possibility of lung or heart and lung transplants in adequately selected cases. The selection of candidates for the transplant programme is crucial for their long term survival, and should include careful monitoring of renal function and signs of renal damage, particularly proteinuria. The patients now admitted to transplant programmes are generally in advanced stages of the disease when secondary complications are more probable. It is very important to exclude the presence of amyloidosis in these subjects. In our opinion, even the mild and occasional proteinuria that is sometimes observed in cystic fibrosis children merits particular attention, with the aim of promptly identifying patients at risk of progressively severe renal impairment and of

prescribing appropriate treatments to prevent or at least delay severe clinical manifestations.

- 1 Abramowsky CR, Swinehart GL. The nephropathy of cystic fibrosis: a human model of chronic nephrotoxicity. *Hum Pathol* 1982;13:934-9.
- 2 Katz SM, Krueger LJ, Falkner B. Microscopic nephrocalcinosis in cystic fibrosis. *N Engl J Med* 1988;319:263-6.
- 3 Ristow SC, Condemi JJ, Stuart ID, Schwartz RH, Bryson MF. Systemic amyloidosis in cystic fibrosis. *Am J Dis Child* 1977;131:886-8.
- 4 Canciani M, Pederzini F, Mastella G, Boccato P. Systemic amyloidosis in cystic fibrosis. *Acta Paediatr Scand* 1985;74: 613-4.
- 5 Rifai A, Millard K. Glomerular deposition of immune complexes prepared with monomeric or polymeric IgA. *Clin Exp Immunol* 1985;60:363-8.
- 6 Coppo R, Basolo B, Martina G, et al. Circulating immune complexes containing IgA, IgG and IgM in patients with primary IgA and with Henoch-Schönlein nephritis: correlation with clinical and histologic signs of activity. *Clin Nephrol* 1982;18:230-9.

Nutritional manipulation in the management of dumping syndrome

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Abstract

Two children with Nissen's fundoplication and either gastrocystoplasty or pyloroplasty developed dumping syndrome. Correction of their blood glucose abnormalities, resolution of symptoms, and weight gain were effectively achieved by addition of fats and uncooked corn starch (50 g/l) to their feeds.

There is a paucity of literature regarding the prevalence and optimal management of dumping syndrome in children. This results from the rapid gastric emptying of a carbohydrate meal, causing hyperglycaemia followed by reactive, symptomatic hypoglycaemia. Dumping syndrome in children has been described almost exclusively as a postoperative complication of Nissen's fundoplication.¹⁻³ Several therapeutic approaches have been tried with mixed results: for example, continuous nasogastric or gastrostomy feeds, frequent small amounts of thickened feeds and the addition of complex carbohydrates such as fibre and uncooked corn starch.^{4,5} We report on two children treated by a combination of complex carbohydrate and fats to delay gastric emptying and provide a stable release of glucose, leading to normoglycaemia and amelioration of symptoms.

Case reports

CASE 1

At 10 months of age this girl underwent bladder augmentation using gastric mucosa (that is, gastrocystoplasty) for treatment of reflux nephropathy. At 15 months of age she had further bladder augmentation with ileocystoplasty and an antireflux procedure (Nissen's fundoplication) for clinically and radiologically significant gastro-oesophageal reflux. At 17

months (two months after surgery) she was readmitted for evaluation of persisting diarrhoea and food aversion. On specific questioning her parents reported that she also had acute abdominal discomfort and lethargy after meals, accompanied by pallor and sweating.

In hospital, a brief tonic-clonic seizure occurred postprandially. At that time she was found to be hypoglycaemic (1.5 mmol/l) and required correction with a glucose infusion. No other metabolic abnormality was detected. A computed tomographic scan of the brain and an electroencephalogram were both normal. Subsequently, in the hospital, an oral glucose tolerance test (2 g/kg glucose) was performed which showed an initial hyperglycaemic response (blood glucose concentration, 7.8 mmol/l at 30 min) followed by hypoglycaemia (blood glucose 3.5 and 2.1 mmol/l at 45 and 60 min, respectively). Barium swallow showed a normal oesophagus, microgastria, and rapid stomach emptying. The latter was confirmed by a gastric emptying scan with ^{99m}Tc-labelled sulphur colloid which showed no gastric residue at 30 minutes. A diagnosis of dumping syndrome was made and she was placed on frequent small feeds and the addition of corn oil. This alleviated the postprandial glucose abnormalities totally and clinical symptoms partially.

Two months later she was readmitted for continued symptoms relating to the dumping syndrome, including irritability, worsening food aversion, and slow weight gain. She was stabilised with continuous nasogastric feeding (Similac PM 60/40, Ross Laboratories). In an attempt to wean her to bolus feeds during the daytime hours, a safflower oil based fat emulsion (Microlipid 50%, Sherwood Medical Industries) was added to her formula feeds to delay gastric emptying but without any success. Subsequently, uncooked corn starch was added to her

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formula feeds at a concentration of 50 g/l. Symptoms improved markedly and her blood glucose concentration remained stable throughout the interval between feeds (4.3–5.6 mmol/l). Gradually, nasogastric feedings were changed to three bolus feeds during the day and a 10 hours' overnight continuous infusion. On this regimen her mood improved dramatically, weight increased by 2100 g over the next 140 days, and she began to take small amounts of food by mouth. She is now 23 months old.

