Hyperphosphataemia after enemas in childhood: prevention and treatment

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Abstract
The case of a child with severe hyperphosphataemia and symptomatic hypocalcaemia secondary to retention of phosphate administered through an antegrade enema is reported. Caution should be exercised with the use of phosphate enemas and prompt action taken to remedy retention. The use of glucose with insulin in the emergency management of acute hyperphosphataemia is discussed.

(Arch Dis Child 1993;68:233–4)

Severe toxicity after the use of magnesium¹ and phosphate,² enemas administered by the rectal route has previously been described. We wish to report toxicity following the administration of phosphate enemas by an antegrade route.

Case report
A girl aged 4·2 years with severe global developmental delay of unknown aetiology was transferred to the regional paediatric surgical unit with probable intestinal obstruction. She had a long history of constipation resistant to management with laxatives and retrograde enemas and had required several manual evacuations under general anaesthesia.

At the age of 2·8 years the girl had had a non-refluxing appendicocoeacoostomy formed to allow the administration of enemas via the caecum to promote colonic emptying.³ After this procedure her constipation had been successfully managed by the instillation of half a phosphate enema (Fletchers' Phosphate Enema, Pharmax UK, containing sodium acid phosphate 12·8 g and sodium phosphate 10·24 g to a total 128 ml aqueous solution) three times a week by her parents.

She was admitted after a three day period when two and a half enemas had been given with no result. She had vomited three times in the 12 hours before admission, become increasingly drowsy, and passed little urine. On admission she appeared unwell, pale, and was poorly perfused. She was apyrexial, with a pulse rate of 140/minute, blood pressure 85/70, and core–peripheral temperature difference 7·8°C. Abdominal examination revealed distension and generalised tenderness; bowel sounds were reduced.

Radiography revealed gross constipation together with a dilated loop of small bowel containing fluid levels. Some 600 ml of faecal fluid was drained via a catheter inserted into the appendicocoeacoostomy, but the bowel signs persisted and urgent laparotomy was arranged.

Her admission electrolytes showed gross imbalance with plasma sodium concentration 159 mmol/l, potassium 4·3 mmol/l, bicarbonate 9·1 mmol/l, urea 23·0 mmol/l, creatinine 114 µmol/l, calcium 1·09 mmol/l (reference range 2·15–2·55), and phosphate 4·76 mmol/l (reference range 1·29–1·78).

Resuscitation was commenced with human albumin solution followed by sodium chloride and dextrose. A 2:1 heart block developed that was attributed to the low plasma calcium. Despite the known risk of precipitating calcium phosphate in the tissues, a bolus of 10 ml of 10% calcium gluconate was given and repeated 90 minutes later. Plasma calcium concentration rose to 1·42 mmol/l and normal cardiac rhythm returned.

At laparotomy a loop of colon was found to be obstructed and partially twisted behind the free edge of the mesentery. The bowel was released and found to be viable. Further faecal fluid was evacuated from small and large bowel and a manual evacuation performed.

Postoperatively her plasma calcium concentration was 1·22 mmol/l, phosphate 3·58 mmol/l, urea 21·8 mmol/l, and creatinine has risen to 152 µmol/l although she was beginning to pass some urine. Plasma sodium concentration was 156 mmol/l and potassium 3·0 mmol/l. Administration of further calcium gluconate was considered hazardous and it was felt that dialysis would be necessary if a good urinary flow was not established within a few hours. As an interim measure, 10% glucose was given intravenously in addition to maintenance fluids in order to promote passage of phosphate into the cells. Insulin was not given in view of the hypokalaemia. The patient’s clinical condition improved and the plasma concentrations of calcium and phosphate returned to normal over the next 48 hours.

Discussion
Two factors contributed to the acute metabolic problems seen in this case. Firstly, a large load of sodium and phosphate was delivered into the colon and retained for longer than 24 hours. Because of the presence of intestinal obstruction, enema fluid pooled in the proximal colon and refluxed through the ileocaecal valve into the small bowel, allowing ready absorption of the salts and causing loss of fluid into the bowel. Secondly, the fluid loss led to prerenal failure and as a result the renal excretion of phosphate was negligible.

