World Journal of Gastroenterology

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DOI: 10.3748/wjg.v20.i30.10525

World J Gastroenterol 2014 August 14; 20(30): 10525-10530
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

RETROSPECTIVE STUDY

Refeeding syndrome in Southeastern Taiwan: Our experience with 11 cases

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Received: December 30, 2013 Revised: March 13, 2014
Accepted: May 1, 2014
Published online: August 14, 2014

Abstract

AIM: To present our experience with refeeding syndrome in southeastern Taiwan.

METHODS: We conducted a retrospective study during a 2-year period at the Mackay Memorial Hospital, Taitung Branch. We enrolled patients with very little or no nutrition intake for more than 10 d, a high risk group of refeeding syndrome, including those suffering from alcohol abuse, cancerous cachexia, chronic malnutrition, and prolonged starvation.

RESULTS: A total of 11 patients (7 males, 4 females) with nasogastric feeding were included as having refeeding syndrome. Most of them had the symptoms of diarrhea, lethargy, and leg edema. The initial nutritional supplement was found to be relatively high in calories (1355.1 ± 296.2 kcal/d), high in protein (47.3 ± 10.4 gm/d), low in vitamin B1 (2.0 ± 0.5 mg/d), low in potassium (1260.4 ± 297.7 mg/d), and low in phosphorus (660.1 ± 151.8 mg/d). Furthermore, hypophosphatemia (2.4 ± 0.9 mg/dL) was noted during follow-up. Based on the suggestions of a dietician and a gastroenterologist, the clinical disorders of diarrhea, malaise and leg edema were significantly improved. The level of phosphate was also increased (3.3 ± 0.6 mg/dL).

CONCLUSION: Refeeding syndrome is an overlooked and risky disorder that has some potentially fatal complications. Nasogastric feeding in nursing homes is an important risk factor for patients and deserves greater attention based on the initial results of this study.

Key words: Refeeding syndrome; Nutrition status; Cachexia; Hypophosphatemia; Risk assessment

Chen LJ, Chen HL, Bair MJ, Wu CH, Lin IT, Lee YK, Chu CH. Refeeding syndrome in Southeastern Taiwan: Our experience with 11 cases. *World J Gastroenterol* 2014; 20(30): 10525-10530 Available from: URL: http://www.wjgnet.com/1007-9327/full/v20/i30/10525.htm DOI: http://dx.doi.org/10.3748/wjg.v20.i30.10525

INTRODUCTION

Refeeding syndrome (RFS) is not an unusual clinical disorder found when treating poorly nourished patients.

Table 1 Criteria for determining people at high risk of developing refeeding syndrome (according to National Institute for Health and Clinical Excellence guidelines)

The patient has one or more of the following:
Body mass index less than 16 kg/m^2
Unintentional weight loss greater than 15% within the last 3-6 mo
Little or no nutritional intake for more than 10 d
Low levels of potassium, phosphate or magnesium prior to feeding
Or the patient has two or more of the following:
Body mass index less than 18.5 kg/m^2
Unintentional weight loss greater than 10% within the last 3-6 mo
Little or no nutritional intake for more than 5 d
A history of alcohol abuse or drugs including insulin, chemotherapy, antacids or diuretics

This topic is generally well-depicted but often overlooked with regards to detailed research. The morbid symptoms and signs were first observed and reported from starved ex-prisoners from World War Ⅱ after their initial reintroduction of intake[1,2]. The definite phenomenon of RFS have had an advanced understanding since the 1940s, which has occurred in malnourished patients undergoing refeeding not only orally and enterally, but also parenterally[3]. The occurrence of clinical fluid and electrolyte imbalance results from pathogenicity of hormonal and metabolic derangements. All of associated complications may lead to serious clinical catastrophes, such as neuromuscular and cardiopulmonary compromise[4-6]. In addition, RFS is very complex in that it may also be characterized by changes in fat, glucose as well as protein metabolism. Furthermore, thiamine deficiency, hypokalemia, and hypomagnesaemia also occur in such patients[3,7]. The National Institute for Health and Clinical Excellence (NICE) guidelines[8] were published in 2006, which highlighted the risk in certain specific disease populations (Table 1). However, RFS still occurs in the clinical setting today because clinicians are often not attentive enough to the potential risk of this disorder.

