

However, nutrition has to be

*Adapted to the **specific consequences of renal dysfunction***

*And of the **type and intensity of RRT***

On metabolism and nutrient requirements

Patients with AKI and HD/CAPD patients with acute catabolic disease

In the early stages of

AKI (RIFLE I/R, AKIN I and II)

Or CKD (stage 3)

Measures must be taken

(Modulation of volume state, haemodynamics, etc)

To prevent

Further deterioration of renal function

Patients with AKI and HD/CAPD patients with acute catabolic disease

This also includes **metabolic/nutritional aspects**,

Such as **maintaining**

Electrolyte balance,

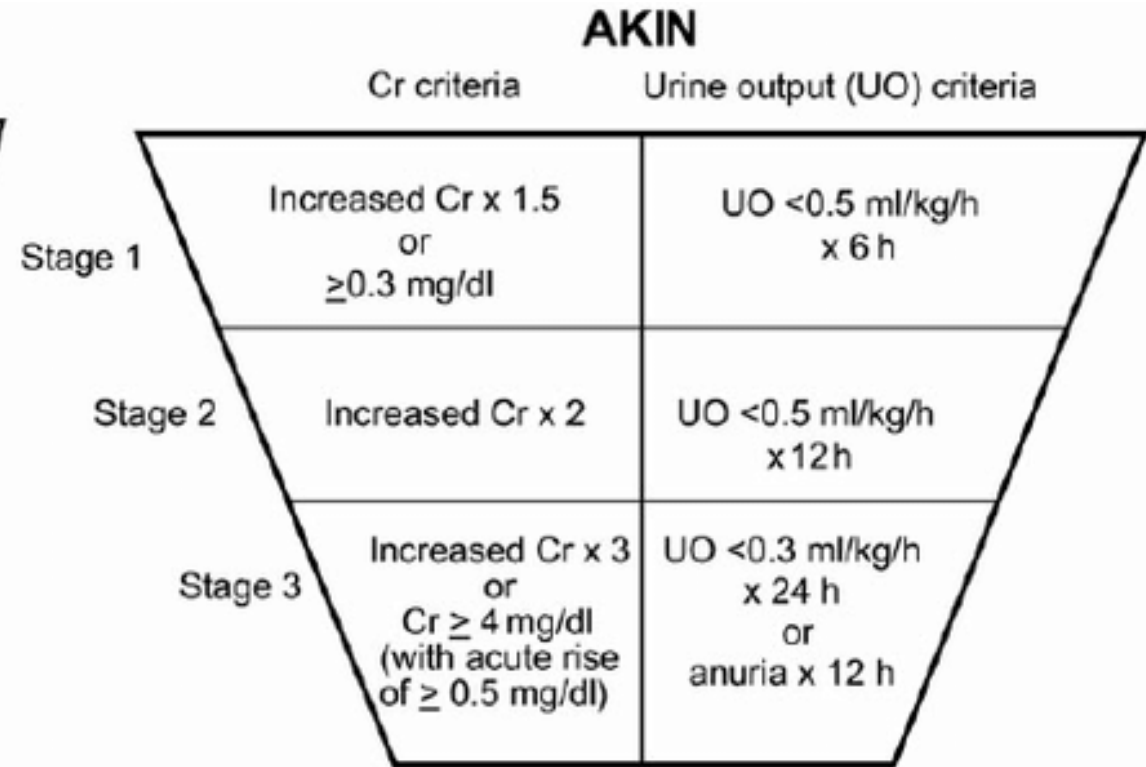
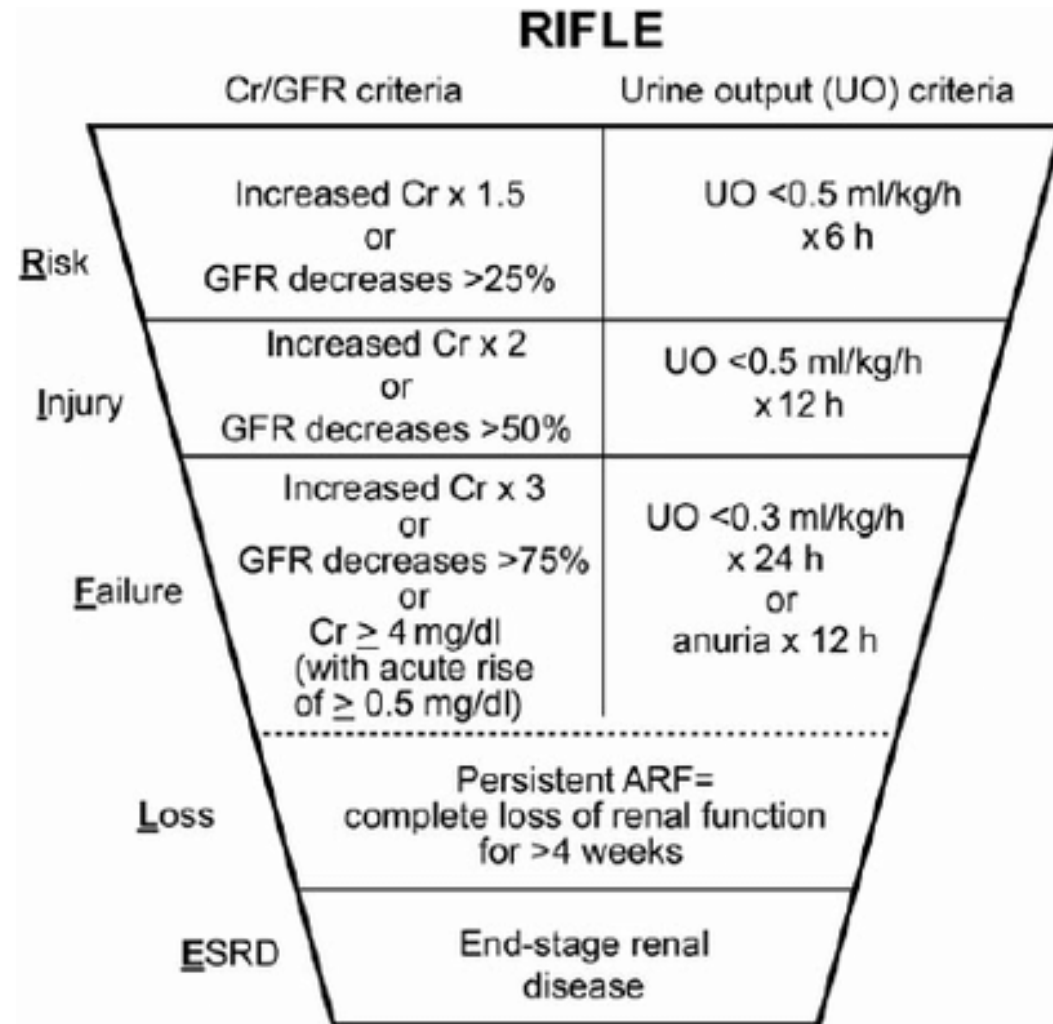
Avoidance of

