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CLINICAL CASE

Severe tetany due to phosphate enemas use, case report

Tetania severa por uso de enemas fosfatados, reporte de caso

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Abstract

Background: Phosphate enemas are frequently used in the treatment of constipation. Errors in dosage and administration can lead to severe complications. **Objective:** To report a case of severe toxicity of phosphate enemas in a child with no risk factors. **Case:** 2 years old female, with functional constipation, was brought to emergency department because abdominal pain. She was diagnosed with fecal impaction and received half a bottle of Fleet Adult® (Laboratorio Synthon, Chile) two times, with no clinical resolution, deciding to start proctoclisis in pediatric ward. Soon after admission, she presented painful tetany, but alert and oriented. Patient was transferred to PICU where severe hyperphosphatemia and secondary hypocalcemia were confirmed. Her treatment included electrolyte correction; removal of residual phosphate enema and hyperhydration. Tetany resolved over 2 hours after admission and no other complications. Proctoclisis was performed and patient was discharged three days after admission with pharmacological management of constipation. **Conclusion:** Phosphate enemas may cause serious complications in children with no risk factors. Errors in dosage, administration and removal of the enema are causes of toxicity in this group. Pediatricians and health personnel must be aware of risks and signs of toxicity of phosphate enema.

Keywords:
Phosphate enema;
constipation;
tetany;
hyperphosphemia;
hypocalcemia.

Introduction

Constipation is a frequent problem in pediatrics, with increasing incidence due to the rising number of children with special health needs, but also in children with no risk factors due to the Western lifestyle. Phosphate enemas are frequently used for the treatment of chronic constipation and fecalomas on primary care, hospitalized children and emergency services. Pediatric patients are at risk of severe toxicity, potentially lethal, from phosphate enemas due to the particularities of their presentation, posology and and administration. We report a case of severe toxicity due to phosphate enema adminstration in a preschooler with functional constipation without other risk factors.

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Clinical case

A 2 years and 7 months old female, with background history of chronic constipation without pharmacological treatment was taken by her mother to the emergency department for 48 hours of colicky abdominal pain, without vomiting, without fever. Her weight at the moment of admission was 15 kg. The mother reported absence of bowel movements in the last 72 hours. Her heart rate was 126 bpm, blood pressure 90/48 mm Hg, 96% saturation on room air. Physical exam revealed dry mucous membranes compatible with mild dehydration, without respiratory distress. Great abdominal distension and pain in the left side was noted. Plain abdominal X-ray film showed distension of the transverse segment of the colon, associated with abundant estercoraceous material in colon suggestive of fecaloma (figure 1).

Phosphate enema was indicated, receiving approximately 60 ml (half bottle) of Adult Fleet® (Synthon, Chile). In the absence of stools, the same dose was repeated 2 hours later. There was no record of elimination of the enema. Due to the failure of elimination of the impacted feces with phosphate enemas, she was admitted for proctoclisis management.

At 2 hours after admission to the pediatric ward, the patient presented painful hypertonia of the upper and lower extremities without impairment of consciousness. It was initially interpreted as a focal seizure and the patient received midazolam 0.1 mg/kg/dose ev, being transferred to PICU for further care. At PICU admission, she had tachycardia of 163 bpm and no respiratory distress, oxygen saturation 97% on room air.

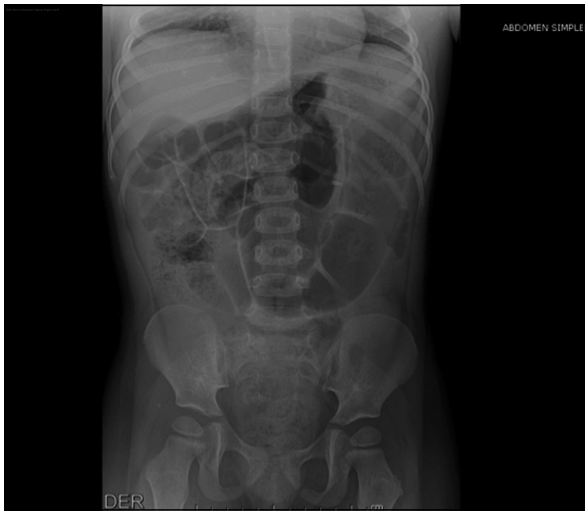

Figura 1. Plain abdominal X ray film showing enlargement of transverse colon associated to abundadnt fecal content on descendant colon, sigmoid and rectum. Findings in accordance to impacted fecaloma diagnosis.

It was noted that she had persistent hypertonic painful extremities, crying, but oriented and appropriate, with Glasgow Coma Scale 15 (Supplementary material, Video 1).

