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Cycling parenteral nutrition in a neonatal surgical patient: An argument for increased utilization

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ABSTRACT

Cycling total parenteral nutrition (TPN) limits hepatic dysfunction in adults and children with intestinal failure. Due to immature glucose homeostasis, this method is infrequently utilized in the neonatal population.

Herein, we describe a successful case of cycled TPN in a neonate with complicated gastroschisis and short bowel syndrome. Within three weeks the patient had a normalized direct bilirubin and did not require pharmacologic intervention. There were two episodes of asymptomatic hypoglycemia associated with intestinal obstruction.

1. Introduction

Cycling total parenteral nutrition (TPN) limits hepatic dysfunction and facilitates improved quality of life in adults and children with intestinal failure [1]. Over time, this has been employed in pediatric patients with functional and anatomical short gut syndrome. However, the application of cycled TPN has been infrequently utilized in neonates due to the concern for severe hypoglycemia [2]. Here we discuss a successful case of cycled TPN in a neonate with short bowel syndrome secondary to complicated gastroschisis.

1.1. Case report

Patient is a Caucasian male with prenatally diagnosed gastroschisis born at 36 weeks gestation to a G2P1 mother. At the time of the delivery he was taken to the operating room for surgical closure and found to have both a dilated duodenum and proximal jejunum, and a small unused distal transverse colon. No other intestinal anomaly was identified during the initial operation. Due to the concern for complicated gastroschisis with suspected atresia, the patient was transferred to a tertiary children's hospital for further care.

The patient was maintained with gastric decompression and parenteral nutrition with the plan to surgically intervene at approximately 6 weeks of age. However, just prior to 4 weeks of age, he developed increasing liver dysfunction demonstrated by a direct hyperbilirubinemia and his TPN was transitioned to a cycling regimen. Due to signs of developing TPN cholestasis, the patient was taken back to the operating room 2 weeks early for exploration. The suspected distal duodenal atresia was confirmed. The bulbous and dilated atretic end was resected, and the remaining few centimeters of distal dilated duodenum were tapered and anastomosed to 47 cm of jejunum and ileum (these additional 47 cm were identified during this laparotomy and not appreciated at the original operation). Two additional atretic segments were identified downstream, resected and anastomosed together, ending in a functional ostomy. Total small intestine length was 60 cm from the Ligament of Treitz to the ostomy incorporating three hand sewn anastomoses. The right colon, hepatic flexure and proximal transverse colon were atretic and removed. A very small caliber mid-transverse colon was identified and brought out as a mucous fistula for eventual rehabilitation. Postoperatively the patient failed to have return of bowel function via the end ileostomy and was diagnosed by contrast imaging with a stricture at the most distal anastomosis, located 4 cm proximal to the stoma. Rehabilitation of

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the distal colon was performed by administering 10 milliliters/kilogram(kg) of normal saline as an antegrade enema via the mucous fistula two times per day. Seven weeks later he underwent stricture resection, stoma takedown and reanastomosis. Although the patient stooled initially, he developed bilious emesis and was taken back to the operating room for a third time. The patient's most proximal anastomosis had obstructed and was revised in a Heineke-Mikulicz fashion.

Cycled TPN was maintained from the original signs of liver dysfunction throughout the patient's complicated course. Ultimately the patient was weaned off TPN 35 days following his third operation. At that time he had advanced to goal volume enteral feeds. Over the next three weeks, the patient had fortification of his enteral feeds to ensure adequate weight gain. He also had soluble fiber and sodium supplementation to counteract the fluid and electrolyte loss due to decreased small intestine length.

Within the first 48 h of life, the patient was placed on fat emulsion 20% at 1 g(g)/kg/day(d) and increased up to 3 g/kg/d. Following the development of direct hyperbilirubinemia, his lipid delivery was restricted to 1 g/kg/d where it remained for the duration of TPN. The glucose infusion rate ranged from 12.15 to 16.15 mg(mg)/kg/minute(min) when on continuous TPN based on the patient's caloric growth needs of 90–105 kilocalories/kg/d. While on cycled TPN the glucose infusion rate was 16.7–19.0 mg/kg/min. The amino acid (*Premasol*) component was 4 g/kg/d in order to assist postoperative healing.

The patient received parenteral nutrition 22 h of the day. Intravenous fluids were infused at 2 ml/h for the remaining 2 h to maintain central line patency. Initially the patient was placed on half normal saline with 0.5 unit heparin during that time, however due to hyponatremia, this was transitioned to normal saline with 0.5 unit heparin.