Overhydration and *glucose disbalance,*

And **prevention of development of**

Overt malnutrition

Patients with AKI and HD/CAPD patients with acute catabolic disease



Patients who receive renal replacement therapy (RRT) are considered to have met the criteria for stage 3 irrespective of the stage that they are in at the time of commencement of RRT.

Patients with AKI and HD/CAPD patients with acute catabolic disease

In more advanced stages of

AKI (RIFLE F or AKIN III)/CKD 5,

RRT is required to compensate for

The *systemic consequences of uraemia*,

To *maintain volume and electrolyte balance*,

And to *support haemodynamics and respiratory function*

Patients with AKI and HD/CAPD patients with acute catabolic disease

In these patients,

The *specific consequences of renal dysfunction*

And of the *type and intensity of RRT*

On

Metabolism and nutrient requirements

Have to be taken into consideration

Aims of nutritional therapy in AKI

In **AKI**, the aim of nutritional treatment

Is not the alleviation of uremic toxicity
And retardation of progression of renal disease
(As in CKD)

But, *as in other acute disease*,

The *stimulation of immunocompetence*,
Of *wound healing*,
And other *reparative functions*

In most situations, *requirement will exceed*

The minimal intake recommended for stable **CKD** patients
Or the recommended allowances (**RDA**) for normal subjects

Metabolic aspects and nutritional requirements

AKI

Is a complication of other conditions

Such as

Sepsis,

Trauma,

Or *multiple-organ failure*

Metabolic changes will be determined by

The *uremic state* plus the *underlying disease process*,

By its *complications* (eg, *severe infections* and *organ dysfunction*),

And by *the type and intensity of RRT*

Metabolic aspects and nutritional requirements

Nevertheless, the *acute loss of excretory renal function*

Affects not only *water*,

Electrolyte,

And *acid-base metabolism*,

But also has a profound effect on

The *metabolism of proteins and amino acids*,

Carbohydrates

And *lipids*,

And *endocrine functions*

Metabolic aspects and nutritional requirements

The **optimal intake of nutrients in AKI**

is influenced more by the

Nature of the illness causing AKI,

The *extent of catabolism,*

And the *type and frequency of RRT*

Rather than renal dysfunction

Note: Patients with AKI present an *extremely heterogeneous group* of subjects *with widely differing nutrient requirements*; individual requirements can *vary considerably during the disease course*

Metabolic aspects and nutritional requirements

All types of RRT

Exert a profound impact on

Metabolism and *nutrient balance*

Metabolic side effects of intermittent haemodialysis

Loss of water-soluble molecules

Amino acids

Water-soluble vitamins

L-carnitine

Electrolyte derangements

Induction of an inflammatory reaction / release of cytokines

IL-1 β , TNF- α

Activation of protein catabolism

Loss of amino acids

Loss of proteins and blood

Inflammatory stat

Increased formation of reactive oxygen species

Metabolic side effects of continuous renal replacement therapy (CRRT)

Loss of heat (= loss of energy)

Intake of substrates = intake of energy

lactate, citrate, glucose

Loss of nutrients

amino acids, vitamins, trace elements, L-carnitine etc

Loss of electrolytes

phosphate, potassium, magnesium

Elimination of peptides/proteins

albumin, hormones, mediators

Metabolic consequences of bioincompatibility

Induction of an inflammatory reaction; activation of mediator-cascades, stimulation of protein catabolism

Nutrient requirements in non-catabolic patients with CKD

Energy	20–25	(max. 35)	kcal · kg ⁻¹ · day ⁻¹
Carbohydrates	2–3	(max. 4)	g · kg ⁻¹ · day ⁻¹
Lipids	0.8–1.2	(max. 1.5)	g · kg ⁻¹ · day ⁻¹
Amino acids / protein conservative therapy + RRT + hypercatabolism	0.6–1.2 1.2–1.5	(max. 1.7)	g · kg ⁻¹ · day ⁻¹ g · kg ⁻¹ · day ⁻¹ g · kg ⁻¹ · day ⁻¹
Vitamins (combination products containing RDA) Water soluble Lipid soluble		2 × RDA · day ⁻¹ (= 2 ampoules/ day) 1 × RDA · day ⁻¹ (= 1 ampoule/day)	(Cave: Vitamin C < 250 mg · day ⁻¹) (higher for vitamin D?)
Trace elements (combination products containing RDA)		1 × RDA · day ⁻¹ (= 1 ampoule/day)	(higher for selenium 200–500 µg · day ⁻¹ ?)
Electrolytes (must be adapted individually)	(Cave: refeeding hypophosphatemia)		

RRT = renal replacement therapy, RDA = recommended dietary allowances

* Please note: These are (and can only be) approximate values; requirements can fundamentally vary between patients but also within a patient during the course of disease!