Taitung County in southeastern Taiwan contains the highest proportion of aboriginal residents[9], and a higher prevalence of alcoholism among aborigines has been previously reported (44.2%-55.2%) in the 1990s[10]. In addition, we noted that Taitung County also had a higher elderly disability prevalence (24.2%) from public health research of the geographic disparity during the years 2000-2010 in Taiwan[11]. Furthermore, poor health knowledge, limited access to healthcare, relatively low socioeconomic class, and inadequate treatment of organic diseases, such as chronic liver disease, cardiopulmonary failure and diabetes, were more common in contrast to western Taiwan. With the above factors, the clinical presentations of RFS may be often found in the clinical nutrition care setting of Taitung, and clinicians should be alerted to this concern. We designed a retrospective study to gather data including the following: basic data, clinical features, laboratory data, and outcomes after nutritional therapy within a 2-year period at Mackay Memorial Hospital, Taitung Branch, a major hospital in southeastern Taiwan.

MATERIALS AND METHODS

All of patient's information was obtained from the medical records of the Mackay Memorial Hospital, Taitung Branch between August 2000 and August 2012. The date of the initial consultation and establishment of initial feed was assembled for each patient as the initial route of feeding. The first mode of feeding sustaining for more than 24 h was recorded. Each patient's risk of developing RFS before feeding was assessed by analysis of the patient's records. We used the malnutrition universal screening tool, which is a five-step screening tool to identify adults who are malnourished or at risk of malnutrition (undernourished). A malnutrition screening assessment was performed if clinicians or nursing members were alerted.

We enrolled the patients within a high risk group, such as those with little or no nutrition intake for more than 10 d, alcohol abuse, cachexia, malnutrition [> 10% weight loss within 2 to 6 mo or body mass index (BMI) $\leqslant$ 17 kg/m^2], and prolonged starvation according to the NICE guidelines[5]. The risk was independently validated by a dietitian after having been assessed by a gastroenterologist. All the patients were adults (> 18 years old), and patients with impaired renal function or pregnancy were excluded. In addition, a low serum electrolyte level prior to feeding was not included as a criterion. The primary endpoint was a decline of serum phosphate in patients who had a normal status of phosphate prior to feeding.

We collected demographics and clinical features associated with the patients that were diagnosed with RFS. The data included initial nutrition support, such as calories, protein, and electrolytes, and the results of laboratory tests were also collected. Nutritional status was assessed by diet history, weight loss and BMI, as well as albumin, cholesterol and lymphocyte values in the plasma. Patients were strictly monitored and analyzed from the start of receiving nutritional supplements in order to detect the correct electrolytic and metabolic disturbances associated with RFS. For each patient, a pathological database was examined to establish whether the phosphate dropped from a normal level before feeding within a week after the establishment of the initial feed.

After nutrition adjustment was performed, we compared the differences in clinical features and laboratory data. The data were inputted into a spreadsheet (PASW Statistics 18). Data are expressed as frequency for nominal variables, and as mean ± SD for continuous variables. Paired-sample *t*-test was used for quantitative variables. A P-value < 0.05 was considered statistically significant.

RESULTS

Of all patients (n = 56) that were at risk of developing RFS after analyzing the clinical variables, most of them (46/56, 82.1%) were fed *via* the nasogastric (NG) tube route. Five patients received total parenteral nutrition, and another five patients received oral feeding by their respective caregiver. A total of 11 patients (19.6%; 7 males, 4 females) were diagnosed as having RFS. All of

Table 2 Demographic and nutritional characteristics of patients with RFS at the beginning of nutrition support

	Mean ± SD
Number	11
Mean admission days	19.1 ± 8.5
Post-NG feeding days with nutrition consultation	5.0 ± 3.0
Sex (M/F)	7/4
Age (yr)	67.9 ± 19.3
Mean height (cm)	157.8 ± 10.1
Body mass index (kg/m^2)	16.7 ± 3.8
Ideal weight (kg)	55.7 ± 7.7
BEE (kcal)	1012.7 ± 222.2
Leg edema (yes/no)	9/2
Diarrhea (yes/no)	7/4
Abdominal distension (yes/no)	4/7

NG: Nasogastric.

