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Meeting Issue

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Lim Vadhana, Congress President

Reiseikai Media

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7th Annual Conference of Cambodian Association of Nephrology

14th, September 2024

Sokha Hotel Phnom Penh, Cambodia

Innovation in Kidney Care in Cambodia

The Cambodian Journal of Nephrology

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Remark by

Prof. Lim Vadhana

President of the Cambodian Association of Nephrology,

At the Seventh Annual Conference of the Cambodian Association of Nephrology

14th, September 2024, Phnom Penh, Cambodia

Greetings, highly respected professors, doctors, colleagues, students, ladies and gentlemen,

The incidence of kidney diseases has significantly increased over the past several years. This rise is a major concern for our society because, without proper care and treatment, the patients may progress to end-stage kidney disease, which requires advanced treatments such as hemodialysis or even renal transplantation. These conditions not only present daily challenges, including financial problems, but also place a burden on families and society as a whole. In response, our doctors, particularly nephrologists, are continuously seeking effective solutions to address this issue, which is supported by the guidance and policies of our government and Ministry of Health, as well as the substantial assistance and collaboration from our partner, Japan.

At today's conference, we will hear from international doctors from Japan and Singapore in the first and second sessions, respectively, followed by presentations from our local doctors in the third session. This year's conference is particularly special as we are introducing our new generation of nephrologists as speakers. Moreover, the presentations will cover a wide range of topics, including hemodialysis, chronic kidney failure, acute kidney injury, glomerulonephritis, as well as many other interesting topics. The main purpose is to raise awareness among the public and medical professionals about the prevention, diagnosis, and management of kidney diseases in order to enhance the well-being of the Cambodian people.

I strongly hope this occasion will be a valuable opportunity for all of us, the participants, to expand our knowledge in nephrology by discussing and sharing information and updates related to kidney diseases. This conference will also benefit our young nephrologists, helping them to further develop their expertise in the field. By doing so, we can improve our ability to provide better care and treatment for our patients.

Last but not least, I want to express my sincere gratitude for your precious participation. I wish you all the very best and am honored to announce the opening of the 7th Annual Conference of the Cambodian Association of Nephrology.

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- Lecturer of Nephrology at University of Health Sciences

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THIM Pichthida M.D., M.H.A.
- Pediatrician in the Nephrology Department at National Pediatric Hospital
- Director of Khmer Kids Pediatric Clinic

The Seventh Annual Conference of Cambodian Association of Nephrology

Innovation in Kidney Care in Cambodia Program

Date : 14th, September 2024 Venu: Sokha Hotel Phnom Penh, Cambodia

OPENING SESSION

7 :30- 8 :15 Arrival of Participants, Delegates and Speakers (Registration)

8 :15- 8 :45 Coffee Break

8 :45- 8 :50 National Anthem

8 :50- 9 :00 Welcoming and Opening Speech by Prof. LIM Vadhana, President of CAN

Session 1 Moderator: Prof. SVAY Kamol

9 :00- 9 :20 Effect of Dialysis Operating Conditions on Solute Removal, by Prof. Ryoichi Sakiyama, Japan

9 :20- 9 :40 Dietary therapy tailored to dialysis treatment, by Prof. Yukie Kitajima, Japan

9 :40- 9 :50 KALBE Presentation

9 :50-10:00 TELPHA Presentation

10:00-10:10 DKSH-Roche Presentation

10:10-10:20 SEARLE Presentation

SESSION 2 Moderator: Dr. SIN Bochhavan

10:20-10:40 Management of CKD-MBD in the aim of organ protection, by Prof. Hitoshi Minakuchi, Japan

10:40-11:00 Understanding and Managing IgA Nephropathy, by Dr. Liew Seng Teck Adrian, Singapore

11:00-11:10 DKSH-Abbott Presentation

11:10-11:20 METAMED Presentation

11:20-11:30 DYNAMIC-NIPRO Presentation

11:30-11:40 Absolute Presentation

11:40-12:30 Panel Discussion (Prof. LIM Vadhana, Dr. LIM Sochun, Dr. NIV Rathvirak, Dr. RITH Sovannara, Dr. SIN Bochhavan and Speakers)

12:30-13:30 Lunch

Session 3 Moderator: Dr. CHY Tith

13:30-13:50 Contrast Induced Nephropathy: Current Practice, by Dr. HOV Chomroeun, Calmette Hospital

13:50-14:10 The Management of Drug-induced Acute Renal tubular Necrosis, by Dr. SENG Raksmey, Calmette Hospital

14:10-14:30 Practical and Cost-Effective Applications of the Furosemide Stress Test in AKI Management, by Dr. LON Virithy, KSFH

14:40-14:40 KALBE Presentation

14:40-14:50 TELPHA Presentation

14:50-15:00 Max Presentation

15:00-15:30 Panel Discussion (Prof. LIM Vadhana, Dr. LIM Sochun, Dr. NIV Rathvirak, Dr. RITH Sovannara, Dr. SIN Bochhavan and Speakers)

15:30-16:00 Remise certificate, Closing, Lucky draw

Sponsor by:

Dietary therapy tailored to dialysis treatment

Yukie Kitajima Ph.D., R.D.

Dept. of Medical Nutrition, Tokyo Healthcare University

The goals of dietary therapy in chronic kidney disease are to slow the decline in renal function, prevent the onset and severity of hemodialysis complications, and prevent nutritional disorders. The basics of a dialysis diet are salt and water restriction, adequate energy and protein intake, and potassium and phosphorus restriction. Phosphorus management is difficult and can accelerate the onset of complications, especially when sufficient dialysis or administration of phosphorus-lowering drugs (phosphate binders) is not possible. The maximum intake of phosphorus is about 800 mg per day, and any more than that will accumulate in the bloodstream. One can absorb accumulated excess phosphorus by using a phosphate binder. However, this assumes that one's phosphorus intake is regulated by food consumption. The higher the variety and amount of food consumed, the higher the intake of phosphorus. The amount of phosphorus in grains is low, while beans and nuts are high in phosphorus. Phosphorus is also not abundant in fruits and vegetables. On the other hand, seafood, meat, dairy products, processed foods, and organs are high in phosphorus. Organic phosphorus is found in natural foods such as meat and vegetables as phosphoric acid bound to proteins. As a result, organic phosphorus is easily hydrolyzed and rapidly absorbed by the intestines. The absorption rate for phosphorus from meat is 50%, while the absorption rate for organic phosphorus found in fruits and vegetables is 30%. Unlike organic phosphorus, inorganic phosphorus found in food additives is not bound to proteins and is therefore rapidly absorbed by the intestines. The absorption rate for inorganic phosphorus is close to 100%. We need to assess what foods our patients eat most often and then guide them in making the necessary dietary changes. Educating them on how to eat well and make good choices will help maintain their nutritional status and prevent the development of complications. However, if standard hemodialysis (3 times/week, 4 hours/treatment) is not possible, a low-protein diet therapy should be used.

Understanding and Managing IgA Nephropathy

Liew Seng Teck Adrian M.D.

