Review Article

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# Refeeding Syndrome: Pathophysiology, Clinical Management, and Implications for Disaster Preparedness

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## Abstract:

Refeeding syndrome (RFS) is a potentially fatal, multisystem disorder triggered by the reintroduction of nutrition (whether oral, enteral, or parenteral) after prolonged starvation or significant undernutrition. Its hallmark is hypophosphatemia, accompanied by hypokalemia, hypomagnesemia, thiamine deficiency, and disordered fluid balance, which together may precipitate cardiac arrhythmia, heart failure, respiratory failure, encephalopathy, rhabdomyolysis, and sudden death. Despite growing recognition, RFS remains underdiagnosed owing to inconsistent definitions and subtle early manifestations. The 2020 American Society for Parenteral and Enteral Nutrition (ASPEN) consensus introduced standardized diagnostic criteria, while the National Institute for Health and Care Excellence (NICE) criteria continue to guide risk stratification. Those at the greatest risk include patients with anorexia nervosa, advanced malignancy, chronic alcohol use disorder, prolonged fasting, prior bariatric surgery, or extended intensive care unit (ICU) admission. Prevention centres on the early identification of at-risk individuals, prophylactic thiamine, correction of electrolyte deficits, and gradual caloric advancement under close biochemical monitoring. Because this review forms part of a special collection on the November 2025 Hat Yai flooding, where prolonged involuntary fasting, disrupted chronic disease care, and abrupt refeeding with carbohydrate-dominant relief food converged across a population of over 3.5 million, a dedicated section addresses RFS recognition, prevention, and management in flood- and disaster-affected populations. Together, these topics reflect the full clinical and public-health spectrum of a preventable but potentially fatal condition.

**Keywords:** disaster, flood, hypophosphatemia, malnutrition, refeeding syndrome, thiamine

This paper is part of the "Hat Yai Flooding 2025" collection
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## Introduction

Refeeding syndrome (RFS) describes the constellation of metabolic, electrolyte, and clinical disturbances that arise when nutrition is reintroduced to a malnourished or starved individual. The phenomenon was systematically described after World War II in survivors of prolonged starvation, including liberated prisoners of war and concentration-camp inmates, who developed cardiac and neurologic deterioration and, in some cases, died within days of resuming a normal diet[1,2]. Burger and colleagues' Dutch famine reports and Schnitker's observations regarding American prisoners of war (POWs) in Asia together established the now-classic clinical picture: edema, congestive heart failure, weakness, and sudden death precipitated by carbohydrate-rich feeding[1-3].

In modern practice, RFS is encountered far beyond the famine context. Hospitalized patients with eating disorders, advanced cancer, alcohol-use disorder, decompensated heart failure, postoperative ileus, malabsorption, and prolonged critical illness all have a substrate of cellular depletion that can be unmasked by the reintroduction of energy[4,5]. Reported incidence varies widely, from 0% to over 80%, depending on the population studied and the definition used, reflecting both heterogeneity in case-mix and the absence of a single, validated criterion until recently[6-9]. The 2020 American Society for Parenteral and Enteral Nutrition (ASPEN) consensus definition has begun to standardize reporting and should facilitate clearer epidemiologic comparisons going forward[10].

Despite decades of clinical experience, RFS remains under-recognized and under-treated. Surveys of clinicians demonstrate substantial variability in screening practices, refeeding protocols, and electrolyte replacement strategies[11]. This review summarizes the contemporary understanding of RFS, integrates the major guideline frameworks, and translates recent randomized evidence into practical bedside recommendations.

The clinical relevance of RFS extends beyond hospital wards to disaster medicine. The catastrophic flooding of Hat Yai and the wider southern Thai provinces in November 2025 exposed millions to conditions predisposing them to RFS. Hat Yai received approximately 630 mm of rain over 72 hours; Hat Yai Hospital was inundated, and more than three million people across nine southern provinces were affected. Many residents were trapped above floodwaters for several days with little or no food; others lost access to dialysis, oncology, and chronic disease care. In evacuation shelters, vulnerable older adults and children received energy-dense but micronutrient-poor relief food, completing the triad of prolonged restricted intake, disrupted chronic disease management, and abrupt carbohydrate-dominant refeeding[12].