Requirements of amino acids and proteins

AKI is characterized by

A *profound activation of muscle protein catabolism*

Of *1.3-1.8 g protein/kg.day*

And consecutive *stimulation of hepatic gluconeogenesis and ureagenesis*,

Utilization of amino acids is altered and several amino acids designated as 'Non-essential' in healthy subjects, such as tyrosine, arginine, and cysteine,

Can *become 'conditionally essential' in renal failure*

Requirements of amino acids and proteins

Protein/amino acid requirements

In patients *not undergoing renal replacement* therapy

Are usually 0.8-1.2 g/kg.day

And with *daily HD/ CRRT*

Increase to 1.2-1.5 g/kg.day (max 1.7 g/kg.day).

Higher intakes (up to 2.5g/kg.day) as suggested by some groups

Have no proven benefit

Carbohydrate metabolism

Hyperglycaemia

Is usually present in patients with AKI

The *major cause* is *peripheral insulin resistance*

A *second feature* is *accelerated hepatic gluconeogenesis*

Mainly from *conversion of amino acids*,

Which can be reduced

(but not suppressed) by exogenous nutrients

Moreover, *insulin metabolism becomes abnormal* in AKI

Carbohydrate metabolism

Glucose

Is the *most important energy substrate*
But *intake should not exceed 2-4 g/kg.day*

Adverse effects of an *excessive intake of glucose*

Are well characterized and include

The augmentation of renal injury in AKI

Hyperglycaemia must be prevented

And *insulin* is often necessary for glucose control

Lipid metabolism

AKI/CKD

Is also associated with *profound alterations in lipid metabolism*

The **triglyceride (TG)** content of lipoproteins is **increased**

And **high- density lipoprotein (HDL)-cholesterol** decreased

The major cause of these disturbances

Is impairment of lipolysis

Lipid metabolism

As a consequence,

Elimination of intravenously infused lipids is delayed

And *clearance reduced by > 50%*

These changes in lipid metabolism

Should not prevent the use of lipids in nutritional therapy

In patients with AKI

Instead, the *amount of lipids infused must be adjusted*

To meet the patient's capacity to utilize lipids

And *1 g/kg.day* will not usually substantially increase plasma triglycerides

Micronutrients

Requirements for **water-soluble vitamins**

Are *increased* in AKI/CKD-5,

Mainly because of **losses associated with RRT**

Despite the fact that *fat soluble vitamins are not lost during RRT*,

Plasma concentrations (with the exception of vitamin K) of vitamins

Are low in AKI

Similarly, *loss of trace elements is negligible during HD/CRRT*

But ***plasma concentrations of several elements,***

Such as selenium or zinc, are decreased

Micronutrients Requirements

Several micronutrients

Such as **vitamin A**, **vitamin C**, **vitamin E**, and **selenium**

Are components of the oxygen radical scavenger system in the body,

Depletion of which can

Contribute to **impaired immunocompetence**

And induce/promote **tissue injury**

In critically ill patients

Electrolytes

Electrolytes requirements

Can vary profoundly between AKI patients
But also during the course of disease
(For instance variations in urinary output)