Mount Elizabeth Novena Specialist Centre, Singapore

IgA Nephropathy is the most common biopsy-proven glomerulonephritis globally, often affecting young patients at the most economically productive period of their lives. While generally considered to be a slow progressive disease, the lifetime risk of end-stage kidney failure remains high. There is a well-recognized ethnicity driven risk of kidney failure with Asians generally accepted to have a more aggressive course. Incidence of the condition continues to be a gross underestimate globally and is determined by biopsy practice, access to health screening and health seeking behaviors. Currently, proteinuria remains the key biomarker that is used to identify individuals at high risk of disease progression with anti-proteinuria therapy, therefore, being long accepted as the standard of care in patients with IgA nephropathy. Recently, the addition of SGLT-2 inhibitors has been shown to improve renal survival outcomes in subgroup analysis of large-scale CKD clinical trials where a significant proportion of IgA nephropathy patients were included in the study population. Despite this, a high proportion of patients continue to develop kidney failure requiring kidney replacement therapy. Until recently, corticosteroids had been the only immunosuppression therapy recommended by guidelines for treatment of high risk individuals but unfortunately, were associated with a high prevalence of unwanted side-effects including life-threatening infections. The elucidation of the pathogenesis of IgA nephropathy over the last 2 decades has improved our understanding of the course of the disease with increased levels of poorly galactosylated IgA1 (Gd-IgA1) being recognized as the key inciting event of the condition. Consequently, this has generated significant interests in testing novel therapeutic agents targeting different aspects of the pathogenic pathway in large number of well-conducted clinical trials. Endothelin A receptor antagonists are set to be included as an agent targeting the downstream cellular proliferation and fibrotic effects of the disease, with studies showing their effectiveness in further lowering proteinuria on top of RAS blockade. More importantly, new studies have started to demonstrate the effectiveness of novel drugs in ameliorating the upstream effect of Gd-IgA1. This can be achieved through a targeted-release budesonide or agents blocking BAFF and/or APRIL pathway, opening up an exciting area of a potential ability to control IgA nephropathy at its root cause. Finally, complement inhibitors affecting different pathways of the complement cascade is being investigated as a potential alternative to systemic corticosteroids in reducing the inflammatory damage and progression of CKD in this patient population. Indeed, the number of therapeutic options for the treatment of IgA nephropathy will increase substantially in the next few years and the clinical practice guidelines are likely to have to keep up with the pace of drugs being approved for this condition.

Contrast Induced Nephropathy : Current Practice

HOV Chomroeun M.D.

Calmette Hospital

Radiological procedures utilizing intravenous iodinated contrast agents are being widely utilized for both therapeutic and diagnostic purposes. This has resulted in an increasing incidence of procedure-related, contrast-induced nephropathy (CIN) when the kidney function altered severely in particularly. CIN is commonly defined as a decline in kidney function occurring in a narrow time window after administration of iodinated contrast agents. Although self-limiting in most cases, CIN carries a risk of more permanent renal insufficiency, dialysis, and death.

It remains a common and serious complication among at-risk patients after exposure of contrast agents. Therefore, it is important to identify patients who are at risk during early stages to implement preventative strategies to decrease the incidence of CIN.

Minimizing the amount of contrast administered and providing adequate hydration are the cornerstones of an effective preventative approach. This topic focuses on the basic concepts of CIN and summarizes the current understanding of its pathophysiology. In addition, it provides current practical recommendations with respect to CIN prevention and management.

Practical and Cost-Effective Applications of the Furosemide Stress Test in AKI Management

LON Virithy M.D.

Khmer Soviet Friendship Hospital

Acute kidney injury is a serious condition that increases morbidity and mortality in hospitalized patients. progress to severe AKI requiring renal replacement therapy (RRT) is crucial for timely intervention and resource allocation. Despite advances in AKI management, there remains a critical need for reliable prognostic tools to guide clinical decision-making.

The Furosemide Stress Test (FST) has emerged as a pivotal diagnostic tool in the clinical assessment of acute kidney injury. The FST, involving a single dose of the safe and inexpensive diuretic furosemide and measuring urine output response, provides a dynamic assessment of kidney function. FST has significant benefits in predicting the need for renal replacement therapy, including hemodialysis, and in forecasting the recovery of kidney function in AKI patients on dialysis.

Key points include the FST's physiological basis, methodology, and interpretation. Evidence indicates that the FST is a reliable predictor of severe AKI progression and the need for hemodialysis. Additionally, the FST predicts kidney function recovery in dialysis patients. The test's cost-effectiveness and safety enhance its clinical utility, often outperforming similar tests in AKI management.

In conclusion, the FST is a valuable addition to the diagnostic tools for AKI, aiding in early detection of patients at risk for hemodialysis and supporting predictions of kidney function recovery. Its dual utility enhances clinical decision-making, optimizes patient management, and improves outcomes, making it a cost-effective and safe approach for better patient care.

The Management of Drug-Induced Acute Tubular Necrosis

SENG Raksmey M.D.

Calmette Hospital, Phnom Penh, Cambodia

Drug-induced acute tubular necrosis (ATN) is a significant and potentially life-threatening condition characterized by the rapid deterioration of renal function due to toxic effects of medications on renal tubular cells and is the common cause of acute kidney injury (AKI). Antibiotics are one of the most common causes, especially Vancomycin and aminoglycoside.

The pathophysiology of drug-induced ATN involves direct toxicity to renal tubular epithelial cells, leading to cellular dysfunction, necrosis, and impaired tubular reabsorption. Risk factors such as preexisting renal impairment, advanced age, proteinuric diabetic or hypertensive, hypotension in liver and heart failure and high cumulative doses of nephrotoxic drugs increase susceptibility to ATN.

Clinical presentation varies from asymptomatic renal dysfunction to severe acute kidney injury (AKI)

with oliguria, fluid overload, electrolyte disturbances, and uremia. Diagnosis is typically made based on clinical history, detailed medication history, laboratory findings (e.g., elevated serum Cheatinine, decreased urine output), and sometimes renal biopsy may be necessary in ambiguous cases to confirm the diagnosis.

Management strategies focus on discontinuing the offending drug promptly, supportive care including fluid and hemodynamical management, electrolyte correction, and renal replacement therapy in severe cases. Prevention involves vigilant monitoring of renal function in patients at risk, dose adjustment in those with impaired renal clearance, and avoidance of nephrotoxic drug combinations whenever possible.

In conclusion, drug-induced ATN represents a critical clinical entity necessitating early recognition and intervention to mitigate renal injury and improve patient outcomes. Healthcare providers should remain vigilant in identifying potential nephrotoxic insults and managing them appropriately to minimize morbidity and mortality associated with drug-induced ATN.

KEYWORDS: Acute kidney injury (AKI), Drug-induced Acute tubular necrosis (ATN), Antibiotic, Fluid management, Renal Replacement Therapy (RRT)

Effect of Dialysis Operating Conditions on Solute Removal

Ryoichi Sakiyama Ph.D.
Department of Biomedical Engineering
Osaka Institute of Technolog

Hemodialysis is one of the most popular treatments for renal failure patients in the world.

In hemodialysis, uremic toxins are removed from the blood to the dialysate side by diffusion based on the difference in solute concentration between the blood and dialysate sides. Clearance, the removal performance of the dialyzer, increases with increased blood and dialysate flow rates. Therefore, for the same duration time of treatment, the flow rate operating conditions will affect the blood of the patient's solute removal and thus the treatment effect. However, while increased blood flow increases clearance, it also increases fouling, and the adhesion of proteins to the membrane surface, and there is concern that fouling may degrade membrane performance.

The ß2 micro globulin (ß2MG) (molecular weight 11800) is an important marker of the removal effect of dialysis. In a 2013 JSDT report, the risk of death in patients with pre-dialysis ß2MG concentrations of 25-30 mg/L, assuming 1.0, decreased to 0.907 at 20-25 mg/L, while it increased to 1.281 at 30-35 mg/L. Maintaining low blood ß2MG levels in patients with renal failure by increasing ß2MG clearance and providing treatment three times a week is important in terms of life prognosis.

This presentation will discuss the effect of improving clearance through operating conditions on solute removal.

Management of CKD-MBD in the aim of organ protection

Hitoshi Minakuchi M.D., Ph.D.
National Defense Medical College of Japan

CKD-MBD (Chronic Kidney Disease-Mineral and. Bone Disorder) is defined as systemic disease including renal osteodystrophy and vascular complications, and we have to control the concentrations of serum Ca, IP, iPTH in hemodialysis patients with appropriate methods. There are guidelines for CKD-MBD of hemodialysis patients, for example, KDIGO (Kidney Disease Improving Global Outcomes, World), KDOQI (Kidney Disease Outcomes Quality Initiative ,USA), JSDT (Japanese Society for Dialysis Therapy, Japan), and some differences exist among these guidelines. New evidence came over recently, episode study 2021 proved that more severe control of serum IP ameliorate the progression of coronary artery calcification, and Parathyroidectomy vs calclmimetics study 2022 revealed that severe control of serum Ca, IP, iPTH with parathyroidectomy improve the mortality of hemodialysis patients.