Table 3 Diet differences at initial presentation and after consultation

	Initial status	After nutritional consultation
Calorie (kcal)	1355.1 ± 296.2	907.2 ± 117.9[a]
Calorie/kg (kcal)	33.8 ± 9.5	22.7 ± 3.9[a]
Protein (g)	47.3 ± 10.4	49 ± 8
Thiamine (mg)	2.0 ± 0.5	8.5 ± 6.2[a]
K (mg)	1260.4 ± 297.7	1615.9 ± 265[a]
P (mg)	660 ± 151.8	862.1 ± 105.5[a]
Ca (mg)	672.3 ± 147.3	908.4 ± 199.1[a]
Mg (mg)	262 ± 62.2	269.6 ± 25.9

[a]$P < 0.05$ *vs* control (paired-sample *t*-test).

Table 4 Biochemical differences at initial presentation and after nutritional adjustment

	Initial	Nutrition consult
K (mmol/L)	3.6 ± 0.5	4.4 ± 0.8[a]
P (MG/DL)	2.4 ± 0.9	3.3 ± 0.6[a]
Ca (MG/DL)	7. 9 ± 0.7	8.1 ± 0.8
Mg (MG/DL)	1.9 ± 0.4	2.0 ± 0.3
Na (mmol/L)	135.8 ± 5.6	134.6 ± 5.7
Glucose (MG/DL)	122 ± 47	117 ± 38

[a]$P < 0.05$ *vs* control (paired-sample *t*-test).

them were fed *via* the NG tube route, and their mean age was 67.9 ± 19.3 years. Most of them (n = 10, 90.9%) had chronic diseases, such as heart failure, diabetes, old stroke, chronic obstructive pulmonary disease, and senile dementia. In addition, most of them lived in a nursing home, with the exception of one young adult (39 year-old) who lived at home and had alcohol abuse with cachexia. Based on our primary analysis, all of our patients were observed to be in a malnourished state (mean albumin, 2.7 ± 0.7 g/dL) and had a lower BMI (16.7 ± 3.8 kg/m^2) (Table 2).

The major symptoms were leg edema (9/11, 81.8%), diarrhea (7/11, 63.64%), and abdominal distension (4/11, 36.36%), as well as poor ventilator weaning. At the beginning of their nutritional therapy (Table 3), the patients underwent a formula that involved high calories (1355.1 ± 296.2 kcal/d), low potassium (1260.4 ± 297.7 mg/d), low thiamine (2.0 ± 0.5 mg/d), low calcium (672.3 ± 147.3 mg/d), and low phosphorus (660.1 ± 151.8 mg/d). When a poor response to medical treatment for metabolic derangement was observed, nutrition consultations were requested several days after admission (5.0 ± 3.0 d).

After dietician and gastroenterologist consultations were provided, the diagnosis of RFS was considered to be the most likely. The total calorie intake regarding nutrition was predominantly decreased (907.2 ± 117.9 kcal/d). In addition, the supplements of thiamine (8.5 ± 6.2 mg/d), potassium (1615.9 ± 265 mg/d), calcium (908.4 ± 199.1 mg/d) and phosphorus (862.1 ± 105.5 mg/d) were increased (Table 3). The clinical condition of diarrhea and leg edema was significantly improved after two weeks. The levels of potassium and phosphate were found to have a major increase, but the levels of sodium, calcium, magnesium, and glucose were generally not changed (Table 4). Although improvement in the clinical symptoms of RFS was noted, three patients (27.27%) died of sepsis and respiratory failure.

DISCUSSION

RFS is a well-known but often ignored disorder. It has been observed since the early 1940s, as severe metabolic acidosis, encephalopathy, rhabdomyolysis, fetal arrhythmia, and respiratory failure were observed after intensive nutrition refeeding *via* an enteral or parenteral route in malnourished patients. Edema and sudden death caused by an electrolyte imbalance and cardiopulmonary failure were most often observed in such patients[3,12]. The edema coming from the extracellular fluid overload is presumed to develop due to impaired function of the sodium/potassium pump in the cell membrane. For the malnourished patients after reintroduction of feeding, these ion exchange pumps gradually return to active function. They expel sodium from the cells, which causes a hyperosmolar extracellular status that saves water and results in edema. From our current clinical knowledge, sudden death was strongly related to severe hypophosphatemia and hypomagnesemia which were the best biochemical hallmark of RFS. After the carbohydrate-rich food intake in patients involved in long-term fasting, insulin secretion is seen to have rigorous activation, which increases the cellular inward transportation of glucose, water, potassium, magnesium and phosphate. All of them result in a rapid decline of serum glucose, potassium and phosphate. Based on our related knowledge, intracellular phosphate and potassium play a crucial role in many cellular pathways including the Krebs cycle, glycolysis, and protein kinases. However, pathophysiologic hypokalemia and hypophosphatemia result in muscle weakness (such as respiratory and cardiac decompensation). Furthermore, increased glucose metabolism usually leads to thiamine deficiency. Clinicians must also consider cardiomyopathy

and Wernicke syndrome.