Parallels with other contemporary humanitarian crises, most notably the documented refeeding-syndrome risk emerging from famine in Gaza, underscore that any acute disruption of the food–health system can recapitulate the conditions in which RFS first came to medical attention[13]. Because the present article is part of the special collection on the 2025 Hat Yai flooding, we devote a dedicated section to the recognition, prevention, and management of RFS in disaster- and flood-affected populations.

### Definition and diagnostic criteria

RFS lacks a single universally accepted definition, but two frameworks dominate practice. The National Institute for Health and Care Excellence (NICE) 2006 guidance defines patients "at risk" by clinical criteria and frames the syndrome in terms of severe biochemical shifts and clinical sequelae after refeeding[14]. The 2020 ASPEN consensus defines RFS as a measurable decrease in any of phosphate, potassium, or magnesium (or thiamine deficiency) developing within five days of the reintroduction of calories, and stratifies severity (Table 1)[10]. The ASPEN definition makes biochemistry central and decouples

diagnosis from clinical syndrome, recognizing that overt cardiopulmonary collapse represents only the most severe end of the spectrum.

## Pathophysiology

### Adaptation to starvation

During fasting, glycogen stores are depleted within 24–72 hours. Insulin secretion falls, glucagon rises, and the body shifts to lipolysis and ketogenesis to spare protein. Cellular uptake of phosphate, potassium, and magnesium decreases, and intracellular stores are progressively depleted even when serum concentrations remain within reference ranges[15]. Total body deficits of phosphate, potassium, and magnesium occur after several weeks of inadequate intake, although serum levels typically remain normal because of buffering and renal compensation[16]. Thiamine, a water-soluble vitamin with limited body stores (~30 mg) and a half-life of ~11 hours, is similarly depleted[17].

### Carbohydrate reintroduction and the insulin surge

The reintroduction of carbohydrates produces a rapid rise in serum insulin. Insulin drives glucose, phosphate, potassium, and magnesium into cells; stimulates the anabolic synthesis of glycogen, fat, and protein (which itself consumes phosphate as adenosine triphosphate [ATP] and 2,3-diphosphoglycerate [2,3-DPG]); and promotes renal sodium and water retention[4]. The result is a precipitous fall in already-depleted serum phosphate, potassium, and magnesium, often within 24–72 hours of feeding. Increased thiamine demand for glucose oxidation in pyruvate dehydrogenase and α-ketoglutarate dehydrogenase pathways unmasks deficiency, predisposing to lactic acidosis, Wernicke encephalopathy, and wet beriberi[18,19]. The pathophysiological mechanisms of refeeding syndrome are summarized in Figure 1.

### Hypophosphatemia: the cardinal feature

Phosphate is required for ATP synthesis, 2,3-DPG-mediated oxygen delivery, intracellular signaling, and membrane integrity. Severe hypophosphatemia (<0.32 mmol/L) impairs myocardial contractility, diaphragmatic strength, and erythrocyte function, and may precipitate hemolysis, rhabdomyolysis, seizures, and ventricular arrhythmia[20]. Hypophosphatemia is the most consistent biochemical marker of RFS and is incorporated into virtually all definitions[10,14].