Enough dialysis dose, dietary restriction, and suitable prescription of phosphate binders are important to achieve severe control of serum IP. It is known that proton pump inhibitors which are frequently prescribed to hemodialysis patients decrease the effect of phosphor binders such as calcium carbonate and lanthanum carbonate. Adding on CKD-MBD control, the load of Mg, Fe, Zn and vitamin K or correction of pre-dialysis metabolic acidosis might be effective to avoid artery calcification. We have to think about osteoporosis among hemodialysis patients to reduce bone fracture, but there are few evidences for the osteoporosis among hemodialysis patinents.

Nutritional advantage of pre-dilution on-line hemodiafiltration for end stage renal patients

Motoko Kato [1, 2], Shunichiro Urabe[1, 3], Akira Kato[1, 2], Shohei Matsuzawa[2], Momoko Hukazawa[2], Kamol Svay[4], Sovandy Chan[4, 5], Yukie Kitajima[1, 5, 6], Kenichi Kokubo[1, 5, 7], Toru Hyodo[1, 4, 5]

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2: Eijin Clinic, Hiratsuka, Japan
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7: Kitasato University School of Allied Health Sciences, Sagamihara, Japan

Key words
Pre-dilution on-line hemodiafiltration, Hemodialysis, Amino acid, Nutrition, Sarcopenia, Frailty

Abbreviations

Pre O-HDF : pre-dilution on-line hemodiafiltration		HDF : hemodiafiltration
HD : hemodialysis	CKD : chronic kidney disease	PEW : protein-energy wasting
NEAAs : nonessential amino acids	TAAs : total amino acids	EAAs : essential amino acids
BCAAs : branched-chain amino acids	CS : clear space	Cpre : pre-dialysis concentration
m : milligram	Kt/V : K: clearance, t: time, V: volume	
AA : amino acid	Post O-HDF : post-dilution on-line HDF	

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Introduction

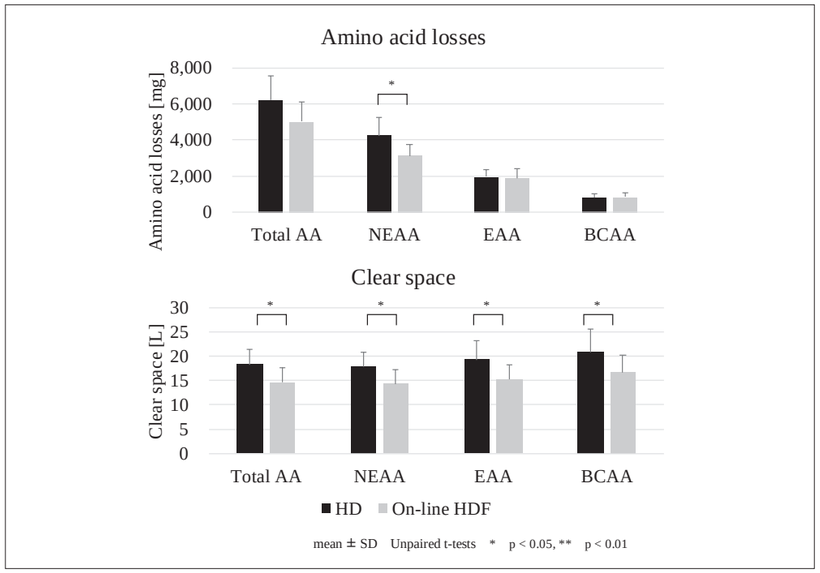
In Japan, pre-dilution on-line hemodiafiltration (Pre O-HDF) is known to yield better overall survival than hemodialysis (HD). In particular, Pre O-HDF with a high substitution volume (≥40.0 L/session) has been shown to yield better all-cause and cardiovascular survival than Pre O-HDF with a low substitution volume (<40.0 L/session) or HD [1].

It is well established that patients on HD become malnourished and have an altered catabolic rate [2-4], and malnutrition has been shown to be associated with reduced quality of life and increased mortality among patients on HD [5, 6]. The multiple nutritional and catabolic alterations that occur in chronic kidney disease (CKD) was termed "protein-energy wasting (PEW)" by the International Society of Renal Nutrition and Metabolism [7]. PEW is the result of multiple mechanisms inherent to CKD, including undernutrition, systemic inflammation, comorbidities, hormonal derangements, the dialysis procedure, and other consequences of uremic toxicity [7].

Several studies have demonstrated that amino acid and protein losses during the dialysis session combined with low nutrient intake result in a lack of nutrients for muscle synthesis [8-11], indicating that dialysis treatment itself affects the protein and energy homeostasis of patients. Moreover, a substantial increase in net skeletal muscle protein catabolism occurs during dialysis treatment [12,13] and the undesirable effects of which persisted for at least 2 hours after the completion of HD [12].

There are two possible approaches to address this issue in blood purification therapy. The first is to minimize loss of beneficial substances such as albumin and amino acids through dialysis. The second approach is to administer an intravenous amino acid solution for patients with renal failure to HD patients with poor nutritional status during dialysis. This method has long been used in conventional HD in Japan.

Figure 1: Amino acid loss and their clear spaces of patients on on-line HDF and HD



Patients on Pre O-HDF lost significantly less NEAAs than patients on HD. Patients on Pre O-HDF also showed significantly lower clear spaces for total AAs, NEAAs, EAAs, and BCAAs than patients on HD.

Amino acid leakage in HD and Pre O-HDF at equal Kt/V for urea

We investigated differences in amino acid losses between HD and Pre O-HDF with equal Kt/V for urea to determine which modality is gentler, less invasive, more biocompatible, and ensures better nutrition [14]. Patients on Pre O-HDF lost significantly less glutamine and arginine ($p<0.01$ and $P = 0.032$) and significantly less nonessential amino acids (NEAAs) than patients on HD ($P = 0.013$). They also showed significantly lower their clear spaces of total amino acids (TAAs), NEAAs, essential amino acids (EAAs), and branched-chain amino acids (BCAAs) than patients on HD (TAA: $p = 0.019$, NEAA: $p = 0.018$, EAA: $p = 0.024$, BCAA: $p = 0.042$) **[Fig.1]**.

Yamashita reported that loss of solutes with different pre-dialysis concentrations is better to be calculated as a ratio of the two and compared because the loss of solutes and pre-dialysis concentration are directly proportional [15]. Clear space (CS) denotes the volume at which the concentration of the solute of interest becomes zero. CS was calculated using the following equation.

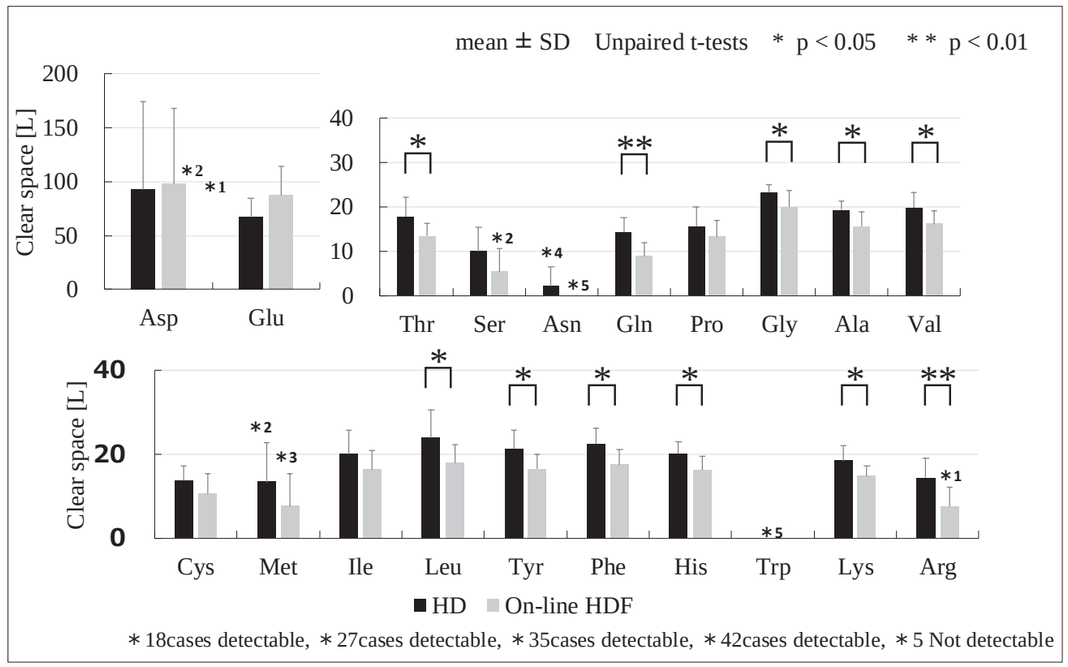
$$CS = m / Cpre$$

Here, Cpre is the pre-dialysis concentration of amino acids and m is milligram.

