Hypophosphataemia as an element of 'refeeding syndrome' – a complication of nutritional treatment in an extremely neglected 5-year-old girl with infantile cerebral palsy

Hipofosfatemia jako jeden z elementów *zespołu refeeding* – powikłanie leczenia żywieniowego u skrajnie zaniedbanej 5-letniej dziewczynki z mózgowym porażeniem dziecięcym

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Abstract

Extreme malnutrition in children, similarly as among adults, most often occurs during the course of chronic diseases, but it can also occur in cases of neglect. A 5-year-old girl was admitted due to symptoms of cachexia. Physical examination revealed the following abnormalities: features of cerebral tetraplegia, significant body mass deficiency (8 kg, 44.4%), atrophic subcutaneous and muscular tissue, and pale, dry mucosa of the oral cavity. Laboratory tests showed extreme anaemia in two consecutive assays (Hb 1.7 g/dl). Transfusion of erythrocyte concentrate was urgently performed. Considering the observed aversion to food, highly caloric and high-protein diet and partial parenteral nutrition with complete vitamin and microelement requirements were applied. Laboratory tests during 2-5 days showed decreased levels of phosphorus and calcium characteristic for 'refeeding syndrome', which indicated a long period of starvation. She was nourished only orally for 6 days. The girl gained weight. Gradually red blood cell parameters became normal. We present this case to point out the possibility of complications concerning phosphorus-calcium balance during nutritional treatment in a small child.

Streszczenie

Skrajne niedożywienie u dzieci podobnie jak u dorosłych występuje najczęściej w przebiegu chorób przewlekłych, ale również może zdarzyć się w przypadku zaniedbań. Dziewczynka, lat 5, została przyjęta do szpitala z powodu wyniszczenia. W badaniu przedmiotowym z odchyleń od stanu prawidłowego stwierdzono cechy porażenia mózgowego czterokończynowego, znaczny niedobór masy ciała (8 kg), zanikową tkankę podskórną i mięśniową oraz blade, podsychające śluzówki jamy ustnej. W wykonanych badaniach laboratoryjnych wykazano skrajną niedokrwistość w dwóch kolejnych badaniach (Hb – 1,7 g/dl). W trybie pilnym przetoczono koncentrat krwinek czerwonych. Zastosowano dietę bogatokaloryczną, bogatobiałkową oraz częściowe żywienie pozajelitowe z pełnym pokryciem zapotrzebowania witamin i mikroelementów. W 2.–5. dobie w badaniach laboratoryjnych obserwowano zmniejszone stężenia fosforu i wapnia charakterystyczne dla *zespołu refeeding*, co świadczyło o długim okresie głodzenia. Od 6. doby dziewczynka była żywiona wyłącznie doustnie, zwiększyła się jej masa ciała, stopniowo normalizowały się parametry czerwonokrwinkowe. Autorzy przedstawiają przypadek, aby zwrócić uwagę na możliwość powikłań gospodarki wapniowo-fosforanowej podczas leczenia żywieniowego u małego dziecka.

Introduction

Malnutrition among children is still one of the leading health problems in the modern world. Most often malnutrition is observed during chronic diseases, but it can also emerge as a result of insufficient supply of nutritional components. This problem mainly concerns developing countries. It is estimated that half of child deaths in the world are directly or indirectly caused by malnutrition [1, 2]. The largest number of malnourished or cachectic children come from Africa, South America and Asia [3].

There are three types of malnutrition: marasmus, kwashiorkor and mixed type. 'Marasmus' is a protein-calorie malnutrition and emerges as a result of prolonged, insufficient intake of all alimentary components. It is characterized by decrease below 60% of proper body mass, poorly indicated hypoalbuminaemia, height deficiency, decreased muscular force following diminished content of somatic-structural protein, lipoatrophy and immunity depletion for infectious factors. Anaemia often accompanies marasmus and mainly is a result of iron and/or vitamin B_{12} deficiency in the diet. The term 'kwashiorkor' defines protein malnutrition and proceeds with significant hypoalbuminaemia, disturbances concerning water and electrolyte balance and also with swellings that conceal significant malnutrition. Body mass of malnourished patients is 60-80% of proper body mass [1, 3, 4]. Mixed type of malnutrition can appear in the case of significant hypercatabolism with protein deficiency. Mixed type of malnutrition contains features of both marasmus and kwashiorkor. Assessment of nutritional status is performed to determine malabsorption grade, among other things using anthropometric and biochemical examinations [1, 4].