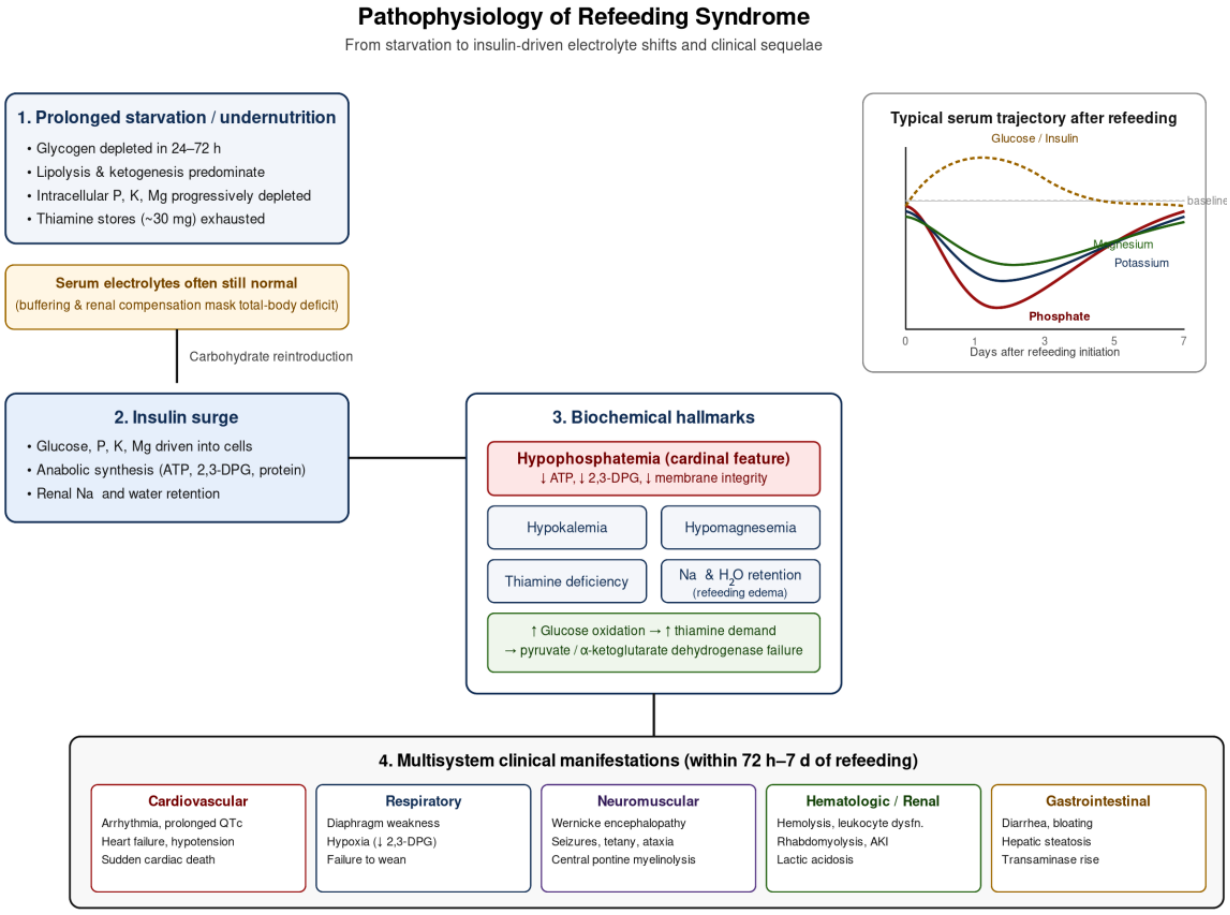
### Fluid and sodium handling

Carbohydrate feeding stimulates insulin-induced renal sodium reabsorption and water retention, producing extracellular volume expansion. In patients with reduced cardiac mass from prolonged starvation, even modest sodium loads can precipitate pulmonary edema and

**Table 1** ASPEN 2020 consensus diagnostic and severity criteria for refeeding syndrome (adapted from da Silva et al.)[10]

| Severity | Electrolyte decreases from baseline (any of P, K, or Mg) | Clinical features |
|---|---|---|
| Mild | 10–20% decrease | No organ dysfunction |
| Moderate | 20–30% decrease | No organ dysfunction |
| Severe | >30% decrease, or any electrolyte critically low, or organ dysfunction due to low electrolyte/thiamine deficiency | Organ dysfunction, life-threatening |

ASPEN=American Society for Parenteral and Enteral Nutrition, P=phosphate/phosphorus, K=potassium, Mg=magnesium




ATP=adenosine triphosphate, 2,3–DPG=2,3–diphosphoglycerate, AKI=acute kidney injury, P=phosphorus, K=potassium, Mg=magnesium, Na=sodium, $H_2O$=water, QTC=corrected QT interval