When an amino acid has a large clear space, it means that a large proportion of that amino acid is removed relative to its blood concentration, and that a large volume of amino acids flows from extravascular pools into

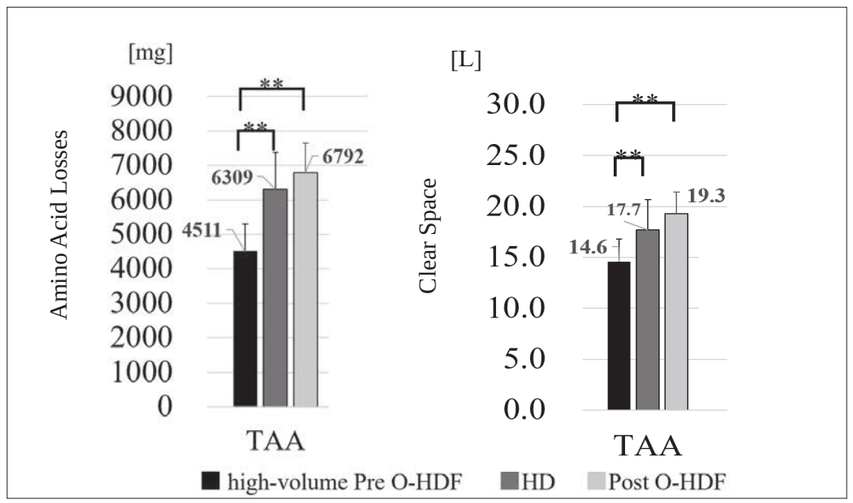
Review article

Figure 2: Clear spaces for individual amino acids in patients on on-line HDF and HD



The amino acid with the greatest clear space in patients on either dialysis modality was aspartic acid, followed by glutamic acid.

Figure 3: Total amino acids loss and their clear spaces in high-volume Pre O-HDF, HD and Post O-HDF



High-volume Pre O-HDF showed significantly the lower total amino acid losses and their clear spaces than HD and Post O-HDF.

blood vessels. The amino acid with the greatest CS in patients on either dialysis modality in this study was aspartic acid, followed by glutamic acid **[Fig.2]**.

It can be said that because a smaller loss of amino acids was observed with Pre O-HDF when Kt/V for urea was equal, the amino acids volume that flowed into the blood from muscles and other amino acid pools was lower. This suggests that the protein catabolic rate is lower with Pre O-HDF.

Significance of amino acid leakage in the high-volume Pre O-HDF

In our hospital, hemodiafilters, through which albumin leakage is slight, are used in elderly HD patients or those with malnutrition. In addition, high-volume Pre O-HDF is performed to reduce the diffusion efficiency of micromolecular substances. However, the degree of amino acid (AA) (micromolecular substance) leakage remains to be clarified. In this study, we compared AA leakage among high-volume Pre O-HDF, HD, and post-dilution on-line HDF (Post O-HDF).

AA leakages after high-volume Pre O-HDF were significantly lower than HD and Post O-HDF. The CSs after high-volume Pre O-HDF were significantly lower than HD and Post O-HDF **[Fig.3]**.

High-volume Pre O-HDF reduces AA leakage in comparison with HD and Post O-HDF. This blood purification method may be the most advantageous in preventing sarcopenia and frailty from the viewpoint of nutrition and dietetics.

Summary and Conclusions

When the body experiences trauma from an invasive procedure, it increases metabolism in an attempt to repair damaged tissues [16]. The body cannot meet this increased energy demand with sugar alone, so it breaks down muscle protein and uses amino acids such as glutamine and alanine, which are then released into the bloodstream to provide energy and repair damaged tissue. Because HD is an invasive procedure, it can be presumed that not only does HD directly leads to the loss of amino acids, but also that the body reacts to the trauma of bioincompatible extracorporeal circulation by increasing energy metabolism and catabolic rate. Low-molecular-weight amino acids are subsequently lost through HD and HDF, which remove substances through diffusion and convection. In fact, studies have shown that 4–12 g of amino acids are removed in a single dialysis session [17-20]. However, an increase in the protein catabolic rate and protein breakdown may result in excessive removal of amino acids. A gentler, less invasive, and more biocompatible dialysis technique would theoretically reduce amino acid losses. In elderly patients, the muscle protein synthetic response to protein ingestion is reduced. [21] Furthermore, the plasma AA leaks during dialytic therapy by diffusion and convection. The decomposition of the skeletal muscle may progress in order to replenish a lack of the pooled plasma AAs. [22] Considering these negative aspects of dialysis therapy, the treatment conditions for reducing AA leakage are required to prevent sarcopenia and frailty, which have recently been emphasized, especially in elderly patients receiving dialysis.

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A simple diabetes diet instruction book for Cambodian citizens [1]: How to do Basic Carbohydrate Counting: Easy Diabetic Dietary Therapy

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1: What is the dietary component to become blood sugar?

Food is made up of three nutrients. These are carbohydrates, proteins and fats. Of these, carbohydrates are the main source of blood glucose when eaten. Protein and fat do not turn into blood sugar. Strictly speaking, only a little goes into blood glucose. As shown in Figure 1, blood glucose rises more after a meal of rice than after a meal of steak and butter, which have more calories. In other words, it is not the calories that affect blood glucose after a meal, but how much carbohydrate is consumed [1, 2]. **Figure 2** summarizes the relationship between food type and postprandial blood glucose in a simple table. Only foods that contain carbohydrates result in blood glucose. Staple foods are mainly carbohydrates. They are rice, bread and noodles. In Cambodia it would be rice. Milk, vegetables, and fruits are not staple foods, but they do contain some carbohydrates that raise blood glucose after a meal.

2: What is the Basic Carbohydrate Counting?

According to the fact that only carbohydrate raises blood glucose, not fat, not protein, the basic carbohydrate counting (indication for all diabetic patients) is to teach the diabetic patients that only carbohydrate raises blood glucose, not fat, not protein, and to teach the diabetic patients that the patient should eat the same amount of carbohydrate at every meal.

Figure 3 shows an image of the basic carbohydrate counting. To make the amount of carbohydrates easier to understand, the amount of carbohydrates is represented by 1 cup of rice. The basic carbohydrate counting keeps postprandial blood glucose constant by keeping the amount of rice, or carbohydrate, in the staple food constant. In the diabetic patients, before the introduction of basic carbohydrate counting, the amount of rice eaten at each meal is not constant, so that, for example, the green dotted line blood glucose level is also high after dinner. Thus, even the same amount of rice per day will result in hyperglycemia. After the introduction of basic carbohydrate counting, the amount of rice eaten at each meal is constant, so the postprandial blood glucose changes at each meal also show an even pattern for the three meals, as shown by the blue line. This pattern shows a slight increase in blood glucose after each meal. This is the pattern of blood glucose changes in a healthy person without

diabetes. So the basic carbohydrate counting is to keep the amount of carbohydrate at each meal constant. **Figure 4** shows the approximate amount of rice. One handful is about 200 grams of rice. The amount of rice per serving for a diabetic adult is about 200 g. The amount of carbohydrates is about 75 g. **Figure 5** shows an example of the basic carbohydrate counting in the Cambodian diet. The staple meal of rice is 200 g per meal, consumed equally in each of the three meals.