The clinical picture of malabsorption depends on the ability of the child to adapt to a deficient diet. The point is that anabolic processes are limited within the field of synthesis of substances with minor significance for life and catabolic processes are intensified to obtain structural and energetic substrates from endogenous sources to support the most important life functions [1]. Increased glycolysis, gluconeogenesis, lipolysis, ketogenesis and deceleration of glucose metabolism in the liver of about 50% of cases occur during the first period of malabsorption. Increased catecholamine production happens as a result of progressive destabilization of the organism caused by hunger stress. Catecholamines increase lipolysis and cause hyperglycaemia. The next stage is decrease of basal metabolism by about 30%, limitation of insulin secretion, but also immune system dysfunction as a result of lymphatic tissue atrophy. Consequences of malabsorption are particularly threatening for children, because introduction of nutritional treatment too late can irreversibly disturb their growth and development. The main manifestation of long-term alimentary deficiencies in children is disorder of somatic development: body mass deficiency and growth inhibition. Insufficient intake of alimentary components in relation to requirements of the developing child also causes disturbances of body metabolism [5, 6].

Nutritional treatment is a long-term process in this situation. It consists in providing proper intake of all alimentary components by the digestive tract and/or by parenteral nutrition to normalize disturbed metabolic processes, to compensate deficiencies of nutritional status and to preserve further proper development. Realimentation management causes an energy expenditure increase of about 50%, but the requirement resulting from tissue restitution and growth increases as much as tenfold. Energy expenditure and body composition normalize not before three months. Properly balanced feeding prevents the complications that can result from intensive feeding of a cachectic patient [5, 7].

Metabolic disorders in cachectic children resulting from long-term starvation, but also from intensive feeding, can lead to death. During realimentation, both enteral and parenteral, the most dangerous complication is 'refeeding syndrome'. This syndrome is caused by insufficient adaptive mechanisms in response to increased food intake. Conversion from catabolism that occurs during starvation and in which energy originates from fat metabolism, to anabolism caused by beginning of patient feeding, initially leads to hyperglycaemia that causes increase of insulin secretion. Insulin activity results in transfer of phosphorus and other macro- and microelements from the extracellular to the intracellular compartment. Phosphorus is used intracellularly during formation of molecules of adenosine triphosphate (ATP), high-energy compounds that in a great amount originate during rapid metabolism increase that is a result of increased carbohydrate intake [6]. Also extended during realimentation, intake of protein, which is a main structural substrate, causes additional utilization of phosphorus and other mineral compounds (particularly calcium, magnesium, potassium) and also vitamins (particularly vitamin B_1, involved in the Krebs cycle) during the process of tissue restitution, phospholipid synthesis of newly formed cells and during phosphorylation of intermediate metabolic products for

protein and glycogen synthesis. As a result of decreased level of serum inorganic phosphorus (so-called 'refeeding syndrome') ATP amount decreases and the process of cellular respiration becomes disturbed [6, 8]. The following symptoms occur in consequence: disturbances of the cardiovascular system, initially tachycardia, subsequently bradycardia, conduction blocks, decrease of heart ejection volume and heart failure [9]. Neurological disturbances observed in this syndrome most often appear in the form of paraesthesias, seizures and coma. Massive rhabdomyolysis, embolisms and systemic insufficiency, mainly renal insufficiency, can appear as a result of muscle hypoxia [9].

