Fat overload syndrome in pediatric patients: One case and ten at risk

doi: 10.6133/apjcn.202511/PP.0002
Published online: November 2025

Running title: Fat overload syndrome in pediatric patients

Puthita Saengpanit MD, Phakwan Laohathai MD, Supawan Kunnangja MSc, Narumon Densupsoontorn MD[1]

Division of Nutrition, Department of Pediatrics, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

Authors' email addresses and contributions:
Puthita Saengpanit,
Email: puthita.sae@mahidol.ac.th
Individual contribution: Conception and design, data collection or compilation work, data analysis and interpretation, preparation of draft manuscript, doing revisions, and overall scientific management
Phakwan Laohathai,
Email: phakwan.lao@mahidol.ac.th
Individual contribution: Conception and design, data analysis and interpretation, doing revisions, providing critique, and overall scientific management
Supawan Kunnangja,
Email: supawan.kun@mahidol.ac.th
Individual contribution: Conception and design, data collection and compilation work
Narumon Densupsoontorn,
Email: narumon.den@mahidol.ac.th
Individual contribution: Providing critique and overall scientific management.

Corresponding Author: Dr Puthita Saengpanit, Division of Nutrition, Department of Pediatrics, Faculty of Medicine Siriraj Hospital, Mahidol University, 2 Wanglang Road, Bangkok Noi, Thailand 10700. Tel: +66 2 419 5674. Email: puthita.sae@mahidol.ac.th

ABSTRACT

Background and Objectives: Fat overload syndrome is a rare but serious complication associated with intravenous lipid emulsion in parenteral nutrition. Timely identification of causes, recognition of symptoms, close monitoring of complications, and appropriate management are vital for improving outcomes and preventing recurrence. This case series reviews etiologies, complications, management, and outcomes in pediatric patients who developed fat overload syndrome as well as those identified to be at risk of this condition. **Methods and Study Design:** A retrospective chart review was performed over a five-year period at a tertiary care hospital, including 11 pediatric patients who received lipid emulsions at infusion rates exceeding the recommended limit. Data on patient demographics, lipid emulsion details, clinical presentations, laboratory results, managements, and outcomes were collected and analyzed. **Results:** Eleven patients were included, ranging in age from 9 months to 15 years, with a male-to-female ratio of 7:4. The identified causes of intravenous lipid administration incidents were inadvertent switching of infusion rates between the parenteral nutrition solution bag and the lipid bag, and incorrect infusion pump programming. Fat overload syndrome was identified in one patient, presenting with oliguria and metabolic acidosis. Management primarily consisted of discontinuing intravenous lipid emulsions and providing supportive care. Although most patients had no complications, four required additional supportive interventions. **Conclusions:** Although uncommon, fat overload syndrome requires prompt recognition and timely intervention. This case series underscores the importance of careful parenteral lipid administration and vigilant monitoring of at-risk patients. Root cause analysis is critical to preventing recurrence of such incidents.

Key Words: fat overload syndrome, hypertriglyceridemia, pediatrics, intravenous fat emulsions, parenteral nutrition

INTRODUCTION

Intravenous lipid emulsion (ILE), also referred to as intravenous fat emulsion in literature, is a crucial component of pediatric parenteral nutrition (PN). It serves as a noncarbohydrate energy source and is delivered as an iso-osmolar emulsion with a high caloric density of 2 kcal/ml for 20% ILE. In addition to providing energy, ILE supplies the essential fatty acids linoleic acid and alpha-linolenic acid and facilitates the absorption of fat-soluble vitamins.[1] To prevent essential fatty acid deficiency, the minimum daily parenteral requirement of linoleic acid is ≥0.25 g/kg for preterm infants and ≥0.1 g/kg for term infants and children.[1]

The triglyceride (TG) portion of ILE is hydrolyzed by endothelial lipoprotein lipase, an enzyme whose activity is affected by factors such as prematurity, malnutrition, plasma lipid levels, metabolic acidosis, sepsis, and catabolic states. The maximum recommended ILE infusion rates are 3–4 g/kg/day (0.13–0.17 g/kg/hour) in infants and 2–3 g/kg/day (0.08–0.13 g/kg/hour) in children.[1,2] Infusion rates exceeding these limits can overwhelm the body's ability to oxidize fatty acids, increasing the risk of developing fat overload syndrome (FOS).

