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Correcting hypophosphataemia in a paediatric patient with Sanjad–Sakati syndrome through a single oral dose of potassium phosphate intravenous solution

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Abstract

Sanjad–Sakati syndrome is an autosomal recessive disorder that is quite common in Kuwait. Among a wide range of complications in Sanjad–Sakati syndrome patients is the vulnerability to infections and subsequent hypophosphataemia. Hypophosphataemia is a metabolic alteration that contributes to numerous consequences such as cardiac arrhythmia. Therefore, if hypophosphataemia is left unresolved, it may culminate in death. A 20-month-old boy of 2.5 kg body weight diagnosed with Sanjad–Sakati syndrome was initially admitted to the paediatric intensive care unit after recovering from COVID-19, and then shifted to the general ward. He was diagnosed with recurrent pneumonia and urinary tract infection. After 9 days, the patient showed severe hypophosphataemia with serum phosphate concentration reaching 0.33 mmol/L. Despite the availability of potassium phosphate intravenous solution, it was difficult to administer potassium phosphate intravenously because of the small body size and low body weight of the patient. Therefore, 0.6 mL potassium phosphate containing 2.4 mEq of potassium and 5.3 mEq of phosphate was administered through a nasogastric tube. The patient showed rapid response after a single dose through the nasogastric tube. Such an intervention in Sanjad–Sakati syndrome patients shows possible advantages of shifting drug administration from intravenous to oral route that includes a convenient route of administration, whether in the intensive care unit or in the general ward. Moreover, shifting drug administration from the intravenous to oral route overcomes the risk of cannula-induced infection and reduces nurses' workload.

Keywords

Sanjad–Sakati syndrome, hypophosphataemia, paediatrics

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Introduction

Sanjad–Sakati syndrome is an autosomal recessive disorder, originating mainly in the Middle East and specifically the Arabian Gulf countries, where it was first reported in 1988.[1] In Kuwait, the estimated prevalence of Sanjad–Sakati syndrome is 7–18 per 100,000 live births.[2] Sanjad–Sakati syndrome starts during early stages of embryogenesis and is characterised by intrauterine growth retardation. Children with Sanjad–Sakati syndrome present with hypocalcaemia tetany and seizures due to hypoparathyroidism at an early stage in their lives that is associated with hyperphosphataemia.[1,3] Furthermore, Sanjad–Sakati syndrome patients are prone to infections such as pneumonia due to their compromised immunity.[2,3] Therefore, despite the characteristic feature of hyperphosphataemia in Sanjad–Sakati syndrome patients, their vulnerability towards infection may lead to severe hypophosphataemia.[4] Hypophosphataemia is a metabolic alteration that contributes to a wide range of consequences such as suppressing the production of cellular energy adenosine triphosphate (ATP).[5] Physiological phosphate level ranges from 0.8 to 1.3 mmol/L. However, hypophosphataemia

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Table 1. Patient's electrolyte concentration upon admission.

Electrolyte	Concentration (mmol/L)	Normal range (mmol/L)
Phosphate	0.87	1–1.65
Magnesium	0.82	0.75–0.95
Calcium	2.19	2.21–2.61
Sodium	152	135–150
Potassium	3.3	3.5–5
Chloride	123	98–110

is defined as serum phosphate concentration below 0.8 mmol/L.[5] According to recent studies, the prevalence of hypophosphataemia in the paediatric intensive care unit (PICU) is approximately 50%.[6] Symptoms of hypophosphataemia are of a wide range and include cardiac arrhythmia, fatigue, irritability and reduced diaphragm contractility.[6] Such a wide range of symptoms renders hypophosphataemia less diagnosable and hence may lead to silent and progressive complications, which, if left unresolved, may culminate in death.[7]

Case report

Here, we report a 20-month-old boy of 2.5 kg body weight diagnosed with Sanjad–Sakati syndrome alongside other comorbidities and complications. These included epilepsy that is controlled through phenobarbitone syrup 5 mg twice daily (4 mg/kg/day), gastro-oesophageal reflux disease that is controlled through domperidone 1 mg twice daily (0.8 mg/kg/day) and omeprazole 5 mg once daily (2 mg/kg/day), and congenital heart disease (atrial septum defect, ventricular septum defect, and patent ductus arteriosus) that is managed with furosemide 2 mg twice a day (1.6 mg/kg/day).

The patient recovered from COVID-19 at the Jaber Hospital and then was transferred to Al-Adan-Kuwait Hospital, where he was initially admitted to the PICU because of recurrent pneumonia and urinary tract infection (UTI), which were treated through amikacin administered intramuscularly 37.5 mg once daily (15 mg/kg/day). The main challenge of controlling our patient's condition was in the inability to administer drugs through intravenous route due to his compromised body growth. Therefore, we administered injectable drugs through the intramuscular route and oral drugs through a nasogastric tube (NGT). Upon his admission to the PICU, the initial serum electrolyte concentrations were as shown in Table 1.

Based on the electrolyte levels at admission, and since potassium concentration was low, the patient was administered 2.5 mL of potassium chloride (1 M) containing 2.4 mEq potassium once daily by NGT. The patient was stable and hence was moved to the general ward within 24 h of admission. After 9 days, the patient showed severe hypophosphataemia with serum phosphate concentration reaching 0.33 mmol/L, while the concentrations of other electrolytes were normal, as shown in Table 2.

Table 2. Patient's electrolytes concentration 9 days after admission.