This retrospective study disclosed the current status on refeeding in southeastern Taiwan, where a higher prevalence of protein-calorie malnutrition was noted due to alcoholism and elderly disability. A total of 56 patients had high risk factors, but only 11 patients were found to have RFS. In our opinion, this number may have been underestimated due to incomplete data collection and insufficient knowledge with regards to RFS. In our hospital, we could not perform a good initial nutrition assessment for each patient due to a shortage in manpower. Most enrolled patients were alerted by their clinician or nursing members, but this was usually done several days (5.0 ± 3.0 d) after admission. Furthermore, based on the NICE criteria for RFS[9], we only recognized the high risk groups, but there were no definitive criteria for diagnosis or confirmation in a real world setting. The characteristic signs of RFS were pathogenic hypophosphatemia and its associated complications[13]. Brooks *et al*[5] found that of 321 RFS patients, only about a quarter were at high risk according to NICE criteria to develop hypophosphatemia (n = 23, 7%). On the contrary, the majority of them did not develop hypophosphatemia (n = 69, 21%). Because of non-specific presentations and indefinite diagnosing criteria, RFS becomes a difficult challenge for clinicians to overcome. As such, the global reported incidence is prone to be underestimated in clinical practice[14]. As a result, we need to establish another guideline, which involves definitive criteria that are more sensitive and specific. In addition, clinicians must be educated to recognize this syndrome.

In our study group, all of our patients adopted NG feeding and lived in nursing homes, which means that there was poor nutrition adjustment in these nursing homes. In a study of 111 residents from four nursing homes in Kaohsiung of Taiwan[15], it was found that as many as 41.1% of patients had a low BMI (< 18.5 kg/m^2) and 18.9% had a malnourished status (serum albumin < 3.0 g/dL). The route of feeding makes a nutritional difference in clinical observation. The residents on NG feedings compared to residents on oral feedings had an approximately four times higher risk of a low BMI and eight times the risk of a low albumin level. Taitung County is noted to have a higher prevalence of elderly disabled residents, and the most of them are patients receiving long term care in nursing homes. In addition, more of them had chronic diseases, and NG feeding was a common route of nutrition. If patients have NG feeding and are based in a nursing home, clinicians should be alert to the potential risk of RFS.

Alcoholic patients often consume alcohol, which contains little phosphate, and they have low body phosphate stores and are therefore at risk after a sudden increase in carbohydrate intake[16]. In addition, hyperphosphaturia may occur in cases of alcoholism, and thiamine required for the intracellular transport of glucose may be depleted[17]. Even though chronic alcohol abuse is a risk factor for RFS, we had only one patient who was known to have alcoholism in this study. In our opinion, this may have also been underestimated because most alcoholic patients had liver cirrhosis and chronic pancreatitis, which could mask the underlying status of malnutrition. When RFS does occur, this alcoholic patient with cachexia was fed an NG diet during admission. Although the clinical features of diarrhea and edema were improved by nutrition adjustment, the patient ultimately died of sepsis.

The nutritional consultation was always requested in the real world after several days of admission (5.0 ± 3.0 d), which meant that RFS happened earlier. In a prospective cohort study of patients in intensive care units reported in 1996, a total of 34% of patients developed hypophosphatemia approximately two days after feeding (1.9 ± 1.1 d)[18]. Therefore, it is necessary to identify groups that are at risk as soon as possible when initiating nutrition supplement. In our study group, nutrition adjustment was lower daily calories and increased thiamine, potassium, and phosphorus. The clinical manifestations of RFS were significantly improved in all patients.