FOS is characterized by elevated plasma TG levels and arises when the lipid infusion rate surpasses the body's capacity to utilize fat. The two main causes of FOS are (1) excessive administration of ILE despite normal fat clearance and (2) normal infusion rates in the presence of reduced fat oxidation capacity.[3] FOS presents with a variety of symptoms, including headache, fever, respiratory distress, seizures, and hematological abnormalities, such as anemia, hemolysis, leukopenia, thrombocytopenia, low fibrinogen levels, coagulopathy, and spontaneous hemorrhage. Additional manifestations may include metabolic acidosis, transaminitis, conjugated hyperbilirubinemia, and pancreatitis.[4-6] These clinical features result from systemic lipid deposition and subsequent organ damage.

The pathogenesis of FOS is thought to involve lipid accumulation in the capillaries of vital organs - including the brain, lungs, kidneys, liver, and spleen - resulting in direct tissue damage and endothelial injury, which may trigger primary fibrinolysis through the release of tissue plasminogen activator.[6]

No specific treatment exists for FOS. Management focuses on discontinuing lipid infusions, monitoring for complications, and providing supportive care to relieve symptoms and prevent further deterioration.[3-6] Plasma exchange is reserved for severe cases to rapidly remove lipids and alleviate acute FOS symptoms.[4] These strategies are guided by the pathophysiology of FOS and aim to mitigate the systemic effects of lipid overload.

This study describes pediatric patients who received ILE at infusion rates exceeding recommended limits, placing them at risk for FOS. By analyzing these cases, we aim to identify the causes, clinical manifestations, and management strategies, with the goal of informing future research and improving the safety of PN in clinical practice.

MATERIALS AND METHODS

Study design

This retrospective study reviewed pediatric patients at risk of FOS who were admitted to Siriraj Hospital, Bangkok, Thailand, between July 2017 and June 2022. The study was approved by the Research Ethics Committee of the Faculty of Medicine Siriraj Hospital (COA No. Si 064/2023). Incident reports related to PN were examined, with parenteral lipid administration above the recommended limits defined as ILE exceeding 0.17 g/kg/hour in infants or 0.13 g/kg/hour in children older than 1 year. This criterion was applied to identify cases in which infusion rates may have exceeded the physiologic lipid metabolism capacity and potentially contributed to the development of FOS.

Data collection

Variables reviewed included patients' underlying diseases, anthropometric data, etiologies of administration error, and details of the ILE administration (types of fat, infusion rate, and total dosage). Nutritional status was classified according to the World Health Organization's classification criteria with a z score < -2 for weight-for-age, height-for-age, and body mass index-for-age defined as underweight, stunted, and wasted/thin, respectively.[7]

Clinical and laboratory assessment

Clinical manifestations of FOS were reviewed and included systemic symptom (fever), neurologic symptoms (headache, seizure, weakness), respiratory distress, hematologic complication (spontaneous hemorrhage), renal involvement (oliguria) and metabolic or organ-specific abnormalities (metabolic acidosis, pancreatitis), reflecting its systemic nature.

Laboratory investigations included serum TG levels, complete blood count (CBC), coagulation profile with fibrinogen, and liver function tests, which were used to evaluate the presence and severity of FOS.

Management and outcomes

Management consisted of discontinuing ILE, supportive care, and plasma exchange when indicated. Effectiveness was evaluated by clinical recovery and improvement in laboratory parameters, with outcomes reviewed to inform future practice.

RESULTS

Participant characteristics

Eleven pediatric patients who received ILE at rates exceeding age-specific recommendations were included (Table 1). Ages ranged from 9 months to 15 years, and seven males. Nutritional assessment identified one patient as underweight, one as thin, and one as stunted according to the World Health Organization's criteria.[7] Underlying diseases varied and included hematologic disorders, metabolic conditions, malignancies, and other complex chronic illnesses.

Etiologies of parenteral lipid administration errors and details of ILE

Table 2 summarizes the etiologies of parenteral lipid administration errors: inadvertent switching of infusion rates between the PN solution bag (containing dextrose, amino acids, and minerals) and the lipid bag (6/11 cases, 55%) and incorrect infusion pump programming (5/11 cases, 45%). Administered ILE included pure soybean oil and fish oil-containing composite lipid emulsions with infusion rates ranging from 0.18 to 2.88 g/kg/hour. All patients received continuous infusion except for patient 11, who was on cyclic PN; following an unintentional rate switch, ILE was titrated from 0.27 to 0.63 g/kg/hour over 2 hours, then 1.25 g/kg/hour for 50 min until completion.