In agreement with the physical exam, the laboratory tests showed a severe electrolytic alteration with hyperphosphatemia, hypocalcemia and hypernatremia associated to mild metabolic acidosis (table 1, hour zero). Calcium suplementation with intravenous calcium gluconate 10% 50 mg/kg for 2 times, until hypertonia was terminated. In addition, she received intravenous maintenance fluids with 2.5% dextrose in ½ normal saline plus potassium chloride 2 g/l (calculating 1,800 ml/m^2/day) and 5 mg intravenous furosemide for forced diuresis, obtaining an urinary output of 6.4 cc/kg/h after. It did not require analgesia once the tetany had ceased. Given the recent use of phosphate enemas, emergency proctoclisis was performed with 0.9% SF 1,000 cc + 10 mL liquid vaseline for 6 h at 150 mL/h, achieving the fecaloma disimpactation. Subsequent electrolyte supplementation was performed with 10% calcium gluconate, 150 mg/kg/day, until normocalcemia was achieved.

There was a rapid clinical improvement following the management of hypocalcemia, resolving hypertonia at 2 hours of admission. There was no development of renal failure, hemodynamic instability or other secondary electrolyte disturbances. Table 1 shows the most important laboratory results during her PICU stay. He was discharged home 3 days after admission, with resolution of her fecaloma and maintenance pharmacological treatment for constipation.

Discussion

Constipation is a common problem in pediatrics, accounting for about 3% of general pediatric consultations and up to 25% of gastroenterological consultationss, with a prevalence of approximately 10%[1]. It is estimated that 1 out of 3 children with functional constipation present at least one episode of fecaloma throughout their life[2]. Fecal disimpactation can be performed with oral or enteral osmotic laxatives, osmotic enemas, lubricants and glycerin suppositories[3-5].

Among the multiple options for disimpactation of fecalomas, sodium phosphate enemas are frequently used at the emergency room[3]. As an example, in the pediatric emergency department of our center 885 phosphatic enemas are performed per year as part of the treatment of different pathologies. There is no specific report in the literature on the number of adverse events associated with the use of phosphate enemas, but there are numerous clinical cases reported.

Phosphate enemas are hyperosmolar compounds

Table 1. Main laboratory results after admission. All timepoints are relative to addmission time

	0 h	2 h	8 h	12 h	24 h
BUN (mg/dl)	16	15	14	7	4
Creatinine (mg/dl)	0.42	0.47	0.54	0.43	0.31
Creatinkinase total (U/l)	677	244	584	843	407
Plasma calcium (mg/dl)	4.2	4.8	5.8	6.8	9.9
Plasma phosphate (mg/dl)	37.9	17.2	6.3	3.8	3.1
Plasma magnesium (mg/dL)	1.7	1.5	1.8	1.8	2
Plasma sodium (mEq/l)	151	155	152	142	142
Plasma potasium (mEq/l)	3.6	3	2.3	2.5	4.7
Plasma chloride (mEq/l)	110	112	114	107	107
pH	7.27	7.33	7.41	7.40	
Carbon dioxide (mmHg)	33.1	30.9	30.4	28	
Plasma sodium bicarbonate (mmol/l)	15.2	16	18.9	17.1	
Base excess (mmol/l)	10.4	8.6	-4.4	6.5	

due to the high concentration of sodium and phosphorus (table 2). Thus, they produce a great movement of fluids towards the intestinal lumen, developing a third space that allows the emptying of the sigmoid-rectal fecal contents. Dehydration secondary to the intraluminal hyperosmolar effect of the solution and hypocalcemia secondary to absorption of high phosphorus content[5-7] are the main adverse effects of errors in dosing, administration and elimination of the enema. From the pathophysiological point of view, it is important to note that phosphorus is generally absorbed in duodenum and jejunum, but colonic absorption is possible secondary to high concentrations in the rectum[8]. A rapid increase in phosphorus produces acute hypocalcemia due to chelation of calcium and precipitation of calcium phosphate salts in soft tissues and kidney. Other mechanisms, such as the decrease in the production of 1,25-dihydroxycholecalciferol and the reduction of the absorption of calcium at the intestinal level, are slower to establish and therefore play a secondary role in cases of intoxication[6,8,9], but may be important in cases of chronic exposure. In addition, excess phosphorus as inorganic acid and loss of bicarbonate in the intestinal lumen produce metabolic acidosis[6].

Phosphate enemas are more frequent in pediatrics, especially in children younger than 5 years and with risk factors such as kidney failure (decreased phosphorus clearance), altered bowel motility (paralytic ileus, Hirschsprung's disease, myelomeningocele , Colostomy), but is exceptional in children with no underlying pathologies. In the latter group the error in the posology is the most frequent cause[5-7,10].