3: Basic Carbohydrate Counting Tips

Most of the carbohydrates in the diet come from staple foods. In other words, we only need to focus on the amount of rice, noodles, and bread. Main dishes such as entrees and side dishes contain little or no carbohydrates. By simply keeping track of the carbohydrates in rice, noodles, and bread, and making sure that the same amount of carbohydrates is consumed at all three meals, the diabetic patients will have similar daily blood glucose patterns as healthy people. Carbohydrate is almost from staple food. It is roughly enough to calculate carbohydrate of staple food.

4: Foods with and without carbohydrates

Figure items 6 through 13 are foods that contain carbohydrates. **Figures 14-17** are side dishes that contain a little carbohydrate but not too much, called main and side dishes. **Items 18-20** are vegetables that contain little or no carbohydrates. **Number 21** is tea, water, and artificial sweeteners, which contain no carbohydrates.
Figures 22 through 28 show specific examples of food items that are primarily staple foods, their weights, and carbohydrate content. The 200 g of rice in **Figure 22**, which shows 10 g of carbohydrates as 1 carbo, contains 74 g of carbohydrates, which means it contains about 7.5 carbo. **Figure 23** shows that a cup of noodles contains 36.4 g of carbohydrates, which is about 3.5 carbo. **Figure 24** is a 5.5 carbc food, **Figure 25** is a 6 carbo food, **Figure 26** is a 7 carbo food, and **Figure 27** is a 3.0 carbo and 1.5 carbo food. The potato in **Figure 26** would be a 2.5 carbo food.

សៀវភៅណែនាំអំពីរបបអាហារសម្រាប់អ្នកជំងឺទឹកនោមផ្អែម សាមញ្ញសម្រាប់ប្រជាជនខ្មែរ [១] ៖ របៀបធ្វើការគណនាកាបូអ៊ីដ្រាតមួយដង្ហាន ៖ ការព្យាបាលដោយរបបអាហារសម្រាប់អ្នកជំងឺទឹកនោមផ្អែម ដែលងាយស្រួល

ផុន អេលីន [១,២], នៅ តាំ [១,២], តាន់ សុភ័ក្ត្រ [១,២], គីម សាំអូំ [១,២], ស៊ូ សុខជា [១,២], គង់ យី [១], ធីម ពិជ ធីតា [១,២], យិម សុវណ្ណបុញ្ញ [១,២], សុយ កាមល់ [២], ចាន់ សុវណ្ណី [១,២], សាគូមី ហាហ្គ [៣], តួរ ហ៊ីយ៉ូដូ [១,២,៣], យូគី គីតាជីម៉ា [១,៣,៤]

១: សាកលវិទ្យាល័យអន្តរជាតិ មហាវិទ្យាល័យវេជ្ជសាស្ត្រ ភ្នំពេញ កម្ពុជា
២: សមាគមតម្រង់នោមកម្ពុជា ភ្នំពេញ កម្ពុជា
៣: អង្គការសំអាតឈាមអន្តរជាតិ យូកូហាម៉ា ជប៉ុន
៤: នាយកដ្ឋានអាហារពេទ្យ សាកលវិទ្យាល័យវេជ្ជសាស្ត្រតូក្យូ តូក្យូ ជប៉ុន

ពាក្យគន្លឹះ ជំងឺទឹកនោមផ្អែម ការគណនាកាបូអ៊ីដ្រាតមួយដង្ហាន ការព្យាបាលដោយរបបអាហារ ទំនាក់ទំនងអ្នកនិពន្ធ វេជ្ជបណ្ឌិត ផុន អេលីន និង វេជ្ជបណ្ឌិត បណ្ឌិត តួរ ហ៊ីយ៉ូដូ

១. អ្វីទៅជាសមាសធាតុអាហារដែលក្លាយជាស្ករក្នុងឈាម?

អាហារមានសមាសធាតុចិញ្ចឹមបីប្រភេទ រួមមានកាបូអ៊ីដ្រាត ប្រូតេអ៊ីន និងខ្លាញ់ ។ ក្នុងចំណោមនេះ កាបូអ៊ីដ្រាតជាប្រភពសំខាន់នៃជាតិស្ករក្នុងឈាមនៅពេលញ៉ាំ ។ ប្រូតេអ៊ីន និងខ្លាញ់មិនប្រែក្លាយទៅជាស្ករក្នុងឈាមទេ ។ តាមពិត មានតែបរិមាណតិចតួចប៉ុណ្ណោះដែលប្រែក្លាយទៅជាស្ករក្នុងឈាម ។ ដូចបានបង្ហាញក្នុងរូបភាពទី១ ជាតិស្ករក្នុងឈាមកើនឡើងច្រើនបន្ទាប់ពីញ៉ាំបាយ ជាងបន្ទាប់ពីញ៉ាំសាច់គោ និងប៊ឺ ដែលមានកាឡូរីច្រើន ។ និយាយដោយឡែក វាមិនមែនជាកាឡូរីដែលប៉ះពាល់ដល់ជាតិស្ករក្នុងឈាមបន្ទាប់ពីញ៉ាំអាហារនោះទេ ប៉ុន្តែជាបរិមាណកាបូអ៊ីដ្រាតដែលបានបរិភោគ [១, ២] ។ រូបភាពទី២ បង្ហាញពីទំនាក់ទំនងរវាងប្រភេទអាហារ និងជាតិស្ករក្នុងឈាមបន្ទាប់ពីញ៉ាំអាហារក្នុងតារាងសាមញ្ញមួយ ។ មានតែអាហារដែលមានផ្ទុកកាបូអ៊ីដ្រាតប៉ុណ្ណោះដែលបណ្តាលឱ្យមានជាតិស្ករកើនក្នុងឈាម ។ អាហារសំខាន់ប្រចាំថ្ងៃគឺជាប្រភេទអាហារសំបូរកាបូអ៊ីដ្រាត ។ វាមានដូចជាជាបាយ នំបុ័ង និងមី ។ បើនៅកម្ពុជាអាហារដែលអាហារសំខាន់ប្រចាំថ្ងៃ (staple food) គឺជាបាយ ។ ទឹកដោះគោ បន្លែ និងផ្លែឈើមិនមែនជាអាហារអាហារសំខាន់ប្រចាំថ្ងៃ (staple food) នោះទេ ប៉ុន្តែវាមានផ្ទុកកាបូអ៊ីដ្រាតមួយចំនួនដែលបណ្តាលឱ្យមានជាតិស្ករក្នុងឈាមបន្ទាប់ពីញ៉ាំអាហារដែរ ។