Clinical and laboratory findings

Among the 11 patients, one (patient 11) developed oliguria and another (patient 4) had fever consistent with prior records, not deemed clinically significant. No patients exhibited headaches, seizures, motor weakness, dyspnea, or spontaneous bleeding.

Laboratory results are summarized in Table 3. Three patients demonstrated hypertriglyceridemia meeting the ESPGHAN/ESPEN/ESPR/CSPEN threshold for ILE dose reduction: patient 1 (408 mg/dL; infant cutoff 265 mg/dL), patients 10 (587 mg/dL) and patient 11 (1,684 mg/dL; cutoff 400 mg/dL for older children).[1]

Management and outcomes

Clinical and laboratory findings indicative of FOS were observed in one patient (patient 11). Most patients required no additional interventions after discontinuing ILE, whereas patients 2, 4, and 8 needed further management.

Patient 11

Patient 11, a 2-year-old girl with Down syndrome, atrial septal defect, and acute myeloid leukemia, developed severe enteritis with bowel perforation requiring bowel resection surgery and jejunostomy placement. On the day of the event, she was medically stable and received 14-hour cyclic PN. Owing to a switch in infusion rates between the PN solution and lipid bags, ILE was administered at titrated rates of 0.27 g/kg/hour for 1 hour, 0.63 g/kg/hour for the second hour, and 1.25 g/kg/hour for the final 50 minutes.

Her vital signs remained stable (temperature 36.4 □C, blood pressure 105/46 mmHg, respiratory rate 30/min, and oxygen saturation of 95% on room air). Blood investigations revealed severe hypertriglyceridemia (TG 1,684 mg/dL), hemoglobin (Hb) 11.3 g/dL, white blood cell (WBC) 2,080/cu mm, and platelet 43,000/cu mm, with CBC parameters comparable to those of the previous day. Additional laboratory values included blood urea nitrogen 12.4 mg/dL, creatinine 0.3 mg/dL, sodium 140 mmol/L, potassium 3.6 mmol/L, chloride 95 mmol/L, and bicarbonate 15 mmol/L.

After discontinuation of PN, urine output over 8 hours decreased to 0.5 mL/kg/hour while receiving maintenance intravenous fluid (5% dextrose in half-normal saline). Other clinical conditions and vital signs remained stable. Two 20 mL/kg normal saline boluses were subsequently administered, resulting in improved urine output to more than 1 mL/kg/hour.

Serum TG decreased to 102 mg/dL and bicarbonate increased to 26 mmol/L within 9 hours, while creatinine remained stable. Serum amylase (34 U/L; reference range 25-101 U/L) and lipase (38 U/L; reference range 4-39 U/L) were both within normal limits at 9 hours. Liver enzymes were mildly elevated (alanine aminotransferase [ALT] rising from 38 U/L to 90 U/L, and aspartate aminotransferase [AST] from 18 U/L to 62 U/L) at 9 hours, then declined to 55 U/L and 28 U/L, respectively, by day 3.

Patient 2

Patient 2, with β-thalassemia/HbE post-bone marrow transplantation complicated with mucositis and diarrhea, received ILE at 200 mL/hour for 15 minutes due to incorrect infusion pump programming instead of the intended 3.2 mL/hour. He remained asymptomatic immediately after the event. However, 12 hours later, laboratory tests revealed a drop in

platelet count from 31,000/cu mm the previous day to 17,000/cu mm, necessitating platelet transfusion.

Patient 4

Patient 4, with Gorham–Stout disease, pulmonary hypertension, and chronic disseminated intravascular coagulation (DIC), was admitted with pneumonia and pleural effusion and received PN due to dyspnea and poor oral intake. On the day of the incident, a switch in infusion rates between the PN solution and lipid bags resulted in ILE administration at 0.18 g/kg/hour for 4.5 hours. The patient developed a fever of 38 °C and had a respiratory rate of 30-35/minute, consistent with his baseline condition; oxygen saturation remained 98%.