**Figure 1** Pathophysiology of refeeding syndrome. Prolonged starvation depletes intracellular phosphate, potassium, magnesium, and thiamine even when serum concentrations remain normal because of buffering and renal compensation. Reintroduction of carbohydrates triggers an insulin surge that drives glucose, P, K, and Mg into cells, stimulates anabolic synthesis (consuming additional ATP and 2,3–DPG), and promotes renal sodium and water retention. The result is a precipitous fall in already–depleted serum phosphate (the cardinal feature), potassium, and magnesium within 24–72 hours, with unmasking of thiamine deficiency. Multisystem clinical sequelae include cardiac arrhythmia and heart failure, respiratory failure, encephalopathy and seizures, hemolysis and rhabdomyolysis, and gastrointestinal dysfunction.

congestive heart failure, the so-called "refeeding edema"[21,22]. Glucose-driven anabolism also raises metabolic rate and oxygen demand, further stressing a deconditioned cardiopulmonary system.

### Risk stratification and At-Risk populations

Identifying at-risk patients before nutrition support is the single most important step in preventing RFS. The NICE criteria (Table 2) remain the most widely cited screening framework, although they have only modest predictive value for biochemical or clinical RFS[14,23]. The 2020 ASPEN consensus offers an updated set of risk criteria that integrate body mass index (BMI), recent weight loss, intake history, prior abnormal electrolytes, and high-risk clinical contexts (e.g., advanced cancer, prolonged ICU stay)[10].

### High-risk clinical populations

Several patient groups deserve heightened vigilance:

Anorexia nervosa and other restrictive eating disorders, particularly with BMI <14 kg/m$^2$ or rapid recent weight loss

Advanced malignancy, especially with anorexia–cachexia syndrome, head-and-neck cancer, or post-chemoradiation mucositis

Chronic alcohol-use disorder and decompensated liver disease (often with concurrent thiamine deficiency)

Prolonged critical illness with cumulative caloric debt and post-ICU acquired weakness

Postoperative patients with prolonged nil-per-os states, particularly after upper gastrointestinal or bariatric surgery

Frail older adults with dysphagia, depression, or social isolation

Patients with chronic malabsorption (inflammatory bowel disease, short bowel, cystic fibrosis)

Refugees, hunger strikers, and survivors of famine or prolonged caloric deprivation

Among hospitalized adults, retrospective cohorts report RFS incidence of 2–14%, while in selected high-risk groups (advanced anorexia nervosa, severe ICU malnutrition), the reported incidence approaches 30–50% when biochemical definitions are used[7-9,24].

### Clinical manifestations

RFS is a multisystem disorder. Manifestations typically appear within 72 hours but may occur up to 5–7 days after refeeding begins.

### Cardiovascular

Sinus tachycardia, ventricular ectopy, prolonged QTc, torsade de pointes, congestive heart failure, hypotension, and sudden cardiac death have all been reported[22].

**Table 2** NICE 2006 criteria identifying patients at "high risk" of refeeding syndrome[14]

| Major criteria (any 1) | Minor criteria (any 2) |
|---|---|
| BMI <16 kg/m$^2$ | BMI <18.5 kg/m$^2$ |
| Unintentional weight loss >15% in the past 3–6 months | Unintentional weight loss >10% in the past 3–6 months |
| Little or no nutritional intake for >10 days | Little or no nutritional intake for >5 days |
| Low pre-feeding serum potassium, phosphate, or magnesium | History of alcohol misuse, or drugs including insulin, chemotherapy, antacids, or diuretics |

NICE=National Institute for Health and Care Excellence, BMI=body-mass index

Pre-existing cardiac atrophy from prolonged starvation reduces stroke volume and ventricular reserve, magnifying the impact of fluid loading and electrolyte shifts.

### Respiratory

Hypophosphatemia impairs diaphragmatic contractility and 2,3-DPG–mediated oxygen unloading, predisposing to respiratory failure and difficulty weaning from mechanical ventilation[22].

### Neuromuscular and neurologic

Weakness, paresthesia, tetany, seizures, encephalopathy, ataxia, ophthalmoplegia (Wernicke), and central pontine myelinolysis (with rapid sodium correction) are all described[19,22].

### Hematologic and renal

Hemolysis, leukocyte dysfunction, thrombocytopenia, acute kidney injury secondary to rhabdomyolysis or hypotension, and metabolic acidosis from thiamine-related lactic accumulation can complicate refeeding[4,18].