Requirements must be determined

Individually on a *daily basis*

Electrolytes

Note: In contrast to stable CKD,

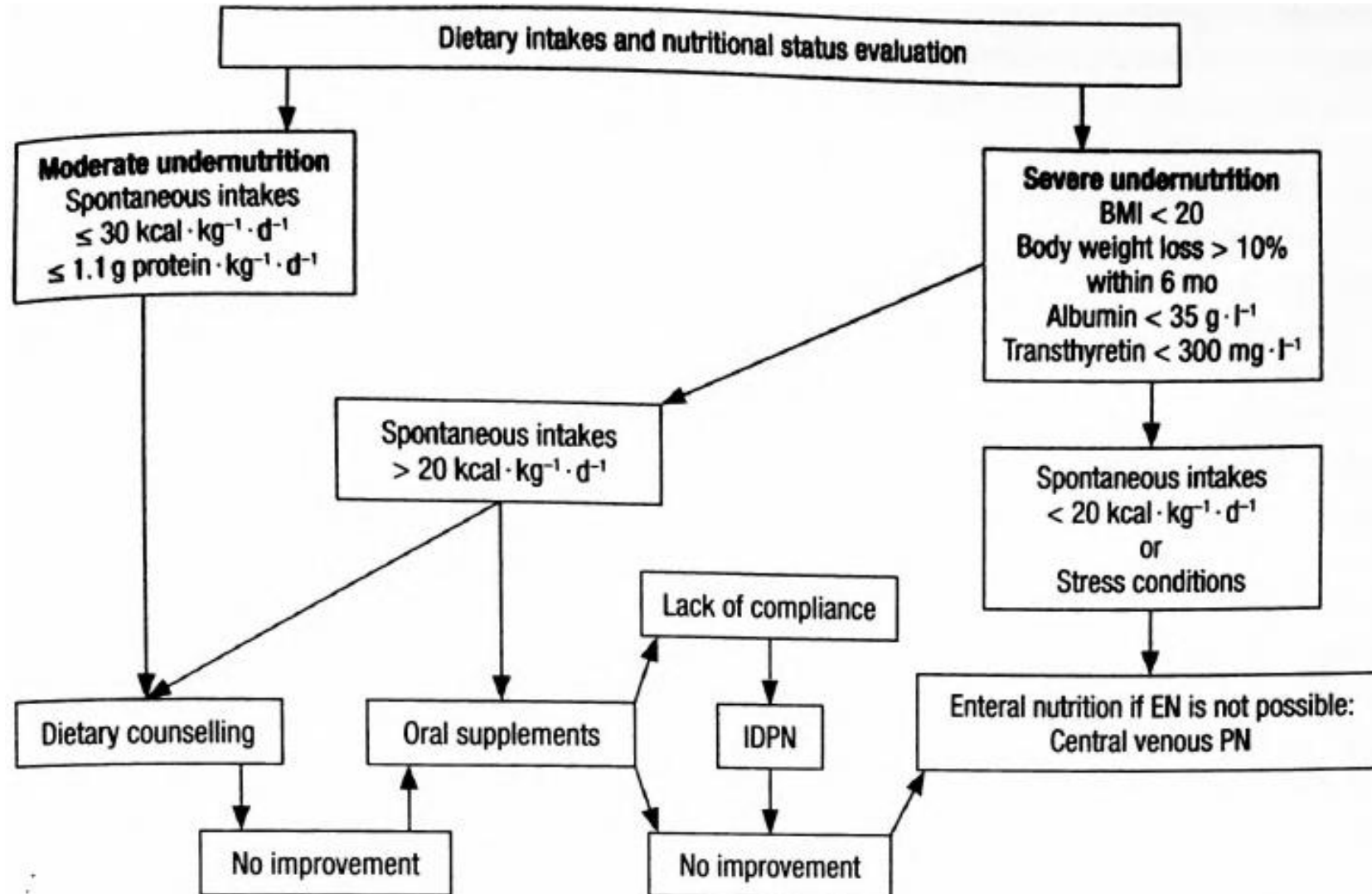
Many patients with AKI can present with

hypokalemia/hypophosphatemia,

Which can also develop during nutritional therapy
("Refeeding")

Or during CRRT with low electrolyte solutions

Decisional algorithm for nutritional support In haemodialysis patients



Outline

1- Pathophysiology

2- Nutritional therapy for patients with renal disease

Non-catabolic patients with stable chronic kidney disease (CKD)

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

Patients with AKI and HD/CAPD patients with acute catabolic disease

Solutions for nutritional support

Complications and monitoring of nutritional support

3- Nutritional management of renal transplantation (RTX)

Nutritional therapy for patients with renal disease

Solutions for nutritional support

Enteral nutrition

Enteral diets

Parenteral nutrition

Amino acid solutions

Lipid emulsions

All-in-One solutions

Enteral nutrition

Enteral nutrition has become the

Preferred type of nutritional support

In patients with **renal failure**

Besides the many well-described beneficial effects,

EN may *exert specific consequences*

in patients with AKI by improving renal perfusion/ function

Enteral nutrition

One *limitation* to EN in patients with AKI/ CKD,

However, is

The fact that *renal insufficiency*

Augments the *impairment of gastrointestinal motility*

And, in many patients,

The use of *prokinetics* may be required

To improve nutrition tolerance

Enteral nutrition

EN should be started at a low rate

(About 20% of the final target)

