



higher rate of mechanical, metabolic, and infectious complications than peripheral vein PN. Mechanical complications include those related to insertion of the central venous catheter (e.g., pneumothorax, hemothorax, malposition of the catheter, thrombosis). Infectious complications include catheter-related bloodstream infections and non-catheter-related infections. The risk for these infections appears to be increased with use of non-subclavian vein central venous access (e.g., jugular vein, femoral vein) and multiple-use catheters with non-dedicated PN infusion ports used for additional purposes such as blood drawing or medication administration. Poorly controlled blood glucose levels (>140 to 180 mg/dL) are not uncommon in patients requiring central vein PN and are associated with an increased risk of nosocomial infection. Risk factors for hyperglycemia include poorly controlled blood glucose at PN initiation; use of high dextrose concentrations (>10%) in the initial few days of PN administration or too rapid an increase in total dextrose load; insufficient exogenous insulin administration; inadequate monitoring of blood glucose responses to central vein PN administration; and administration of corticosteroids and vasopressor agents such as norepinephrine (which stimulate gluconeogenesis and cause insulin resistance).


Recent data also suggest that inadequate or no provision of the amino acid glutamine may increase infection risk in patients requiring PN. This amino acid appears to be conditionally essential in catabolic states and serves as an important fuel for immune cells and cells of the gut mucosa. Several expert panels now recommend that glutamine be routinely added to the PN in ICU patients, but this practice remains controversial because some studies show no benefit (or even harm) in certain patient subgroups and an improvement in hospital mortality has not been documented.

Studies on nutrient utilization efficiency and metabolic complications in severely catabolic patients suggest that lower amounts of total energy and protein/amino acids should be administered than were routinely given in the past, particularly in unstable and ICU patients. High calorie, carbohydrate, amino acid, and fat loads (“hyperalimentation”) are easily administered via central vein PN but can induce severe metabolic complications, including carbon dioxide overproduction, azotemia, hyperglycemia, electrolyte alterations, and hepatic steatosis and injury (E-Table 68-5). Dextrose and lipid doses in PN should be advanced over several days after initiation, with close monitoring of the blood glucose concentration, electrolytes, triglycerides, organ function tests, intake and output measurements, and the clinical course.

Refeeding syndrome with central vein PN administration is relatively common in patients at risk, including those with preexisting malnutrition, electrolyte depletion, alcoholism, or prolonged periods of intravenous hydration therapy (e.g., 5% dextrose) without nutritional support, all of which are common in hospital patients. Refeeding syndrome is mediated by administration of excessive intravenous dextrose (>150 to 250 g, for example in 1 L of PN containing 15% to 25% dextrose). This, in turn, markedly stimulates insulin release, which rapidly lowers blood concentrations of potassium, magnesium, and especially phosphorus as a result of intracellular shifts and utilization in

carbohydrate metabolic pathways. Administration of high doses of carbohydrate also consumes thiamine, which is required as a cofactor for carbohydrate metabolism and can precipitate symptoms of thiamine deficiency (see E-Table 68-2), especially in patients with poor thiamine nutriture at baseline. Hyperinsulinemia also tends to cause sodium and fluid retention at the level of the kidney. Together, fluid and sodium retention, the drop in electrolytes (which can cause arrhythmias), and hypermetabolism due to excessive calorie provision can result in heart failure, especially in patients with preexisting heart disease and cardiac muscle atrophy due to prolonged protein-energy malnutrition. Prevention of refeeding syndrome requires vigilance to identify patients at risk; use of initially low PN dextrose concentrations; empiric provision of higher doses of potassium, magnesium, and phosphorus based on current blood levels and renal function; and supplemental thiamine (100 mg/day for 3 to 5 days).

If home PN is indicated, the primary physician should consult with social service professionals to identify appropriate home care companies and nutrition support professionals to assess intravenous line access, metabolic status, and the home PN order and to arrange for follow-up care and monitoring of PN. It is important not to arrange for rapid discharge of hospitalized patients newly started on PN. Obtaining appropriate venous access and monitoring of fluid and electrolyte status over a 2- to 3-day period is an important aspect of care for most patients started on PN, and it is imperative for those with severe malnutrition and those at risk for refeeding syndrome.

 For a deeper discussion on this topic, please see Chapters 214, “Nutritional Assessment,” and 215, “Protein-Energy Malnutrition” in Goldman-Cecil Medicine, 25th Edition.

SUGGESTED READINGS

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