### Gastrointestinal

Bloating, diarrhea, hepatic steatosis, and elevated transaminases reflect the metabolic stress of rapid carbohydrate loading on a hypofunctional gut and liver[16].

## Prevention

The mainstay of prevention is anticipating RFS in at-risk patients and modifying the refeeding strategy accordingly. A consolidated, stepwise approach is summarized in Table 3 and discussed below.

### Step 1–2: screen, stratify, and obtain baseline investigations

Apply the NICE or ASPEN risk criteria at admission and again before initiating any nutrition therapy (oral, enteral, or parenteral). Document baseline weight, BMI, recent intake, weight trajectory, alcohol history, and relevant medications. Order baseline serum phosphate, potassium, magnesium, calcium, sodium, glucose, urea, creatinine, and (where available) thiamine status before feeds begin[10,14].

### Step 3: replace thiamine before carbohydrate

Administer thiamine 100–300 mg intravenously or orally before any carbohydrate-containing feed and continue daily for at least 5–10 days in at-risk patients. In suspected Wernicke encephalopathy or overt deficiency, give 500 mg intravenously (dissolved in 100 ml of normal saline, given by infusion over a period of 30 min) three times daily for 2–3 days, then 250 mg daily for 3–5 days, or until clinical improvement[19].

### Step 4: correct electrolytes before and during feeding

Pre-existing hypophosphatemia, hypokalemia, or hypomagnesemia should be corrected (or replacement initiated) before nutrition is started; doing so does not require delaying feeding for more than a few hours. Empiric supplementation in high-risk patients, even with normal baseline electrolytes, reduces the incidence of biochemical RFS[10].

### Step 5–9: start low, advance slowly, monitor closely

The traditional recommendation for high-risk patients is to begin at 5–10 kcal/kg/day, increase to 25–30 kcal/kg/day over 4–7 days, restrict sodium and fluid initially, and check phosphate, potassium, and magnesium every 12–24 hours for the first 3–5 days. Patients deemed extremely high-risk (e.g., BMI <14 kg/m$^2$, no intake for >15 days) may begin at 5 kcal/kg/day[14]. Concurrent multivitamin and trace-element supplementation is recommended[10].

**Table 3** Stepwise approach to the prevention of refeeding syndrome in at-risk hospitalized patients

| Step | Action | Practical detail |
|---|---|---|
| 1 | Screen for RFS risk | Apply NICE/ASPEN criteria at admission and before each escalation |
| 2 | Baseline labs | P, K, Mg, electrolytes, glucose, urea, creatinine; consider thiamine |
| 3 | Thiamine first | 100–300 mg IV/PO before carbohydrate; continue 5–10 d |
| 4 | Replace electrolytes | Empiric P, K, Mg in high-risk patients; correct deficits before feeding (if severe) or during feeding. |
| 5 | Start low calories | 5–10 kcal/kg/d in high-risk; 5 kcal/kg/d if extreme risk (BMI <14) |
| 6 | Advance gradually | Increase by 5–10 kcal/kg/d every 24–48 h to goal 25–30 kcal/kg/d |
| 7 | Fluid and sodium | 20–25 ml/kg/d in high risk; increase slowly to 25–35 ml/kg/d; Monitor fluid balance closely; restrict Na to <1 mmol/kg/day in high risk |
| 8 | Monitor | P, K, Mg q12–24 h×3–5 d; daily weight, fluid balance, ECG if severe |
| 9 | Continue micronutrients | Multivitamin, B-complex, trace elements daily |

RFS=refeeding syndrome, NICE=National Institute for Health and Care Excellence, ASPEN=American Society for Parenteral and Enteral Nutrition P=phosphate/phosphorus, K=potassium, Mg=magnesium, BMI=body-mass index, Na=sodium, ECG=electrocardiography/electrocardiogram

### Management of established refeeding syndrome

When RFS is recognized, management has three concurrent priorities: aggressive electrolyte and thiamine replacement, attenuation of the caloric load, and supportive care for any organ dysfunction.