And the infusion rate should be increased slowly

To ensure

Gastrointestinal tolerance

And to avoid

The *evolution of metabolic complications*

Parenteral nutrition

When a (quantitatively sufficient)
enteral nutrition is *not possible*
and *tolerance* cannot be increased by
provision of *prokinetis*
and/ or placement of a *duodenal tube*,
supplementary or *total parenteral nutrition (PN)*
should be provided

Parenteral nutrition

PN

Should be *started at a low rate*

(i.e. **20%** of the final infusion rate)

And the *infusion rate should be increased slowly*

In order to **monitor**

The *utilization of the nutrients provided*

And to **avoid**

The *development of metabolic complications*

Nutritional therapy for patients with renal disease

Complications and monitoring of nutritional support

Complications and monitoring of nutritional support

Complications of nutritional support are *similar*

In *non-uraemic and renal failure patients*

However,

The *frequency of complications is high*

Because of the

Impairment of gastrointestinal functions,

The *reduced tolerance to volume load and electrolytes,*

And *alterations in the utilization of various nutrients*

Thus, nutritional therapy in ***patients with renal failure***

Requires a tight schedule of monitoring

Complications and monitoring of nutritional support

Note: By starting nutrition

(Enteral and parenteral)

At a *low infusion rate* and by *gradually increasing intake*,

Utilization of nutrients can be ensured

And *development of metabolic complications minimized*

Outline

1- Pathophysiology

2- Nutritional therapy for patients with renal disease

Non-catabolic patients with stable chronic kidney disease (CKD)

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

Patients with AKI and HD/CAPD patients with acute catabolic disease

Solutions for nutritional support

Complications and monitoring of nutritional support

3- Nutritional management of renal transplantation (RTX)

Nutritional management of renal transplantation (RTX)

Pre-transplant period

Transplant period

Early post-transplant period

Late post-transplant period - long-term management

Points to be considered are

Nutritional management of renal transplantation (RTX)

Renal transplantation

Is the most common solid organ transplant procedure

From a *metabolic* and *nutritional point of view*,

Transplant recipients are

An *extremely complex and heterogeneous group* of subjects

In whom *only general recommendations on isolated aspects* can be given

Nevertheless, *nutrition interventions have a major impact*

On *short* and *long-term outcomes* after kidney transplantation

Nutritional management of renal transplantation (RTX)

Nutritional management

Of patients undergoing *renal transplantation*

Can be separated into

The *pre-transplant* period,
Peri-transplant surgery
Early post-transplant period,
Late post-transplant period,
And *long-term management*

Of these patients

Pre-transplant period

The **goal** of pre-transplant nutritional management is

To **optimize** outcomes

In the early and late post-transplant period

Pre-transplant nutritional intervention

Should avoid *volume over load* and *electrolyte derangements*,

Promote *adequate intake of protein and energy*

To decrease the risk of infection,

Enhance wound healing, and maintain muscle mass.

The optimal BMI for transplant candidates to be aimed for *remains a matter of controversy*, but certainly should not exceed 35 kg/m²

transplant period

During transplantation,

The **main task** is to *prevent and treat mild or severe overhydration*

Because *surgery is frequently associated with fluid retention*

Monitoring and correction of *electrolytes*

(Especially *potassium, phosphorus, magnesium*),

And *acid-base balance*

Is imperative

transplant period

In addition to the usual *postoperative stress* encountered by patients after surgery, renal transplant recipients receive *high doses of corticosteroids* Which accelerate protein catabolism

After surgery, *hyperglycemia* develops in *many non-diabetic patients*

Tight Glucose control during this period can
Reduce the risk of developing
Post-transplant diabetes mellitus (post-TX-DM)