២. ការគណនាកាបូអ៊ីដ្រាតមួយដង្ហាន

យោងតាមការសិក្សាដែលបញ្ជាក់ថា មានតែកាបូអ៊ីដ្រាតប៉ុណ្ណោះដែលបង្កើនជាតិស្ករក្នុងឈាម ហើយមិនមែនជាខ្លាញ់ ឬ ប្រូតេអ៊ីននោះទេ ការគណនាកាបូអ៊ីដ្រាតមួយដង្ហាន (សម្រាប់អ្នកជំងឺទឹកនោមផ្អែមទាំងអស់) គឺដើម្បីបង្រៀនអ្នកជំងឺទាំងនេះថា មានតែកាបូអ៊ីដ្រាតប៉ុណ្ណោះដែលបង្កើនជាតិស្ករក្នុងឈាម មិនមែនជាខ្លាញ់ ឬ ប្រូតេអ៊ីននោះទេ ហើយនិងត្រូវបង្រៀនអ្នកជំងឺទឹកនោមផ្អែមថា គួរតែញ៉ាំកាបូអ៊ីដ្រាតក្នុងបរិមាណដូចគ្នានៅគ្រប់ពេលអាហារ ។
រូបភាពទី៣ បង្ហាញពីរូបភាពនៃការគណនាកាបូអ៊ីដ្រាតមួយដង្ហាន ។ ដើម្បីធ្វើឱ្យបរិមាណកាបូអ៊ីដ្រាតងាយយល់ បរិមាណកាបូអ៊ីដ្រាតត្រូវបានតំណាងដោយបាយមួយពែង ។ ការគណនាកាបូអ៊ីដ្រាតមួយដង្ហានរក្សាលំនឹងជាតិស្ករក្នុងឈាមបន្ទាប់ពីញ៉ាំអាហារឱ្យស្ថិរភាពដោយរក្សាបរិមាណបាយ ឬ កាបូអ៊ីដ្រាត ក្នុងអាហារសំខាន់ប្រចាំថ្ងៃឱ្យនៅថេរ ។ ចំពោះអ្នកជំងឺទឹកនោមផ្អែម មុនពេលណែនាំការគណនាកាបូអ៊ីដ្រាតមួយដង្ហាន បរិមាណបាយដែលបានញ៉ាំនៅពេលអាហារនីមួយៗមិនថេរនោះទេ ដូចឧទាហរណ៍ កម្រិតជាតិស្ករក្នុងឈាម បន្ទាត់ពណ៌បៃតងព្រិលៗ ក៏ខ្ពស់បន្ទាប់ពីអាហារពេលល្ងាចផងដែរ ។ ដូច្នេះ ទោះបីជាបរិមាណអង្ករដូចគ្នាក្នុងមួយថ្ងៃក៏បណ្តាលឱ្យមានជាតិស្ករក្នុងឈាមខ្ពស់ដែរ ។ បន្ទាប់ពីណែនាំការគណនាកាបូអ៊ីដ្រាតមួយដង្ហាន បរិមាណបាយដែលបានញ៉ាំនៅពេលអាហារនីមួយៗមានស្ថិរភាព ដូច្នេះការផ្លាស់ប្តូរជាតិស្ករក្នុងឈាមបន្ទាប់ពីញ៉ាំអាហារនីមួយៗក៏បង្ហាញពីលំនាំស្មើៗគ្នាសម្រាប់អាហារទាំងបីពេល ដូចបានបង្ហាញដោយបន្ទាត់ពណ៌ខ្សៀវ ។ លំនាំនេះបង្ហាញពីការកើនឡើងបន្តិចនៃជាតិស្ករក្នុងឈាមបន្ទាប់ពីអាហារនីមួយៗ ។ នេះគឺជាលំនាំនៃការផ្លាស់ប្តូរជាតិស្ករក្នុងឈាមរបស់មនុស្សដែលមានសុខភាពល្អដែលមិនមានជំងឺទឹកនោមផ្អែម ។ ដូច្នេះការគណនាកាបូអ៊ីដ្រាតមួយដង្ហានគឺដើម្បីរក្សាបរិមាណកាបូអ៊ីដ្រាតនៅពេលអាហារនីមួយៗឱ្យស្ថិរភាព ។ រូបភាពទី៤ បង្ហាញពីបរិមាណបាយប្រហាក់ប្រហែល ។ បាយ

Review article

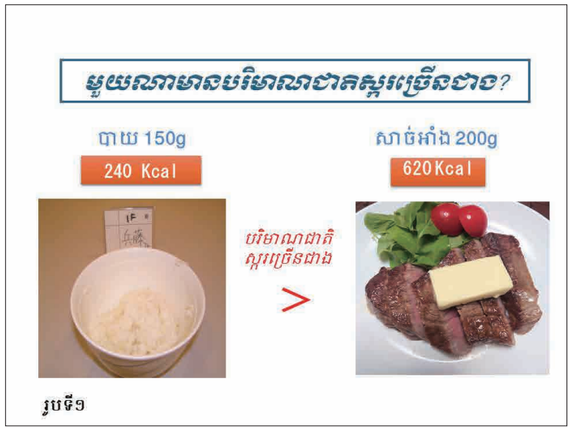
មួយកណ្តាប់ដៃមានទម្ងន់ប្រហែល ២០០ ក្រាម ។ បរិមាណបាយក្នុងមួយចំណែកសម្រាប់មនុស្សពេញវ័យដែលមានជំងឺទឹកនោមផ្អែមគឺប្រហែល ២០០ ក្រាម ។ បរិមាណកាបូអ៊ីដ្រាតគឺប្រហែល ៧៥ ក្រាម ។ រូបភាពទី៥ បង្ហាញពីឧទាហរណ៍នៃការគណនាកាបូអ៊ីដ្រាតមូលដ្ឋានក្នុងរបបអាហារខ្មែរ ។ អាហារសំខាន់ប្រចាំថ្ងៃគឺបាយដែលមានទម្ងន់ ២០០ ក្រាមក្នុងមួយពេល បរិភោគស្មើៗគ្នានៅគ្រប់អាហារទាំងបីពេល ។

៣. ការណែនាំស្តីពីការគណនាកាបូអ៊ីដ្រាតមូលដ្ឋាន

កាបូអ៊ីដ្រាតភាគច្រើននៅក្នុងរបបអាហារមកពីអាហារសំខាន់ប្រចាំថ្ងៃ ។ និយាយដោយឡែក យើងត្រូវតែផ្តោតលើបរិមាណបាយ មី និងនំបុ័ង ប៉ុណ្ណោះ ។ មុខម្ហូបសំខាន់ៗ ដូចជាមុខម្ហូបចាប់ផ្តើម (សំរន់) និងមុខម្ហូបបន្ថែម (ល្បោយ) មានផ្ទុកកាបូអ៊ីដ្រាតតិច ឬ គ្មាន ។ ដោយគ្រាន់តែតាមដានកាបូអ៊ីដ្រាតក្នុងបាយ មី និងនំបុ័ង និងធានាថាបរិមាណកាបូអ៊ីដ្រាតដូចគ្នាត្រូវបានបរិភោគនៅគ្រប់អាហារទាំងបីពេល អ្នកជំងឺទឹកនោមផ្អែមនឹងមានលំនាំជាតិស្ករក្នុងឈាមប្រចាំថ្ងៃដូចគ្នានឹងមនុស្សដែលមានសុខភាពល្អ ។ កាបូអ៊ីដ្រាតមកពីអាហារសំខាន់ប្រចាំថ្ងៃ (staple food) ស្ទើរតែទាំងអស់ ។ ដូចនេះ ការគណនាកាបូអ៊ីដ្រាតនៃអាហារសំខាន់ប្រចាំថ្ងៃ (staple food) ប្រហែលជាល្អគ្រប់គ្រាន់ ។