The rate of TPN infusion was tapered in order to prevent a sudden decrease in the patient's glycemic status. At 4 p.m. daily, the TPN was initiated at ¼ goal rate and increased to ½ goal rate 1 h later. From 6 p.m. to 12 p.m. the following day, the TPN was infused at goal rate. From 12 to 1 p.m. it was then decreased to ½ goal rate again, and then decreased to ¼ goal rate the following hour. From 2 to 4 pm the TPN was discontinued (Fig. 1). Rates were calculated based on the total volume of the daily TPN created.

The electrolytes were closely monitored and adjusted accordingly. A basic metabolic profile, liver function tests, magnesium and phosphorus levels were obtained at the minimum biweekly. If significant adjustments were made, additional laboratory values were obtained. The most significant perturbation was the chloride:acetate balance. While clinically obstructed, the patient's high gastric output required elevated chloride concentrations (up to 11 mEq(milliequivalents)/kg/d) with minimal acetate to counteract electrolyte loss. The sodium concentration was also increased (10 mEq/kg/d). With the return of bowel function following surgery, resultant decrease in gastric output, and initiation of enteral feeds, these concentrations were decreased to normal range. Chloride to acetate ratio was then maintained at 2:1.

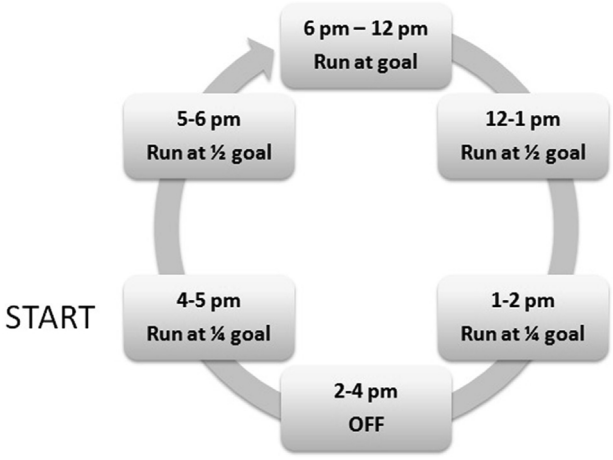

Fig. 1. Cycled TPN regimen.

The patient's weight fluctuated as expected during perioperative periods. However, he demonstrated consistent growth remaining just under the 3rd percentile (Fig. 2).

The patient only had two episodes of hypoglycemia (glucose level <50 mg/dL). These episodes were during acute illness secondary to a proximal intestinal obstruction. The patient was clinically asymptomatic at the time of phlebotomy.

Following the transition to cycled TPN, the patient's direct bilirubin began to decrease and returned to normal limits. His direct bilirubin improved from 3.4 mg/(deciliter)dL to 0.1 mg/dL prior to discharge. The direct hyperbilirubinemia had decreased below 1.0 mg/dL within 3 weeks (Fig. 3). He did not require medical therapy with ursodiol or phenobarbital.

2. Discussion

Parenteral nutrition-associated liver disease ranges from temporal cholestasis to cirrhosis and even mortality. Cycling TPN has been implemented as one method to minimize liver damage in these patients [3]. The temporary cessation of amino acids and dextrose infusion has been theorized to improve substrate utilization and decrease lipogenesis within the liver [2,3].

Although prior data is limited, case reports similar to above, have prompted not only cyclic TPN as a therapeutic measure but also as prophylaxis. One retrospective review of prophylactic cycled TPN versus continuous TPN in gastroschisis patients found time to direct hyperbilirubinemia was significantly longer [4]. A separate review in surgical neonates comparing prophylactic cycled TPN versus therapeutic cycled TPN also found a significantly lower incidence of elevated direct bilirubin. Of note, these patients did not demonstrate an increase in hypoglycemia or hypertriglyceridemia [2].

In complicated gastroschisis, TPN is the only source of nutrition and may be required for extended periods of time. Moreover, in complicated gastroschisis with short bowel syndrome, long term TPN will need to be utilized to maximize caloric requirements. This is paramount as a longer TPN course and elevated dextrose infusions are independent risk factors for TPN cholestasis [5]. Given the inherent dysmotility which occurs in gastroschisis, only compounded by short bowel syndrome, we sought to maximize caloric intake while trying to minimize the toxicity of TPN. We opted to cycle TPN in hopes of minimizing or even reversing the hepatotoxicity while the intestine was allowed to heal from operative interventions. As demonstrated by the return of direct bilirubin to normal range and continued growth, this strategy was not only effective in protecting the liver but was also safely performed in this neonate. Of note, the rehabilitation of the distal intestine via the mucous fistula also appeared to allow better post-operative intestinal function, and thus, shorter time to full enteral nutrition.

Despite these early successes, cycled TPN has not experienced widespread acceptance. Further research, particularly in neonates, will provide the basis for future implementation.