### Caloric strategy and electrolyte replacement

Stopping nutrition altogether is generally unnecessary and may worsen outcomes by prolonging starvation. Most authorities recommend reducing the caloric prescription by 50% and holding the rate constant until electrolytes have normalized for at least 24 hours, then resuming gradual advancement[10,25]. Intravenous electrolyte replacement should be used for symptomatic deficits, severe hypophosphatemia (<0.32 mmol/L), or any patient unable to tolerate oral or enteral routes[26].

### Monitoring during refeeding

Daily weight, fluid balance, vital signs, and a focused cardiopulmonary examination are essential. Phosphate, potassium, magnesium, glucose, and renal function should be checked at least every 12–24 hours during the first 3–5 days, with continuous electrocardiography for patients with severe hypophosphatemia, marked bradycardia, or QTc prolongation. Daily glucose monitoring is necessary because hyperglycemia is common with parenteral nutrition (PN) and may itself worsen the syndrome by increasing insulin secretion[26].

## Disaster and flood-affected populations: lessons from the 2025 Hat Yai flood

### How disasters create the conditions for RFS

Acute large-scale disasters create the metabolic preconditions for RFS in three converging ways: (i) involuntary fasting or markedly restricted intake during the impact phase; (ii) loss of access to chronic disease care (dialysis, oncology, heart failure, parenteral feeding) that drives further catabolism; and (iii) abrupt re-exposure to carbohydrate-dominant relief food once aid arrives[13,27]. The 2025 Hat Yai flood illustrates each of these elements (Figure 2). Between 19 and 21 November 2025, the city received roughly 630 mm of rain over 72 hours, the heaviest single-day rainfall in three centuries—with floodwaters reaching two metres in many districts; over 243,000 residents of

Hat Yai and more than 3.5 million people across nine southern provinces were affected, and at least 145–170 deaths were officially confirmed[12]. Hat Yai Hospital itself was inundated; chronic-disease patients lost access to scheduled dialysis, chemotherapy, and clinic visits; and many residents were trapped above floodwaters for several days. In the shelters that absorbed approximately 150,000 stranded people, capacity was repeatedly exceeded. Prince of Songkla University's shelter was built for 3,000 but housed 5,000, and several temple shelters held two to six times their planned occupancy, and the predominant food, when food was available, was instant noodles and rice[12].