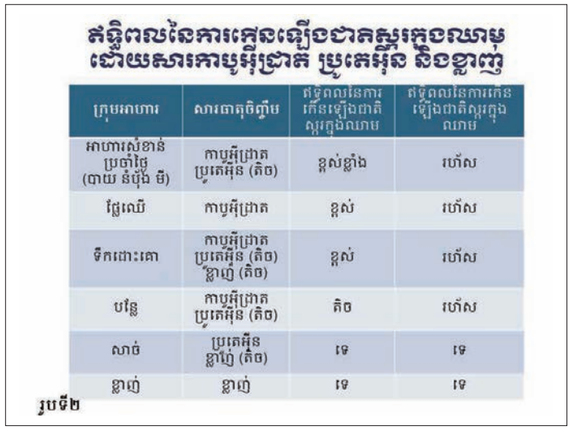
៤. អាហារដែលមាន និងគ្មានកាបូអ៊ីដ្រាត

រូបភាពទី ៦ ដល់ទី ១៣ គឺជាអាហារដែលមានផ្ទុកកាបូអ៊ីដ្រាត ។ រូបភាពទី ១៤ ដល់ទី ១៧ គឺជាមុខម្ហូបបន្ថែមដែលមានផ្ទុកកាបូអ៊ីដ្រាតតិច ប៉ុន្តែមិនច្រើនពេក ហៅថា មុខម្ហូបចម្បង និងមុខម្ហូបបន្ថែម(ល្បោយ) ។ រូបទី ១៨ ដល់ទី ២០ គឺបន្លែដែលមានផ្ទុកកាបូអ៊ីដ្រាតតិច ឬ គ្មាន ។ លេខ ២១ គឺតែ ទឹក និងសារធាតុផ្អែម (ស្ករ) សិប្បនិម្មិត ដែលគ្មានកាបូអ៊ីដ្រាត ។
រូបភាពទី ២២ ដល់ទី ២៨ បង្ហាញពីឧទាហរណ៍ជាក់លាក់នៃអាហារដែលជាអាហារសំខាន់ ទម្ងន់របស់វា និងបរិមាណកាបូអ៊ីដ្រាត ។ អង្ករ ២០០ ក្រាមនៅក្នុងរូបភាពទី ២២ ដែលបង្ហាញពី ១០ ក្រាមនៃកាបូអ៊ីដ្រាតជា ១ កាបូ មានផ្ទុកកាបូអ៊ីដ្រាត ៧៤ ក្រាម ដែលមានន័យថា វាមានប្រហែល ៧,៥ កាបូ ។ រូបភាពទី ២៣ បង្ហាញថា អង្ករមួយពែងមានផ្ទុកកាបូអ៊ីដ្រាត ៣៦,៤ ក្រាម ដែលមានប្រហែល ៣,៥ កាបូ ។ រូបភាពទី ២៤ គឺអាហារ ៥,៥ កាបូ រូបភាពទី ២៥ គឺអាហារ ៦ កាបូ រូបភាពទី ២៦ គឺអាហារ ៧ កាបូ និងរូបភាពទី ២៧ គឺអាហារ ៣,០ កាបូ និង ១,៥ កាបូ ។ ជំឡូងនៅក្នុងរូបភាពទី ២៦ នឹងជាអាហារ ២,៥ កាបូ ។



រូបទី១

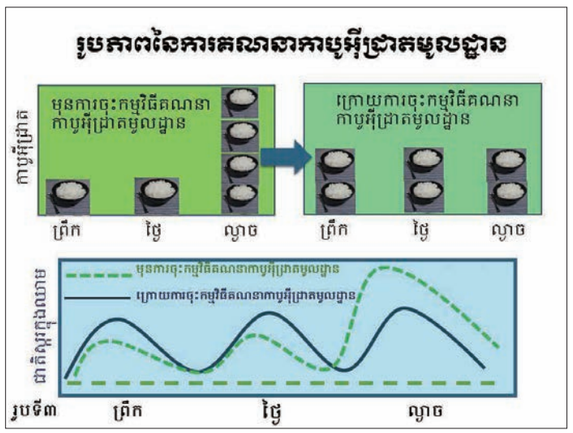
រូបទី៤


ឥទ្ធិពលនៃការក្រេនឆ្អើជាតស្ករក្នុងឈាមដោយសារកាបូអ៊ីដ្រាត ប្រូតេអ៊ីន និងខ្លាញ់

ក្រុមអាហារ	សារធាតុចិញ្ចឹម	ឥទ្ធិពលនៃការកើនឡើងជាតិស្ករក្នុងឈាម	ឥទ្ធិពលនៃការកន្ត្រើឡើងជាតិស្ករក្នុងឈាម
អាហារសំខាន់ប្រចាំថ្ងៃ (បាយ នំបុ័ង មី)	កាបូអ៊ីដ្រាត ប្រូតេអ៊ីន (តិច)	ខ្ពស់ខ្លាំង	រហ័ស
ផ្លែឈើ	កាបូអ៊ីដ្រាត	ខ្ពស់	រហ័ស
ទឹកដោះគោ	កាបូអ៊ីដ្រាត ប្រូតេអ៊ីន (តិច) ខ្លាញ់ (តិច)	ខ្ពស់	រហ័ស
បន្លែ	កាបូអ៊ីដ្រាត ប្រូតេអ៊ីន (តិច)	តិច	រហ័ស
សាច់	ប្រូតេអ៊ីន ខ្លាញ់ (តិច)	ទេ	ទេ
ខ្លាញ់	ខ្លាញ់	ទេ	ទេ

រូបទី២

រូបទី៥



រូបទី៣

រូបទី៦

រូបទី៧

រូបទី១០

រូបទី១៣

រូបទី១៦

រូបទី៨

រូបទី១១

រូបទី១៤

រូបទី១៧

រូបទី៨

រូបទី១២

រូបទី១៥

រូបទី១៨

Review article

រូបទី១៩

រូបទី២២

រូបទី២៥

រូបទី២៨

រូបទី២០

រូបទី២៣

រូបទី២៦

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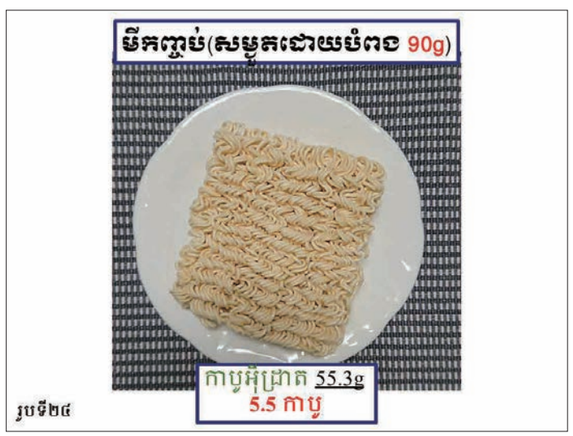
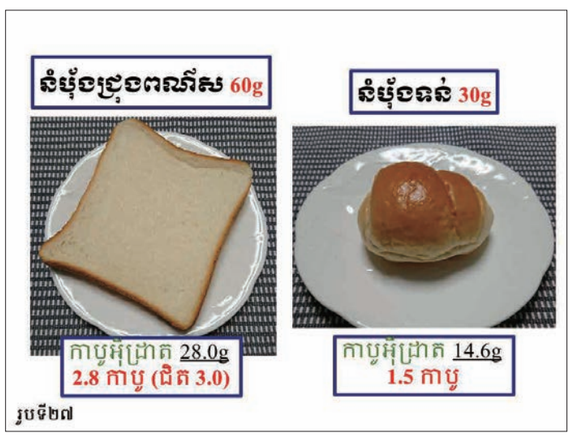


រូបទី២៤



រូបទី២៧

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២: Kitajima Y,Mikami N, Hyodo T, Kawakami J. Carbohydrate counting: a simple method of dietary management forglycemic control in Japanese diabetic hemodialysis patients. Contribution Nephrology.២០១៦;១៨៩

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១. ទស្សនាវដ្តី The Cambodian Journal of Nephrology ត្រូវបានបោះពុម្ពផ្សាយជាភាសាខ្មែរ អង់គ្លេស និង ជប៉ុនដោយក្រុមហ៊ុន Reiseikai Media (Cambodia) Co.,Ltd. ក្នុងគោលបំណងរួមចំណែកក្នុង វិស័យវេជ្ជសាស្ត្រ ក៏ដូចជាការអភិវឌ្ឍធនធានមនុស្សតាមរយៈការណែនាំពីមូលដ្ឋានគ្រិះនៃជំងឺតម្រងនោម និង សមិទ្ធិផលវេជ្ជសាស្ត្រក្នុងវិស័យសុខាភិបាលអោយបានទូលំទូលាយផងដែរ ។

២. អ្នកនិពន្ធ ត្រូវយល់ច្បាស់ និង ទទួលស្គាល់បទប្បញ្ញត្តិនៃការចុះផ្សាយនេះ ។ សូមចំណាំផងដែរថាបទប្បញ្ញត្តិនេះ អាចនឹងត្រូវបានធ្វើវិសោធនកម្មតាមការសំរេចចិត្តរបស់យើងខ្ញុំ ។