After the discontinuation of ILE, an immediate assessment showed a serum TG 356 mg/dL, Hb 8.1 g/dL, platelet 23,000/cu mm, prothrombin time (PT) 16 seconds, an international normalized ratio (INR) 1.36, and partial thromboplastin time (PTT) 39.4 seconds, similar to previous values. Fibrinogen decreased from 69 mg/dL to 51.4 mg/dL, then further to 44.6 mg/dL at 9 hours, prompting cryoprecipitate transfusion.

Patient 8

Patient 8, with acute lymphoblastic leukemia, febrile neutropenia, and typhlitis, received PN solution at 83 mL/hour and ILE at 21 mL/hour. During a blood transfusion, the lipid infusion was temporarily paused, but afterward was mistakenly restarted at 83 mL/hour (0.29 g/kg/hour) for 3 hours 40 minutes before being discontinued.

The patient remained asymptomatic. Post-event labs showed TG 102 mg/dL, WBC 110/cu mm, and platelets 33,000/cu mm. Nine hours later, platelets dropped to 22,000/cu mm, requiring transfusion; no bleeding occurred.

Patient 10

Patient 10, with a history of β-thalassemia/HbE post-bone marrow transplantation, mucositis, and diarrhea, developed thrombocytopenia (platelet 26,000/cu mm) 2 hours after the event, similar to baseline. The initial prolonged PTT of 124.7 seconds decreased to 23 seconds within 10 hours without intervention.

Survival outcomes

All patients survived, except patient 6 who passed away on the 11th day following the event due to severe underlying conditions.

DISCUSSION

In this case series, we reported 11 pediatric patients who received ILE at doses exceeding the age-specific recommendations, thereby placing them at risk of FOS. Most patients did not experience serious complications, and none required plasma exchange. One patient developed clinical features consistent with FOS necessitating fluid resuscitation, while three others required additional supportive treatments beyond lipid cessation, including blood component transfusions. The two leading causes of parenteral lipid administration errors were (1) inadvertent switching of infusion rates between the PN solution bag and the lipid bag and (2) incorrect programming of the infusion pump.

Following the incident, patients 2, 4, and 8 required blood component transfusions, including cryoprecipitate and platelets. However, each of these patients had pre-existing conditions that increased their likelihood of requiring such interventions. Patient 2, who had β-thalassemia/HbE after bone marrow transplantation (day 18), presented with thrombocytopenia prior to the event; his platelet count further declined and necessitated transfusion. Unfortunately, post-event serum TG levels were not measured in this patient. Patient 4 had chronic DIC, predisposing him to thrombocytopenia, coagulopathy, and hypofibrinogenemia; his laboratory results frequently demonstrated low platelet and fibrinogen levels. Patient 8, diagnosed with acute lymphoblastic leukemia, also had pre-existing thrombocytopenia before the incident. Notably, the serum TG concentrations in patients 4 and 8 remained below 400 mg/dL (356 mg/dL in the 11-year-old and 102 mg/dL in the 14-year-old, respectively), underscoring the uncertainty as to whether their clinical and laboratory abnormalities were attributable to FOS, their underlying diseases, or both. In contrast, patients 1 and 10 had elevated TG levels (408 mg/dL and 587 mg/dL, respectively), but neither developed symptoms nor required additional treatment. Collectively, these findings highlight the importance of vigilant monitoring, careful symptom assessment, and individualized management based on each patient's clinical context.

With respect to serum TG levels, previous case reports of FOS have demonstrated that most symptomatic patients, or those who developed complications, had TG levels exceeding 1,000 mg/dL. Reported values ranged from 1,398 to 6,417 mg/dL,[5,6,8-11] or patients exhibited lipidemic blood samples.[12] This is consistent with our findings in patient 11, who had a TG

level of 1,684 mg/dL. She subsequently developed oliguria and new-onset metabolic acidosis following the event, with no alternative explanation identified, and required fluid resuscitation for recovery. Her ALT level was slightly elevated post-event but gradually declined thereafter. Manifestations such as low urine output and metabolic acidosis have also been reported by Schlegelmilch et al.,[8] in which an 11-month-old girl received ILE infusion at 1.12 g/kg/hour with a total dose of 15.68 g/kg. Similarly, metabolic acidosis[4,5,9] and transaminitis[6,11] have been frequently described as complications of excessive lipid infusion.