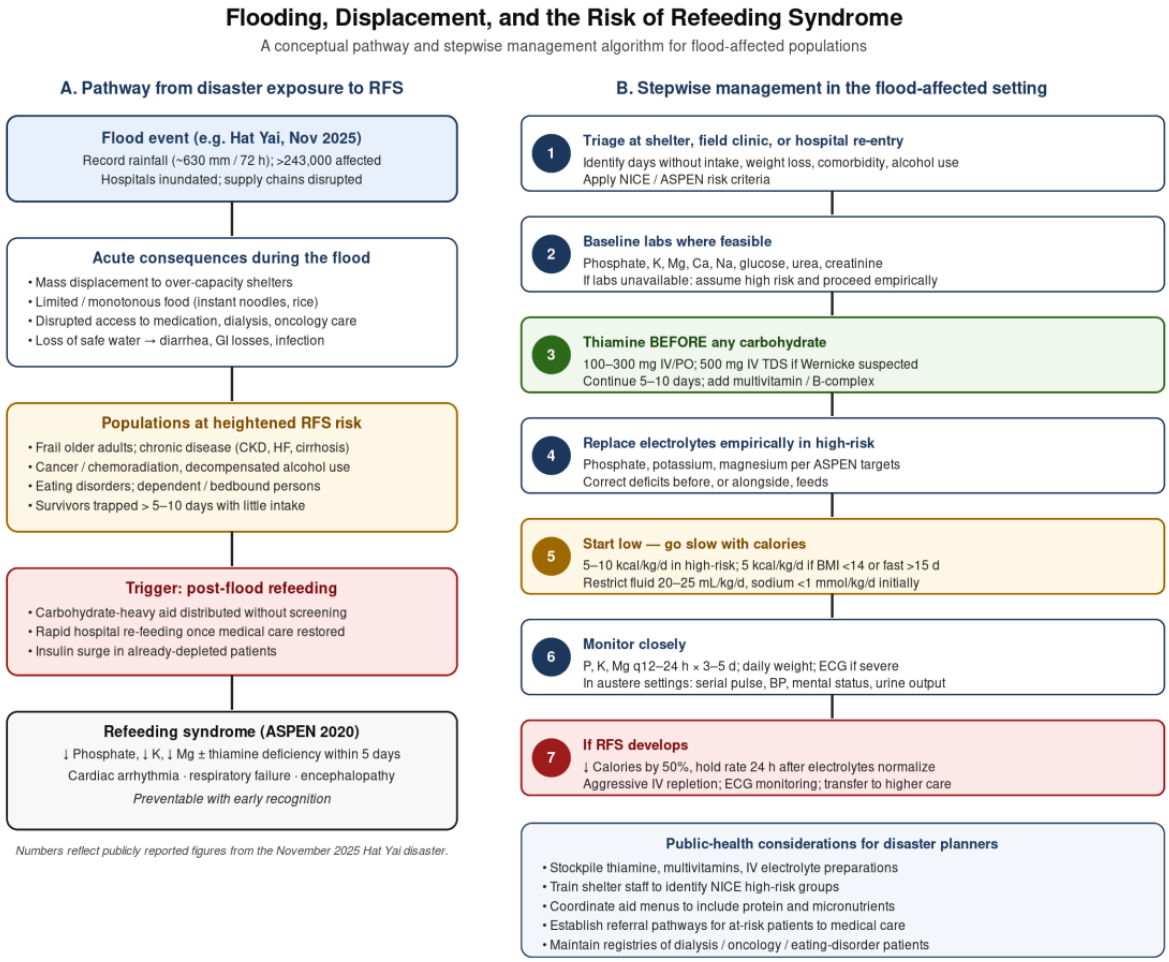
Several features of this and similar disaster scenarios make RFS particularly likely. First, the duration of inadequate intake, commonly 5–10 days, crosses the NICE threshold for "at-risk" status by itself, and longer for those trapped on upper floors without supplies[14]. Second, the affected populations contain a high proportion of patients already vulnerable to RFS: among the help requests catalogued in Hat Yai, there were approximately 2,000 elderly individuals and 1,000 young children identified as urgent; many were dependent, bedbound, or with chronic disease and had not been checked on during the early emergency response[12]. Third, when nutrition is restored, it is often abruptly carbohydrate-dominant. Rice, instant noodles, sweetened beverages, and high-energy biscuits are operationally easy to distribute but provide little phosphate, magnesium, potassium, or thiamine. Loss of safe drinking water and sanitation in flood-affected areas adds gastrointestinal losses (diarrhea, vomiting) that worsen baseline electrolyte and thiamine deficits[28,29]. Finally, displaced patients with eating disorders, advanced cancer, decompensated liver disease, or alcohol-use disorder may not be identified in shelters, and re-presentation to care after the flood often coincides with rapid nutritional rehabilitation in which RFS may be missed unless it is anticipated.

**Bedside and field-clinic management in resource-limited settings**

A practical, low-cost approach for the disaster setting (summarized in Figure 2) is to triage all displaced or recently rescued individuals at shelters and field clinics for RFS risk using simplified NICE/ASPEN criteria, duration without intake, recent weight loss, BMI, alcohol history, and pre-existing chronic disease, and to administer prophylactic thiamine (100–300 mg orally) before any meaningful carbohydrate load, even when laboratory testing is unavailable. Where electrolyte testing is feasible (e.g., in returning hospitals or mobile units), empiric oral phosphate, potassium, and magnesium supplementation should be offered to high-risk individuals. Initial energy provision in vulnerable patients should be deliberately conservative (5–10 kcal/kg/day), with sodium and fluid restriction, before advancing under monitoring. For populations without access to laboratory monitoring, serial clinical assessment, including pulse, blood pressure, mental status, urine output, and peripheral edema, remains the safety net.