In association with immunosuppressive therapy,
Hypophosphatemia and *hypomagnesemia* develops in many patients

Early post-transplant period

The principal objectives of nutritional management

In the **early post-transplant period** are:

- *Maintenance of visceral protein stores*

Despite activated **protein catabolism**

- *Promotion of wound healing*

- *Prevention of infection* associated with **surgery** and **immunosuppression**

- *Prevention of electrolyte complications* that

Accompany **immunosuppression** and **rapid changes in renal function**

Early post-transplant period

Renal transplant recipients

Regain renal function

At different times after transplantation

Some patients *may require HD for several days*

(*Delayed graft function DGF*)

The nutritional needs of these patients

Resemble those of

Other patients with AKI

Late post-transplant period - long-term management

Due to

Differences in the underlying disease,

The *extent of complications*

Such as *hypertension* or *cardiovascular disease*,

Variable graft function,

And *degree of CKD and associated metabolic consequences,*

Patients after RTX present

An extremely heterogeneous group of subjects

These patients require close metabolic monitoring

and a definitively individualized approach

in nutrition counselling and metabolic management

Points to be considered area

When an **RTX patient** is admitted to the hospital

Because of *acute intermittent disease processes*,

Metabolic management and *nutritional support*

Should follow the

Recommendations made for patients with AKI

Summary

Outline

1- Pathophysiology

2- Nutritional therapy for patients with renal disease

Non-catabolic patients with stable chronic kidney disease (CKD)

Non-catabolic CKD patients on chronic RRT (with or without malnutrition)

Patients with AKI and HD/CAPD patients with acute catabolic disease

Solutions for nutritional support

Complications and monitoring of nutritional support

3- Nutritional management of renal transplantation (RTX)