In our cohort, the two leading causes of parenteral lipid administration errors were (1) inadvertent switching of infusion rates between the PN solution bag and the lipid bag and (2) incorrect programming of the infusion pump. These findings are aligned with the report by Cole et al.,[13] in which nine children received rapid lipid infusions at rates ranging from 0.4 to 3 g/kg/hour. In eight of those cases, the infusion rates of the amino acid and the lipid bags were inadvertently switched, while in the remaining case, the total daily lipid dose was mistakenly programmed into the pump as the hourly rate, reflecting incorrect pump programming.

At our institution, PN administration involves two nurses: Nurse A prepares the infusion set, programs the infusion rate, and delivers PN to the patient, while Nurse B rechecks the entire process at the bedside to ensure correct rates for the appropriate bags. Previously, the intravenous infusion pumps and stands for PN solution and lipid bags were identical, with both infusion sets covered by green cloths for light protection and both bags hung on the same stand. This arrangement hindered detection of switched infusion rates during verification. In addition, the small display size of infusion rate numbers on both bags further increased the risk of unnoticed errors.

Following a root cause analysis, a multidisciplinary conference involving the pediatric nutrition team, pharmacists, and nurses identified several safety interventions. The workflow was modified as follows (Figure 1):

1. Light-protection coverings for the infusion sets were color-coded—green for PN solution bag and blue for lipid bag.
2. The PN solution and lipid bags were hung on separate stands.
3. The infusion rate display was enlarged.
4. Name tags indicating “PN solution” or “Fat” were attached to each infusion set after applying the light-protection coverings.

Under the revised workflow, Nurse A performed all PN administration steps, including PN composition validation, patient identification, application of light-protection coverings,

placement of bags on separate stands, and programming and starting the infusion pumps. Nurse B then independently verified each step. This strengthened verification process was implemented to enhance safety and prevent recurrence of similar errors.

All-in-one (AIO) admixtures are considered the gold standard for PN administration because they reduce compounding errors, lower costs, and decrease infection risk. However, in our setting, the use of AIO admixtures remains limited because these formulations are currently unavailable for young children in Thailand, particularly those under 2 years of age. In addition, many of our patients have complex underlying conditions that alter metabolism, affect energy and nutrient requirements, and are frequently complicated by electrolyte imbalances. These factors necessitate individualized PN formulations.

At Siriraj Hospital, PN solution and ILE are prepared by the Pharmacy Intravenous Admixture Services (PIVAS) unit—formally known as the Pediatric Medication Preparation Unit of the Pharmacy Department—which provides centralized compounding of sterile PN solution and ILE tailored to individual patients, thereby ensuring safe and precise administration. Nevertheless, expanding the use of standardized AIO formulations remains an important future goal to further enhance PN safety, efficiency, and accessibility, despite current limitations.

The use of commercially available light-protective tubing could also reduce administration errors by eliminating the need for cloth covering. However, this equipment is not yet available at our institution, and its accessibility remains limited in many hospitals across middle-income countries. By sharing our experience, we aim to provide a practical model that may be adapted in other institutions, particularly those in resource-limited settings, and to contribute to ongoing efforts to improve PN safety.

Conclusions

FOS occurs when the rate of ILE infusion exceeds the hydrolytic capacity of endothelial lipoprotein lipase, resulting in a spectrum of complications. Prevention requires adherence to recommended infusion rates, correct administration practices, and vigilant monitoring of patients with impaired lipoprotein lipase activity, who may be particularly susceptible to symptoms. Early detection, continuous monitoring, and timely management are critical to improving patient outcomes; nevertheless, prevention remains the most effective strategy.

CONFLICT OF INTEREST AND FUNDING DISCLOSURE

All the authors have no conflicts of interest to declare.