**Disaster preparedness: institutional and public-health priorities**

Beyond the bedside, the Hat Yai experience reinforces several public-health priorities for disaster preparedness. Local hospitals and provincial health offices should pre-position parenteral and enteral thiamine, intravenous phosphate (potassium phosphate or sodium glycerophosphate), oral potassium and magnesium preparations, multivitamins, and trace elements as part of disaster pharmaceutical caches. Shelter-based food relief should be designed, ideally in collaboration with clinical nutrition services, to avoid extreme carbohydrate dominance and to provide some protein and micronutrients from day one. Vulnerable-patient registries (dialysis, oncology, eating-disorder programs, parenteral nutrition recipients) maintained at sub-district level can guide outreach




ASPEN=American Society for Parenteral and Enteral Nutrition, BMI=body-mass index, BP=blood pressure, Ca=calcium, CKD=chronic kidney disease, ECG=electrocardiogram, GI=gastrointestinal, HF=heart failure, IV=intravenous, K=potassium, Mg=magnesium, Na=sodium, NICE=National Institute for Health and Care Excellence, P=phosphate, PO=per os (oral), RFS=refeeding syndrome TDS=ter die sumendus/three times a day

**Figure 2** Flooding, displacement, and the risk of refeeding syndrome: a conceptual pathway and stepwise management algorithm for flood-affected populations. Panel A traces the cascade from flood exposure (illustrated by the November 2025 Hat Yai disaster), through acute consequences (mass displacement to over-capacity shelters, restricted and monotonous food, disrupted access to chronic disease care, loss of safe water) and identification of vulnerable subgroups (frail older adults, chronic disease, eating disorders, prolonged entrapment), to the post-flood refeeding trigger and ASPEN-defined refeeding syndrome. Panel B summarizes a seven-step bedside management algorithm that emphasizes triage at shelter or field-clinic level, prophylactic thiamine before any carbohydrate, empiric electrolyte replacement in high-risk individuals, conservative initial energy provision (5–10 kcal/kg/day), close clinical and biochemical monitoring, and early escalation if RFS develops, alongside public-health considerations for disaster preparedness.

during the impact and early recovery phases. Finally, brief refeeding-syndrome teaching modules should be incorporated into disaster-medicine training for emergency physicians, internists, nurses, and shelter coordinators in the flood-prone regions of southern Thailand and equivalent settings worldwide[29].

## Conclusion

Refeeding syndrome is a preventable but potentially fatal complication of nutrition therapy in the malnourished patient. Its biochemical signature, namely hypophosphatemia accompanied by hypokalemia, hypomagnesemia, and thiamine deficiency, reflects the metabolic shift from catabolism to anabolism when carbohydrates are reintroduced. Although increasingly recognized, RFS remains underdiagnosed and undertreated across both hospital and community settings.

The 2025 Hat Yai flooding illustrates that this risk extends well beyond the hospital ward. Large-scale disasters rapidly recreate the core conditions for RFS in populations who are often already nutritionally vulnerable. These conditions include prolonged involuntary fasting, disrupted access to chronic disease care, and abrupt refeeding with carbohydrate-dominant relief food. Clinicians and health systems in flood-prone regions should therefore integrate RFS awareness into disaster preparedness frameworks, pre-position thiamine and electrolyte supplies, and train shelter-based healthcare workers to triage at-risk individuals before food relief is distributed.

Whether in a hospital ward or a disaster shelter, a practical approach to RFS prevention and management rests on four pillars: (1) systematic risk screening before nutrition is initiated, (2) prophylactic thiamine and empiric electrolyte correction, (3) measured caloric advancement under close biochemical monitoring, and (4) institutional preparedness, encompassing pre-positioning of micronutrient supplies and training of disaster-response personnel. Standardized definitions, routine risk screening, and sustained clinician education remain the most cost-effective interventions for reducing the morbidity and mortality of this underrecognized but preventable syndrome.

### Author Contributions

Conceptualization, writing—original draft, and writing—review and editing: Apichat Kaewdech. Supervision and writing—review and editing: Chaitong Churuangsuk. All authors have read and approved the final version of the manuscript for submission.

## Conflict of interest

Apichat Kaewdech received research grants or support from Roche, Roche Diagnostics, and Abbott Laboratories, and honoraria from Roche, Roche Diagnostics, Abbott Laboratories, and Eisai. Chaitong Churuangsuk received honoraria from Abbott Laboratories, Fresenius Kabi, MEGA Lifesciences, Nestle Health Science, Novo Nordisk, Thai Otsuka, and Zuelling Pharma. However, these roles do not present any conflict of interest concerning the content of this work.

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