CASE 2

This 4 year old boy had a history of accidental ingestion of caustic drain cleaner and subsequent development of strictures in the oesophagus, antrum, glottis, and supraglottis. He underwent a tracheotomy and subsequently a feeding gastrostomy, Nissen's fundoplication, and pyloroplasty. Six months after the operation his mother reported that he was experiencing discomfort during and after gastrostomy bolus feeds. After a clinical diagnosis of delayed gastric emptying he was prescribed metoclopramide. His symptoms worsened considerably and he was admitted to hospital.

He was found to have episodes of tachycardia, sweating, and pallor immediately after feeds. His symptoms were worse after the daytime bolus feeds and he had, by himself, adopted a left lateral position during and after feeds to experience some relief of symptoms. He was clinically diagnosed to have 'intolerance' to feeds and his feeds were diluted to 0.75 strength Isosource (Sandoz Canada), an iso-osmolar, 4.18 kJ (1 kcal)/ml, casein-soy based tube feeding formula. Additional energy was supplied by medium chain triglycerides (MCT Oil, Mead Johnson). This dietary manipulation provided some relief of symptoms.

At this stage he was clinically diagnosed to have dumping syndrome which was confirmed as in the first case. His blood glucose concentration at 30 minutes after a 2 g/kg glucose load was 10.0 mmol/l, which dropped to 2.0 mmol/l at 90 min with marked symptoms. His feeds were changed to 940 ml of Isosource with 90 ml 50% Microlipid, 40 g uncooked corn starch, and 440 ml water, which provided 334.4 kJ/kg/day and 1.8 g/kg/day of protein. The feeds were initially given as three bolus feeds during the day with 12 hour continuous drip at night and subsequently changed to 2 daytime bolus feeds of 375 ml each and nine hour continuous feed at night. This was accompanied by a considerable improvement in postprandial blood glucose and symptoms. The amount of corn starch was increased to 70 g in the feeds (about 50 g/l) which resulted in further resolution of symptoms.

Discussion

These two cases illustrate the occurrence of dumping syndrome after two different gastrointestinal surgical procedures, along with Nissen's fundoplication: that is, gastrocystoplasty and pyloroplasty. Both the patients presented with considerable symptoms and failure to thrive, a major component of which was learned aversion to food as in the study by Hirsig *et al.*² In both cases, effective control of symptoms and nutritional rehabilitation was achieved by dietary manipulations alone. Nutritional management was designed in accordance with the underlying altered physiology. Fats were used to delay gastric emptying and uncooked corn starch was used to deliver small amounts of glucose at a steady rate over a relatively longer time period. The latter has been used previously in the management of hypoglycaemia associated with type I glycogen storage disease.⁶

Microlipid was chosen as the fat source for several reasons. Firstly, it is a commercially available fat emulsion that is readily miscible in a liquid phase and thus it is appropriate for use in tube feeding. Secondly, as Microlipid is composed of long chain triglycerides, it will delay the gastric emptying more than medium chain triglycerides. Microlipid also provides a nutritionally balanced fat source with essential fatty acids. The choice of uncooked corn starch was based on several factors: it produces a slow and sustained blood glucose concentration; other commonly used and commercially available glucose polymers (for example, Polycose, Ross Laboratories) have a shorter chain length and produce a sharper and less sustained blood glucose response; corn starch can be used as a bolus and it eliminates the need to use continuous feeding; and finally it is easily available and inexpensive.

The management of dumping syndrome should be aimed at resolving clinical symptoms and improving nutrition simultaneously. This can be effectively achieved by the methods outlined.

- 1 Meyer S, Deckelbaum RJ, Lax E, *et al.* Infant dumping syndrome after gastroesophageal surgery. *J Pediatr* 1981; **99**:235–7.
- 2 Hirsig J, Baals H, Tuchschnid P, Spitz L, Stauffer UG. Dumping syndrome following Nissen's fundoplication: a cause for refusal to feed. *J Pediatr Surg* 1984; **19**:155–7.
- 3 Caulfield ME, Wyllie R, Firor HV, Michener W. Dumping syndrome in children. *J Pediatr* 1987; **110**:212–5.
- 4 Gitzelmann R, Hirsig J. Infant dumping syndrome: reversal of symptoms by feeding uncooked corn starch. *Eur J Pediatr* 1986; **145**:504–6.
- 5 Kneepkens CMF, Fernandes J, Vonk RJ. Dumping syndrome in children. *Acta Paediatr Scand* 1988; **77**:279–86.
- 6 Chen YT, Cornblath M, Sidbury JB. Cornstarch therapy in type I glycogen-storage disease. *N Engl J Med* 1984; **310**: 171–5.