៣. រាល់សារណាទាំងអស់ ដែលបានចុះផ្សាយជាមួយយើងខ្ញុំត្រូវបានរក្សាសិទ្ធដោយ អ្នកនិពន្ធ ។ ក៏ប៉ុន្តែ ក្រុមហ៊ុនយើងខ្ញុំនឹងទទួលបាននូវសិទ្ធធ្វើការថតចម្លង ចែកចាយ ផ្សើរជាសាធារណៈ បកប្រែ និងកែសម្រួលផ្សេងៗ បានទូទាំងពិភពលោក ក្នុងកំឡុងពេលដែលបានព្រមព្រៀងមួយ ។ យោងទៅតាមបទបញ្ជាតិ្ត ក្នុងករណីដែល អ្នកនិពន្ធ ចង់ផ្តល់សិទ្ធនិពន្ធអោយអ្នកដទៃ ត្រូវមកសុំការអនុញ្ញាតិពីក្រុមហ៊ុនយើងជាខ្ញុំមុនសិន ។

៤. សារណាដែលត្រូវបានបកប្រែ និង កែសម្រួលដោយក្រុមហ៊ុនយើងខ្ញុំនឹងត្រូវបានរក្សាសិទ្ធគ្រប់យ៉ាងដោយក្រុមហ៊ុនយើងខ្ញុំ ។ មាត្រាទី២៨ស្តីពីច្បាប់រក្សាសិទ្ធរបស់ប្រទេសជប៉ុន និង ច្បាប់រក្សាសិទ្ធរបស់ប្រទេសផ្សេងៗ មិនត្រូវបានអនុវត្តិឡើយ បើសិនជាគ្មានការយល់ព្រមពីក្រុមហ៊ុនយើងខ្ញុំតាមរយៈលាយលក្ខណ៍អក្សរជាមុននោះទេ គឺមិនអនុញ្ញាតិអោយ អ្នកនិពន្ធ យកសារណាដែលបានកែសម្រួលរួចនៅក្រុមហ៊ុនយើងខ្ញុំយកទៅបោះពុម្ពនៅទស្សនាវដ្តីដទៃនោះទេ ។

៥. អ្នកនិពន្ធ មិនអាចជំទាស់ទៅនឹងការបកប្រែ ឬការកែសម្រួលនៃសារណាដែលក្រុមហ៊ុនយើងបានធ្វើឡើយ ។

៦. អ្នកនិពន្ធត្រូវធានាថា សារណាដែលបានសរសេរ មិនស្ថិតនៅក្រោមការរក្សារសិទ្ធរបស់អ្នកដទៃ ក៏ដូចជាប៉ះពាល់ដល់កិត្តិយស សិទ្ធិផ្ទាល់ខ្លួន ឯកជនភាព និងសិទ្ធិផ្សេងទៀតរបស់អ្នកដទៃឡើយ ។ ក្រុមអ្នកស្រាវជ្រាវផ្នែកវេជ្ជសាស្ត្រត្រូវតែធ្វើតាមសេចក្តីប្រកាសរបស់ទីក្រុង Helsinki និងពិចារណាវិជ្ជាជីវៈប្រកបដោយក្រមសីលធម៌ ។ ការពិសោធន៍ទៅលើសត្វត្រូវធ្វើឡើងនៅក្នុងបែបបទមួយដែលផ្តល់ការយកចិត្តទុកដាក់ទៅលើការការពារសត្វ ។ ក្នុងករណីអត្ថបទដែលបានសរសេរមានជាប់ពាក់ព័ន្ធនឹងអត្ថប្រយោជន៍របស់បុគ្គល ឬ អង្គការ ឬ ក្រុមហ៊ុននោះ អ្នកនិពន្ធត្រូវសរសេរបញ្ជាក់អំពីអត្ថប្រយោជន៍ ឬ ផលប៉ះពាល់ដែលអាចកើតមានទាំងនោះ ទៅក្នុងអត្ថបទដែលបានសរសេរផងដែរ ។

៧. អ្នកនិពន្ធ អាចយកសារណាដែលបានប្រកាសរួចនាឯសិក្ខាសិលាឬទស្សនាវដ្តីផ្សេងៗមកដាក់បាន ។ ក៏ប៉ុន្តែក្នុងករណីចំនុចមួយចំនួនដែលបានសរសេរនៅក្នុងសារណាដែលបានដាក់ជូន មានអត្ថន័យដូចគ្នាខ្លាំងទៅនឹងសារណាដែលបានចេញផ្សាយរួចរាល់នៅក្នុងទស្សនាវដ្តីដទៃនោះ អ្នកនិពន្ធ ត្រូវពិនិត្យអោយច្បាស់ដោយផ្ទាល់ជាមួយនឹងបទបញ្ជាក្តីនៃការចុះផ្សាយក៏ដូចជាបទបញ្ជានៃការចូលរួមសិក្ខាសិលា ឬត្រូវធ្វើការទំនាក់ទំនងជាមុនទៅកាន់ម្ចាស់ដើមនៃសារណាក្នុងករណីចាំបាច់ដើម្បីធ្វើការសុំអនុញ្ញាតិសម្រង់អត្ថបទ ក្នុងគោលបំណងជៀសវាងការរំលោភបំពានលើក្រមសីលធម៌នៃការស្រាវជ្រាវ ។ លើសពីនេះ អ្នកនិពន្ធត្រូវសរសេរអោយបានច្បាស់ពីប្រភពដែលបានដកស្រង់ដូចជាឈ្មោះស្ទៀវភៅ ឈ្មោះអ្នកនិពន្ធ ថ្ងៃចេញផ្សាយ និងទំព័រជាដើមផងដែរ ។

៨. ទស្សនាវដ្តីរបស់យើងខ្ញុំ មិនធ្វើការហាមប្រាម អ្នកនិពន្ធ ពីការយកសារណាទៅចេញផ្សាយនៅទស្សនាវដ្តីដទៃឡើយ ។ ក៏ប៉ុន្តែ យើងខ្ញុំសូមជម្រាបផងដែរថា សារណាដែលដាក់ជូនទស្សនាវដ្តីយើងខ្ញុំហើយ អាចនឹងមិនអាចចុះផ្សាយម្តងទៀតនៅក្រុមហ៊ុនទស្សនាវដ្តីនោះទេ ។ ម្យ៉ាងវិញទៀត យើងក៏សូមបញ្ជាក់ផងដែរថា ក្នុងករណីអ្នកនិពន្ធយកសារណាមកដាក់ជូនយើងខ្ញុំ ធ្វើអោយសារណាដដែលនោះមិនអាចយកទៅផ្សាយក្នុងទស្សនាវដ្តីដទៃ យើងខ្ញុំមិនទទួលខុសត្រូវឡើយ ។

៩. ការជ្រើសរើសសារណា ធ្វើឡើងដោយការវាយតម្លៃពីគណៈកម្មាធិការរៀបចំការបោះពុម្ពផ្សាយ ដោយមានការពិនិត្យឡើងវិញជាមុន ។

១០. សារណាដែលបានដាក់ជូនហើយមិនអាចដកវិញបាននោះទេ ។

១១. ការកែតម្រូវរបស់អ្នកនិពន្ធអាចធ្វើឡើងបានតែម្តងប៉ុណ្ណោះតាមកាលបរិច្ឆេទកំណត់ដែលបានស្នើ ។ អ្នកនិពន្ធ អាចកែតម្រូវបានក្នុងករណីបាត់ពាក្យ ឬ ខុសអក្ខារិរុទ្ធប៉ុណ្ណោះ ។ មិនត្រូវសរសេរបន្ថែមនោះទេ ។

១២. ១) សារណាត្រូវសរសេរដោយប្រើកុំព្យូទ័រ ។
២) ត្រូវប្រើខ្នាត A៤ បញ្ឈរ និង មានគំលាតសមស្រប ។

១៣. ទាំងអ្នកនិពន្ធ និង ក្រុមហ៊ុននឹងមិនត្រូវបានបង់ប្រាក់សម្រាប់ការបោះពុម្ពផ្សាយនៅក្នុងទស្សនាវដ្តីនោះទេ ។

១៤. សូមយកសារណារបស់អ្នកមកដាក់ជូនតាមរយៈអាស័យដ្ឋានខាងក្រោម :

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