Case report

The 5-year-old girl had young healthy parents, and was born after supported gravidity IV, premature labour III (27 hbd), after caesarean section due to threatening fetal asphyxia. Her body mass was 1070 g and she was graded 1 on the Apgar scale. The girl has previously been hospitalized once during the neonatal and infantile period due to prematurity, respiratory failure, pneumonia, patent ductus arteriosus, intraventricular haemorrhage, anaemia, and retardation of psychomotor development. The patient's history related by the mother revealed that the girl was fed mainly with milk and jelly. The girl was admitted to the Department of Paediatrics, Allergology and Gastroenterology for social reasons and extreme neglect, brought by the ambulance service after police intervention. On admission the girl's general state was severe. The child was pale, emaciated, apathetic. Physical examination revealed the following abnormalities: features of tetraplegic cerebral paralysis, significant body mass deficiency that was 44.4% of body mass proper for the age (the girl weighed 8 kg) (Table I). Many scratches were found on the skin of the abdomen and the back, scar on the skin of the chest after the operation of closing the patent ductus arteriosus, and also small, 0.5-1.5 cm in diameter, round scars near the navel and the spine, probably due to cured bedsores and procedures during the neonatal period. The child's behaviour apart from many scratches and scars did not indicate abuse. Moreover, physical examination showed pale, dry mucosa of the oral cavity and the throat. Laboratory tests revealed extreme anaemia confirmed by repeated measurement (Hb 1.7 g/dl, HCT 8.3%, RBC 1.49 M/μl, MCV 55.7 fl, MCH 11.4 pg, MCHC 20.5 g/dl), leucocytosis (13.4-14.9 K/μl), increased platelet level (506 K/μl), indeterminate serum iron level (< 6.0 μg/dl), and insignificantly decreased level of total protein (5.9 g/dl). Emergency transfusion of 160 ml of erythrocyte concentrate and 4 g of human albumins was performed. During the first day of hospitalization the child presented aversion to food, so appetite-stimulating drugs were applied. Parenteral feeding was introduced due to an unsatisfactory amount of food taken enterally. Initially, during the first and the second day of parenteral feeding, 29 kcal/kg/day were administered, including 0.9 g/kg/day of amino acids, 2.8 g/kg/day of glucose, 1.6 g/kg/day of lipids, and also the complete requirements concerning vitamins and microelements, calcium in the amount of 0.16 mmol/kg/day, phosphorus 0.12 mmol/kg/day, potassium 0.47 mmol/kg/day. She received about 2 mmol/kg/day of phosphorus orally. Decreased level of phosphorus (0.60 mmol/l) and calcium (2.15 mmol/l) was noted during the third day, but levels of potassium and magnesium were normal. Then basic enteral feeding, considering improved appetite, contained 2.6 mmol/kg/day of phosphorus and additionally 0.59 mmol/kg/day of phosphorus from added BMF preparation with considerable contents of macroelements. Intravenous intake of phosphorus was increased up to 0.47 mmol/kg/day and calcium up to 2.15 mmol/kg/day. Despite application of significantly higher amounts of phosphates, ten times higher than the value of the basic requirement, further decrease of serum phosphorus level (0.59 mmol/l) was recorded in laboratory tests during the fourth day of hospitalization, and also persistent low calcium level (2.15 mmol/l), while normal values of potassium and magnesium were found. Normal levels of phosphorus (0.94 mmol/l) and calcium (2.23 mmol/l) have been observed since the sixth day and gradually this fact allowed us to reduce intravenous intake of phosphorus and calcium and supplementation by food intensifier (BMF). Further increased levels of these macroelements were obtained during the seventh day (Table II). Moreover, during hospitalization,

Table I. Girl's nutritional status on admission
Tabela I. *Stan odżywienia dziewczynki przy przyjęciu*

	Height [cm]	Body mass [kg]	BMI [kg/m²]	Cole index [%]	Body mass coefficient (BMC)	Procent of standard body mass
Value	82	8.6	10.3	81	8.42	45
Centile	< 3 c	< 3 c	< 3 c		3 c	

Table II. Nutritive and mineral components ingested by patient and results of laboratory tests
Tabela II. *Składniki odżywcze i mineralne przyjmowane przez pacjentkę oraz wyniki badań laboratoryjnych*