Table 2. Composition of Pediatric and adult presentation of Fleet® (Laboratorio Synthon, Chile), per bottle

Fleet enema® (1 bottle)	Adult	Pediatric
Volumen	118 ml	59 ml
Sodium phosphate monobasic	19 g	9.5 g
Sodium phosphate monobasic	7 g	3.5 g
Sodium content	4.4 g	2.2 g

The symptoms of intoxication by phosphate enemas are very diverse, ranging from mild digestive symptoms to cardiorespiratory compromise with lethal outcome. The most frequent symptoms are abdominal bloating, vomiting, explosive or bloody stools, hypernatremic dehydration and hypocalcemia associated arrhythmias such as QT prolongation. In severe cases, neurological symptoms related to severe hypocalcemia, such as irritability, paresthesias, tetany, laryngospasm, progressive alteration of consciousness, coma and hyperthermia due to hypothalamic dysfunction may appear.

These symptoms may occur suddenly between 30 min and 4 hours after administration of the enema[5-7].

In the reported case, the main risk factor for intoxication by phosphate enemas was age. The indication of an approximate total volume of 118 ml of adult Fleet® phosphate enema triggered a clinical picture of tetany secondary to hypocalcemia. It is important to emphasize that the absorption of phosphorus may have been

favoured by the permanence of the solution in the rectal ampulla. Every physician who takes cares of children, and specially pediatricians, should be aware of the risks associated with the use of these drugs. In addition to checking the appropriate dose, the physician and/or health personnel should verify the elimination of the enema, even if the effective removal of the fecaloma is not achieved.

The manufacturer's recommendation for use in children between 2-5 years is the administration of 1/2 bottle of pediatric Fleet®. A full Fleet® bottle can be administered only in children older than 5 years of age. The manufacturer and the literature do not recommend the use of ohsphate enemas in children younger than 2 years of age, due to an increased intestinal absorption[7]. There are no absolute contraindications, but their use should be restricted in patients at risk and patients under 2 years of age, and it is not recommended the administration by parents or caregivers as a home medication[7]. In this group of patients an alternative is the use of glycerol lubricants, such as Babylax® (Synthon, Chile), which has no phosphate and has no osmotic effect[11].

The formulation of pediatric phosphate enemas has a composition similar to that of adults, but the presentation (a bottle or package) has only half the volume. When the adult and pediatric presentation are similarly called, a frequent mistake is to indicate half-bottle or half-package, which can double the volume and amount of monobasic phosphate and dibasic phosphate the patient receives (table 2). The recommendation of the specialists is to indicate 2-5 ml/kg of the solution[11]. The correct use should be through a rectal probe and syringe, to have precision in the dose, especially in patients at risk and under 5 years . For these reasons, the FDA in August 2014 issued an alert regarding the use of phosphate enemas in the treatment of constipation, by its free marketing without prescription and possible serious adverse effects, potentially fatal[12].

The first step of the treatment of hyperphosphemia secondary to phosphate enemas is to remove phosphorus from the intestinal lumen (eg with water enemas)[13], a good renal function should be ensured to determine the next steps. With adequate renal function, hyperhydration and diuresis (phosphorus removal is mainly renal) should be performed[5,6]. The use of phosphorus binders to promote digestive elimination, such as calcium carbonate or calcium acetate, can be performed enterally to reduce phosphorus reabsorption from the intestinal lumen[6] and calcium[7] should be provided in case of symptomatic hypocalcemia.

Calcium replacement should be careful, seeking to alleviate symptoms (cramps, convulsions, arrhythmias), and not seeking to correct serum calcium values to normal limits, since in the presence of hyperphosphemia it can cause renal precipatation of calcium salts[8].

In case of renal failure, any exogenous adminstrstion of phsphrus should be discontinued and renal replacement therapy should be considered, in cases of refractoriness with usual management or persistent oliguria or anuria.

Conclusion

The use of phosphate enemas can produce severe, potentially lethal toxicity in children without risk factors. In this group of children an error in the posology is the main cause of the toxicity. Symptoms of hypocalcemia, such as tetany secondary to hyperphosphemia, require aggressive emergency treatment to avoid associated complications. All physicians and health personnel who attend children should know the dosage, correct administration and verify the elimination of the drug, as well as early recognition of the symptoms and signs of phosphatic enemas intoxication.

Ethical Responsibilities

Human Beings and animals protection: Disclosure the authors state that the procedures were followed according to the Declaration of Helsinki and the World Medical Association regarding human experimentation developed for the medical community.

Data confidentiality: The authors state that they have followed the protocols of their Center and Local regulations on the publication of patient data.

Rights to privacy and informed consent: The authors have obtained the informed consent of the patients and/or subjects referred to in the article. This document is in the possession of the correspondence author.

Conflicts of Interest

Authors state that any conflict of interest exists regards the present study.

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