Hazards of phosphate enemas administered rectally are recognised and include hypocalcaemia and hyperphosphataemia,⁴ hypokalaemia,⁵ tetany,⁶ rectal necrosis,⁷ perforation,⁸ and death.⁹ This is the first case to be reported in

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Accepted 27 August 1992
which an antegrade enema containing phosphate has led to complications, and it highlights a possible enhanced risk as enemas are introduced close to the ileocaecal valve.

The data sheet for Fletchers’ Phosphate Enema suggests that phosphate enemas should not be used in children under 3 years, and it has also been suggested that they be used with caution in older children. We would endorse this advice and feel that it is essential that all phosphate containing enemas are given under strict supervision whether given by the rectal or antegrade route. In the event of retention of the enema, it is essential that urgent steps are taken to recover the phosphate rich fluid. There is no clear indication as to how quickly toxicity may develop after retention but we now advise parents to come to hospital if there has been no result after three hours and under no circumstances to repeat the enema.

Acute hyperphosphataemia occurs rarely in childhood and guidelines for treatment are not readily accessible. It occurs most often during tumour lysis when it is accompanied by hyperkalaemia. Treatment with insulin and glucose to reduce the potassium is accompanied by a simultaneous fall in phosphate as cellular uptake of phosphate is increased. This treatment will produce a temporary reduction in plasma phosphate concentration but as phosphate excretion is almost entirely via the kidneys, the only effective treatment for acute hyperphosphataemia in the face of persisting renal failure is dialysis.

Although infusion of calcium is recommended in many texts and case reports this may result in precipitation of calcium phosphate in the tissues. We would suggest that such treatment should be reserved for cases accompanied by symptoms of hypocalcaemia such as convulsions or cardiac arrhythmias.

Commentary
The antegrade colonic enema (ACE)/Malone procedure is a novel approach to the management of faecal incontinence and intractable constipation for patients who would otherwise need a colostomy or require regular rectal washouts. By providing a continent non-refluxing catheterizable channel into the caecum, antegrade colonic enemas can be easily administered. The original procedure consisted of a reversed antireflux appendicostomy. Catheterisation of the stoma can be performed by children from the age of 5 years and it is particularly advantageous in patients with spina bifida who are physically disabled and would otherwise require alternative forms of assisted bowel evacuation. The other major group of patients for whom this procedure has been extremely beneficial are children with high anorectal malformations with incontinence after reconstructive procedures.

The social acceptability of the stoma has been impressive. The washout procedure comprises instilling half (64 ml) or the whole (128 ml) of a disposable phosphate enema (Fletchers’ Phosphate Enema) into the caecum via a No 10 French gauge catheter. The enema may be diluted 1:1 followed by 100–200 ml of normal saline and it should be evacuated within 20–30 minutes. After the instillation of the phosphate enema the child sits on the toilet until evacuation is complete. A few of the children leak a small amount of fluid by rectum for a short period after the evacuation. For this reason the parents may prefer the procedure to be performed in the evening. The child is then completely clean for the next 24 hours and experiences only minor soiling on the second day. The enemas are generally repeated every 48–72 hours. A second enema should not be administered during the same washout and if the enema has still not evacuated after saline irrigation medical advice should be requested. This practice will avoid the complications of phosphate overdose reported in the above paper.

The improved quality of life in formerly incontinent children after the introduction of the ACE procedure has been remarkable. The children are able to go swimming and partake in normal childhood activities and have all demonstrated a remarkable increase in self confidence and self esteem.

Despite the warning in the current paper, the judicious and carefully monitored use of phosphate is probably the most satisfactory current enema for use with the ACE procedure, but attempts should be made to establish the minimum amount required in an individual child to initiate evacuation, even if this means using a greater volume of saline and possibly taking a longer time over the enema. The use of alternative measures, such as including an oral stimulant laxative, should possibly be investigated in an attempt to reduce further the risk of phosphate overdose.