REFERENCES

1. 1. Lapillonne A, Fidler Mis N, Goulet O, van den Akker CHP, Wu J, Koletzko B. ESPGHAN/ESPEN/ESPR/CSPEN guidelines on pediatric parenteral nutrition: Lipids. Clin Nutr. 2018;37(6 Pt B):2324-36. doi: 10.1016/j.clnu.2018.06.946.
2. Koletzko B, Goulet O, Hunt J, Krohn K, Shamir R. 1. Guidelines on Paediatric Parenteral Nutrition of the European Society of Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) and the European Society for Clinical Nutrition and Metabolism (ESPEN), Supported by the European Society of Paediatric Research (ESPR). J Pediatr Gastroenterol Nutr. 2005;41 Suppl 2:S1-87. doi: 10.1097/01.mpg.0000181841.07090.f4.
3. Zhanqiang Zhang HY, Kaitao Yuan, Hanping Shi. Diagnosis and Management of Fat Overload Syndrome in an Elderly Man. Case Reports in Clinical Medicine. 2014;3:554-6. doi: 10.4236/crcm.2014.310120.
4. Hojsak I, Kolaček S. Fat overload syndrome after the rapid infusion of SMOFlipid emulsion. JPEN J Parenter Enteral Nutr. 2014;38:119-21. doi: 10.1177/0148607113482001.
5. Khasawneh W, Bani Hani S. Intravenous lipid emulsion overdose in infancy: A case report and overview of opportunities, challenges and prevention. Drug Saf Case Rep. 2018;5:13. doi: 10.1007/s40800-018-0079-y.
6. Haber LM, Hawkins EP, Seilheimer DK, Saleem A. Fat overload syndrome. An autopsy study with evaluation of the coagulopathy. Am J Clin Pathol. 1988;90:223-7. doi: 10.1093/ajcp/90.2.223.
7. De Onis M. World Health Organization Reference Curves. In M.L. Frelut (Ed.), The ECOG's eBook on Child and Adolescent Obesity. 2015. [cited 2025/9/29]; Available from: https://ecogobesity.wpenginepowered.com/wp-content/uploads/2014/10/ECOG-Obesity-eBook-World-Health-Organization-Reference-Curves-V1.pdf
8. Schlegelmilch M, Feder J, Creery D. Inadvertent acute lipid injectable emulsion overdose resulting in fat overload syndrome and pancreatitis in a patient with TPN dependence. JPGN Rep. 2021;3:e146. doi: 10.1097/PG9.0000000000000146.
9. Badr M, Goulard M, Theret B, Roubertie A, Badiou S, Pifre R, et al. Fatal accidental lipid overdose with intravenous composite lipid emulsion in a premature newborn: a case report. BMC Pediatr. 2021;21:584. doi: 10.1186/s12887-021-03064-6.
10. Hoofien A, Mozer Y, Guz-Mark A, Hoffer V, Landau D, Shamir R. Inadvertent rapid lipid emulsion administration. Isr Med Assoc J. 2019;21:129-30.
11. Heyman MB, Storch S, Ament ME. The fat overload syndrome. Report of a case and literature review. Am J Dis Child. 1981;135:628-30. doi: 10.1001/archpedi.1981.02130310034012.
12. Khriesat W, Abu-Ekteish F. A Fat overload after fat emulsion high dose infusion in an Infant. Pediatrics & Therapeutics. 2015;5:3. doi: 10.4172/2161-0665.1000246.

13. Cole C, Robertson S. Nine cases of unintentional rapid infusion of lipid emulsion in children: root cause analysis and changes to practice. Arch Dis Child. 2014;99:e3. doi: 10.1136/archdischild-2014-306798.4.

Table 1. Demographic characteristics of patients receiving intravenous lipid emulsion at rates exceeding recommended limits (n = 11)

Patient no.	Age	Sex	Underlying diseases	Anthropometric data					
				Weight (kg)	Length/ height (cm)	WAZ†	LAZ/ HAZ†	WLZ/ WHZ†	BMI z-score†
1	9 mo	F	Skeletal dysplasia, intestinal malrotation, gastrointestinal dysmotility	8.9	67	0.64	-1.37	1.83	1.92
2	3 y	M	β-thal/HbE post-BMT with mucositis and diarrhea	13.9	95	-0.82	-1.43	0.01	0.15
3	7 y	F	Pre-B-cell ALL	40	120	2.67	-0.96	NA	3.81
4	11 y	M	Gorham-Stout disease with PHT, pleural and pericardial effusions, and chronic DIC	41	140	NA	-1.23	NA	1.64
5	9 y	F	MMA with influenza A infection	20.6	121	-2.15	-1.98	NA	-1.29
6	15 y	M	Malignant pheochromocytoma with bowel perforation	42.5	165	NA	-0.83	NA	-2.49
7	11 y	M	MMA with CKD	32.9	138	NA	-1.5	NA	-0.11
8	14 y	M	ALL with typhlitis	58	164	NA	-0.31	NA	0.79
9	6 y	M	Second-degree burn (68% TBSA)	22	118	0.36	0.18	NA	0.34
10	4 y	M	β-thal/HbE post-BMT with mucositis and diarrhea	13.5	98	-1.77	-1.85	-1.08	-0.84
11	2 y	F	Down syndrome with AML and enteritis with bowel perforation	11.2	83	-1.26	-2.73	0.47	0.77