Electrolyte	Concentration (mmol/L)	Normal range (mmol/L)
Phosphate	0.33	1–1.65
Magnesium	0.93	0.75–0.95
Calcium	2.35	2.21–2.61
Sodium	132	135–150
Potassium	3.8	3.5–5
Chloride	89	98–110

Table 3. Patient's electrolytes 15 h after administering potassium phosphate.

Electrolyte	Concentration (mmol/L)	Normal range (mmol/L)
Phosphate	1.28	1–1.65
Magnesium	0.86	0.75–0.95
Calcium	2.08	2.21–2.61
Sodium	132	135–150
Potassium	5.5	3.5–5
Chloride	93	98–110

Accordingly, it was essential to correct phosphate levels. However, as mentioned, the main obstacle towards such a vital step was the inability to administer potassium phosphate intravenously. In discussing possible alternatives with a hospital pharmacist, he suggested administering potassium phosphate through the NGT route, rather than the designated intravenous route of administration. Such a suggestion was based on calculating the milliequivalents (mEqs) of potassium in potassium chloride (KCl) solution and calculating the volume of potassium phosphate based on mEq of potassium in potassium phosphate.

Since KCl (1 M) is of 2.4 mEq potassium, we aimed to provide potassium phosphate through delivering 2.4 mEq potassium. After detailed calculations, it was concluded that administering 0.6 mL from potassium phosphate sterile solution for injection that contains 170.3 mg/mL potassium and 285 mg/mL phosphate provides 2.4 mEq of potassium and 5.3 mEq of phosphate. Therefore, 0.6 mL of potassium phosphate sterile solution was administered through NGT to treat such a case of acute severe hypophosphataemia. 15 h after administering 0.6 mL potassium phosphate through NGT, serum phosphate concentration was normalised as it reached 1.28 mmol/L, while other serum electrolytes concentrations were preserved at normal levels as shown in Table 3.

Discussion

Sanjad–Sakati syndrome is a rare autosomal recessive disorder that exists in the Gulf area. Therapeutic strategies mainly focus on correcting electrolytes and treating infections.[1] Hypophosphataemia is a common complication of infection.

Although hypophosphataemia may be attributed to the use of furosemide or omeprazole, if not both, our patient is using both medicines chronically and has been admitted with normal phosphate concentration (Table 1). Moreover, a previous study revealed that the prevalence of hypophosphataemia in septic patients is approximately 80%.[4] The percentage of paediatric patients suffering from pneumonia-induced hypophosphataemia is approximately 45%, and children with UTI contribute to approximately 18%.[8] Since our patient was diagnosed with both pneumonia and UTI, the observed hypophosphataemia is likely attributed to the infections he was suffering from. Such a metabolic complication, if left untreated, may lead to further complications, including cardiac arrhythmia, which is considered a lethal prognosis for our patient, who is also diagnosed with congenital heart disease.

The restricted and limited ability to administer drugs intravenously is considered a major obstacle in managing infection-induced hypophosphataemia in Sanjad–Sakati syndrome. Such a pathological factor that limits the intravenous accessibility of medicines can be solved through changing the route of administration. Converting the route of administration is a challenging skill that requires careful calculations and considerations to provide the optimal benefit from the administered medicine. The challenges of converting the route of administration stem from the pharmacokinetic and pharmacodynamic differences. Thus, the oral route of administration exerts numerous barriers that contribute to reducing drug bioavailability, whereas these barriers are overcome through the intravenous route in which bioavailability is 100%. These barriers include the rate and percentage of absorption as well as intestinal and hepatic metabolism (first-pass effect).[9] The conversion from intravenous to the oral route is classified mainly into three categories: (1) sequential therapy, where a drug is replaced with its oral counterpart, such as administering esomeprazole (20–40 mg) orally instead of intravenously;[9] (2) switch therapy, where an intravenously administered drug is replaced with an orally administered drug that belongs to the same pharmacological class and is expected to exert similar therapeutic outcome, such as oral administration of cefuroxime (500 mg) twice daily rather than intravenous administration of ceftriaxone (1 g) once daily;[10] and (3) step-down therapy, through which an injectable drug is replaced with an oral agent that belongs to a different pharmacological class of different frequency, dose and the spectrum of activity. A common example of step-down therapy is the shifting from injectable heparin to orally administered warfarin.[11]

Here, we have shown an example of sequential therapy through the administration of potassium phosphate through NGT rather than intravenously. This approach provided rapid correction of serum phosphate concentration. Such a therapeutic intervention provides numerous advantages. Mainly, it reduces the risk of cannula-related infections. Thus, shifting the administration of potassium phosphate from the intravenous to oral route may provide additional benefit through reducing cannula-related infections, hence reducing the need for any additional use of antibiotics, especially in Sanjad–Sakati patients.[9] Moreover, switching the route of administration reduces the workload on nurses as shown in a previous study which revealed that an intravenous administration of antibiotics takes approximately 10 min and therefore, switching to oral route reduces the nurses' workload by approximately 350 h/year.[12] In addition, we have shown the importance of hospital pharmacists as part of a multidisciplinary team, providing important and vital recommendations to provide optimal healthcare service with the available therapeutic resources.[13]

Conclusion

Sanjad–Sakati syndrome patients are prone to recurrent infections, such as pneumonia and UTI, which contribute to severe hypophosphataemia. Correcting hypophosphataemia in those patients is challenging since intravenous cannulation is extremely difficult because of small body size and low body weight. Therefore, shifting the route of administration from intravenous to oral provides a prompt and essential solution for severe hypophosphataemia in Sanjad–Sakati syndrome patients.

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Author contributions

M.A.S. conducted patient supervision and drug administration. Y.A.S. provided drug option and calculations. M.A.S. and Y.A.S. wrote the manuscript.

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