Summary

In renal patients,

The *degree of impairment of renal function*,

Urine output,

Proteinuria,

Underlying disease process,

Electrolyte imbalances,

Hyperparathyroidism,

Bone disorder,

Hypertension,

Impaired glucose tolerance,

And *dyslipidemia*

Will all have an impact on metabolic management and nutritional therapy

Summary

This **broad pattern of factors**

Is not only different between patients

But may *profoundly vary during the course of disease*

In an individual patient

Summary

Renal insufficiency, per se,

Is a *pan-metabolic*

And *pan-endocrine abnormality*

Affecting more or less

Every metabolic pathway in the body

In *no other patient group* is there such a

Narrow range between

The risk of inducing toxic effects

And the *development of nutritional deficiencies or malnutrition*

Summary

Patients with CKD,

Even without concurrent disease,

Are at *high risk of* **malnutrition** due to

Uraemia-associated factors,

Inflammation,

Metabolic acidosis,

Impaired appetite and oral food intake,

And *gastrointestinal side effects of uraemia*

Summary

All types of RRT have a *fundamental impact* on

Metabolism and *nutrient balance*,

Not only because of *losses of nutrients*,

But also by *induction of an inflammatory reaction*

And *activation of protein catabolism*

Summary

In patients with **renal insufficiency**

Complicated by *acute catabolic disease*

And/or in *patients with AKI,*

The *stimulation of immunocompetence,*

Wound healing,

And other *reparative functions*

To overcome the acute disease

Are the *principal goals of nutritional therapy*

Summary

Nutritional assessment and *nutritional education*

Are of crucial importance

In all phases of renal transplantation,

Including the

Peritransplant period

And the long-term management of RTX patients

Summary

Nutrition therapy in **renal failure patients** certainly,

Is the most controversial and challenging field in clinical nutrition

Patients require *more intense training* and *cooperation*

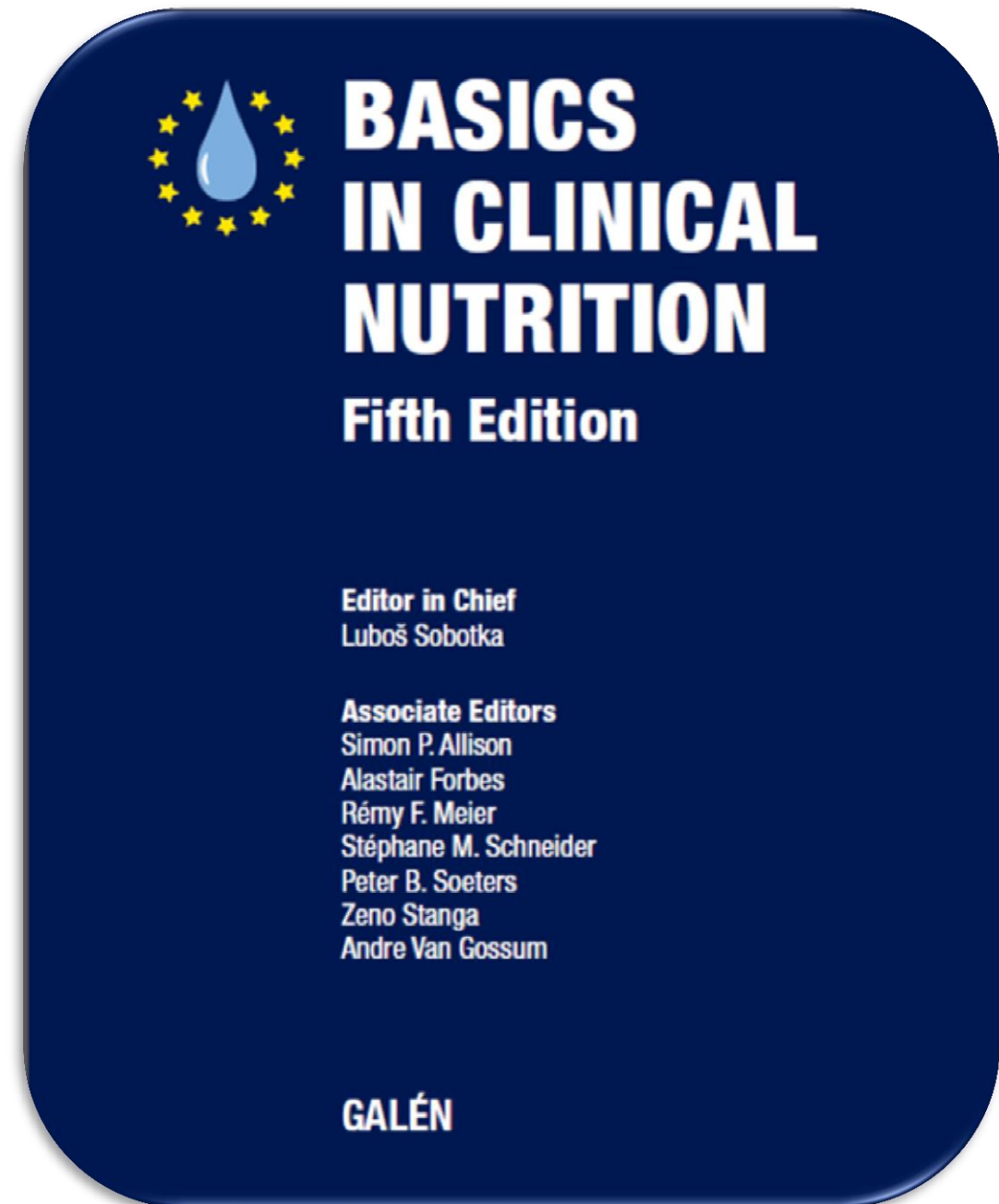
With a *multidisciplinary therapeutic team*,

And *close monitoring of metabolic and nutritional state*

More than in other medical fields, nutritional therapy must follow

An *extremely individualized approach adapted to each patient*

REFERENCE



The background of the slide is a close-up, high-resolution image of several orange slices. The slices are arranged in a slightly overlapping pattern, showing the vibrant orange color of the pulp and the lighter yellow of the pith. The lighting is bright, highlighting the texture of the fruit segments.

Thank you!