ALL, acute lymphoblastic leukemia; AML, acute myeloid leukemia; BMI, body mass index; BMT, bone marrow transplantation; β-thal/HbE, beta-thalassemia/hemoglobin E disease; CKD, chronic kidney disease; cm, centimeter; DIC, disseminated intravascular coagulation; F, female; HAZ, height-for-age z-score; kg, kilogram; LAZ, length-for-age z-score; M, male; MMA, methylmalonic acidemia; NA, not available; no, number; PHT, pulmonary hypertension; TBSA, total body surface area; WAZ, weight-for-age z-score; WHZ, weight-for-height z-score; WLZ, weight-for-length z-score.
†WAZ, LAZ/HAZ, WLZ/WHZ, and BMI z-score were calculated using WHO Anthro and WHO AnthroPlus softwares.

Table 2. The characteristics of participants

Patient no.	Etiologies of FOS		Details of ILE				
	Rate switching between PN solution (dextrose & amino acid) bag and lipid bag	Incorrect infusion pump programming	Type of fat	Trade name	Infusion rate (g/kg/h)	Duration (hours)	Total dosage given (g/kg)
1	+		SO + MCT + OO + FO	SMOFlipid	0.66	4.25	2.8
2		+	SO	Intralipid	2.88	0.25	0.72
3	+		SO + MCT + OO + FO	SMOFlipid	0.53	0.92	0.49
4	+		SO + MCT + OO + FO	SMOFlipid	0.18	4.5	0.81
5		+	SO	Intralipid	0.19	3	0.57
6	+		SO + MCT + OO + FO	SMOFlipid	0.26	2	0.52
7		+	SO	Intralipid	0.24	2.33	0.56
8		+	SO	Intralipid	0.29	3.67	1.06
9	+		SO + MCT + OO + FO	SMOFlipid	0.57	5	2.85
10		+	SO	Intralipid	0.82	1.83	1.5
11[†]	+		SO + MCT + OO + FO	SMOFlipid	0.27	1	1.94
					0.63	1	
					1.25	0.83	

FO, fish oil; ILE, intravenous lipid emulsion; MCT, medium-chain triglycerides oil; no, number; OO, olive oil; PN, parenteral nutrition; SO, soybean oil
[†]Patient 11 was on cyclic PN.

Table 3. Laboratory findings within 4 hours of the event in patients receiving ILE at rates exceeding recommended limits (n = 11)

Patient no.	TG (mg/dL)	Complete blood count			PT (sec)	INR	PTT (sec)	Fibrinogen (mg/dL)	Liver function test					
		Hb (g/dL)	WBC (cells/cu mm)	Platelet (cells/cu mm)					ALT (U/L)	AST (U/L)	TB (mg/dL)	DB (mg/dL)	Alb (g/dL)	ALP (U/L)
1	408	8.4	10 770	199 000	13.2	1.21	22.3	NA	38	37	0.15	0.12	3.8	401
2[†]	NA	9.4	6640	17 000	NA	NA	NA	NA	14	35	0.39	0.19	3.2	207
3	NA	10.2	5690	140 000	15.7	1.39	31.5	205.8	36	15	1	0.36	2.8	93
4	356	8.1	8720	23 000	16	1.36	39.4	51.4	20	35	0.98	0.6	4.3	70
5[†]	220	13.3	4240	116 000	12	1.05	25.7	NA	130	262	0.24	0.12	3.8	142
6	272	10.8	21 630	70 000	15	NA	31.5	329.9	27	46	3.25	2.83	3	168
7[†]	210	8.7	10 020	162 000	10	0.88	18.3	192.7	121	72	0.75	0.47	4.1	107
8	102	9.2	110	33 000	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
9	NA	11	10 350	277 000	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
10	587	8.5	5720	26 000	13.5	1.24	124.7	NA	187	81	0.94	0.66	3.5	238
11	1684	11.3	2080	43 000	12.1	1.09	25.9	NA	38	18	0.61	0.6	2.9	541