There are several proposed strategies for the initial reintroduction of feedings. A cautious recommendation[19,20] suggests that the suitable amount of calories is 20 kcal/kg per day or 1000 kcal/d. However, 15 kcal/kg per day is appropriate in severely malnourished patients. Initial feeding is at 25% of goal calories on day 1 and gradually increased in the following days as clinical need is also increased. Goal rate can be accomplished within 5 to 7 d. In addition, it is very important to supply thiamine and multivitamins when starting nutritional therapy in poorly nourished and starved patients. Those malnourished patients at risk of RFS should be provided with thiamine at doses of 100 mg/d orally or 50 to 100 mg/d intravenously[19,21]. With regards to high risk groups of RFS, not only the clinical manifestations associated with derangement of electrolytes should be closely monitored, but also fluid balance and daily weight must be under careful surveillance[19].

Early suspicion and proactive prevention are the key to successful management of RFS[22]. This arduous task can be achieved by taking a detailed history and thorough clinical examination. Clinicians should recognize the needs for early administration of the nutrition support team in high-risk patients[23,24]. Therefore, physicians must keep RFS in mind and be alert to this serious disorder at an early stage. A well-definite formal nutritional assessment is suitable for those identified as being either malnourished or at high risk of RFS. With a good nutritional assessment of the patients, well-experienced nutrition nurses and qualified dieticians can create an individualized formulation according to NICE strategies for each patient, which may avoid RFS from occurring. Moreover, clinicians should keep alert to the risk groups in developing RFS as early as possible. Therefore, effective communication between clinicians and nutrition teams is vital to achieving the best care possible for such patients. The successful prevention and management of RFS require a multidisciplinary approach and teamwork, involving nutritionists, nurses, and doctors, which regu-

larly discuss the changing nutritional needs of patients.

In conclusion, generally speaking, RFS is an underestimated disorder which has significantly fatal complications in malnourished patients in any way of artificial nutrition. NICE guidelines help the clinician to stratify a patient's risk of developing RFS, but NG feeding in nursing homes highlights a risk factor rarely considered in our study. Derangement of fluid, electrolytes, and glycemic status were the major manifestations of RFS. Fetal complications mostly resulting from hypophosphatemia and cardiopulmonary failure were observed. The early recognition of at-risk patients and the careful monitoring of symptoms and signs during refeeding are highly recommended. However, to date, there have been no specific presentations and well-known diagnosis criteria for RFS. We must establish another set of guidelines, which include more definitive criteria that are more sensitive and specific. Before this is established, the only approach for clinicians is to (1) identify the at-risk population; (2) monitor the clinical features of patients during refeeding; and (3) consult nutritional support teams as early as possible. Moreover, clinical physicians, nursing assistants, pharmacists, and nutritionists must be well-educated regarding this topic and also understand more about the pathophysiology and the complications associated with RFS.

COMMENTS

Background

Refeeding syndrome is a well-described, but often overlooked disorder. It has been recognized as a concern since the early 1940s, as severe serum fluid and electrolyte derangements were observed after refeeding was conducted in malnourished patients.

Innovations and breakthroughs

The authors designed a retrospective study to gather data as follows: basic data, clinical features, laboratory data, and outcomes after nutritional therapy within a 2-year period at Mackay Memorial Hospital, Taitung Branch, a major hospital in southeastern Taiwan.

Applications

Clinical physicians, nursing assistants, pharmacists, and nutritionists must be well-educated regarding this topic and develop an understanding about the pathophysiology of refeeding syndrome (RFS) and the complications associated with RFS.

Peer review

This manuscript discusses an important topic as we are currently in an era with a high number of elderly malnourished patients who are receiving their nutritional support orally, enterally or parenterally in nursing homes. The authors describe the related symptoms and treatments in 11 patients with suspected RFS. As the authors describe the importance of insulin in developing RFS, it is crucial to better understand the serum glucose level of patients with RFS upon admission, during initial feeding (during the development of RFS), and after receiving nutritional consultation. Insulin increases Na-K-ATPase pump activity which, independently of its effect on glucose transport, causes K+ in and Na+ to be carried out of the cells at a 2:3 ratio, leading to a K+ shift into muscle and liver cells. As a result, it is crucial to take care of this not only at the cellular level of phosphate, but also with regards to potassium which is reflected by the QT interval on the electrocardiogram, and/or the onset of arrhythmias.

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P- Reviewer: Kawai C **S- Editor**: Qi Y
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ISSN 1007-9327