Alb, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; DB, direct bilirubin; Hb, hemoglobin; ILE, intravenous lipid emulsion; INR, international normalized ratio; NA, not available; no., number; PT, prothrombin time; PTT, partial thromboplastin time; TB, total bilirubin; TG, triglyceride; WBC, white blood cell
[†]Laboratory investigations were assessed 12 hours (patient 2), 14 hours (patient 5), and 8 hours (patient 7) after the event

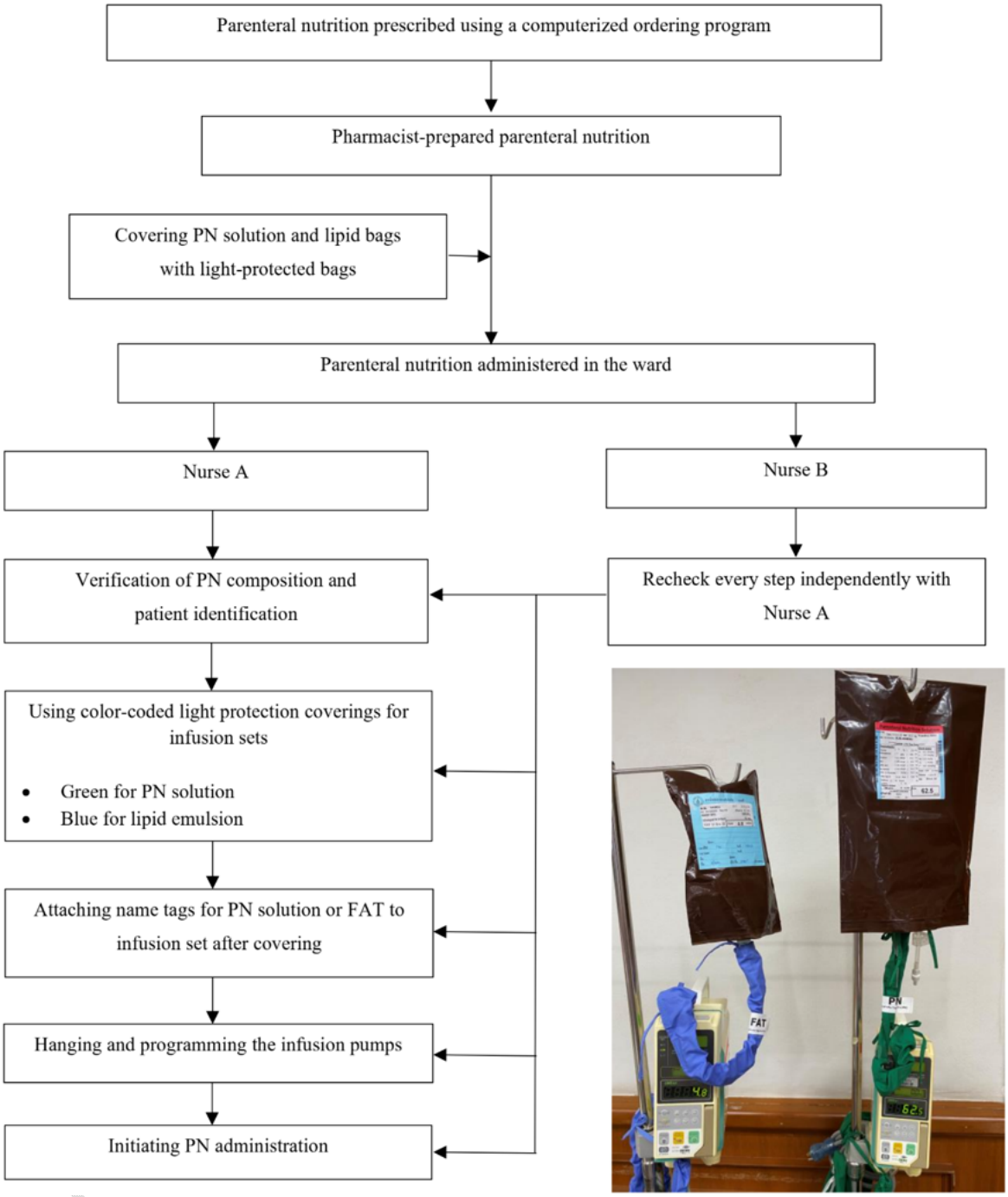

Figure 1. Workflow and verification procedures implemented for parenteral nutrition administration. PN, parenteral nutrition