		Norms	1st day	2nd day	3rd day	4th day	5th day	6th day	7th day	8th day
Intake										
Energy										
[kcal/kg/day]	total	80-86	68.5	100.8	108.1	124	116.4	126.5		
	parenteral			29	29	29	17	12		
	enteral		68.5	71.8	74.1	90.1	94.4	112.5		
	BMF				5	5	5	2		
Protein	total	1.5-1.75*	4	4.8	5	5.3	5.2	6.4		
[g/kg/day]	parenteral			0.9	0.9	0.9	0.9	0.9		
	enteral		4	3.9	4.1	4.4	4.3	5.6		
	BMF									
Carbohydrates	total	10-12*	9.0	6.1	15	16.7	13.3	17		
[g/kg/day]	parenteral			2.8	2.8	3.4	3.4	2.8		
	enteral		9.0	8.9	11	12.5	9.3	13.6		
	BMF				1.2	1.2	1.2	0.6		
Lipids	total	2.0-2.5*	2.2	3.9	3.2	4.4	3.1	3.7		
[g/kg/day]	parenteral			1.6	1.6	1.6				
	enteral		2.2	2.3	1.6	2.8	3.1	3.7		
Sodium	total	1.0-1.5*		1.96	3.75	3.13	2.92	3.15		
[mmol/kg/day]	parenteral			0.70	0.86	0.86	0.86	0.15		
	enteral		1.27	1.26	2.73	2.11	1.91	2.02		
	BMF				0.16	0.16	0.16	0.08		
Potassium	total	1.0-1.5*	3.72	3.12	3.72	4.99	4.61	3.87		
[mmol/kg/day]	parenteral			0.47	0.47	0.47	0.47			
	enteral		3.72	2.65	3.17	4.52	4.14	3.87		
	BMF				0.08	0.08	0.08	0.04		
Calcium	total	0.2-0.4*	1.8		1.81	1.36	3.46	1.76		
[mmol/kg/day]	parenteral			0.16	0.53	0.53	0.53	0.27		
	enteral		1.8	0.67	0.75	0.3	2.4	1.22		
	BMF				0.53	0.53	0.53	0.27		
Phosphorus	total	0.2-0.4*	2.03	2.72	3.11	2.8	3.25	3.46		
[mmol/kg/day]	parenteral			0.12	0.47	0.47	0.36	0.19		
	enteral		2.03	2.6	2.11	1.8	2.36	3.0		
	BMF				0.53	0.53	0.53	0.27		
Laboratory test results										
Potassium [mmol/l]	3.5-5.0	3.8		4.1	4.0		4.7			5.2
Phosphorus [mmol/l]	0.74-1.52			0.6	0.59		0.94			1.21
Calcium [mmol/l]				2.15	2.15		2.23			2.29
Sodium [mmol/l]	136-145	136.4		136.2	140.0		140			137.8

**Normal values during parenteral nutrition based on 'European recommendations for parenteral nutrition' Ass. Prof. Janusz Książyk, MD, PhD*

the girl received iron, both intramuscularly and orally, and erythrocyte parameters were gradually normalized. During the second day haemoglobin level was 7.3 g/dl, during the fifth day – 7.9 g/dl, iron – 17.9 μg/dl; during the fifteenth day haemoglobin – 10.6 g/dl. Regarding persistent leucocytosis, urine culture was performed among all, finding a significant titre of pathogenic flora in urine from the catheter. Treatment consistent with the antibiogram was applied (control urine culture was negative). Regarding the extreme anaemia that occurred in the child during the day of admission, stool examination for occult blood was performed to exclude

source of bleeding. The result of stool examination for occult blood was negative and endoscopic examination of the upper part of the digestive tract did not reveal macroscopic lesions responsible for such a significant degree of anaemia. Simultaneously, malabsorption syndrome was excluded on the basis of histopathological examination of a biopsy specimen from the small intestine. The girl was hospitalized in the Department for 15 days. She was admitted when she weighed 8 kg and she was discharged when she weighed 9.5 kg. She was cheerful, joyful. She willingly participated in rehabilitation training. She willingly accepted the recommended diet for small children. No technical difficulties connected with feeding were observed. The mother was instructed to feed her children properly considering both nutritional compounds and methods of care for a child with infantile cerebral palsy. After discharge from hospital, the family remains under the control and care of the Regional Social Care Centre.

Discussion

The first reports concerning 'refeeding syndrome' appeared during the 1940s. Fighting at the fronts, concentration camps, prisoner-of-war camps, and ghettos predisposed to states of chronic malabsorption. Intensive feeding of victims who survived captivity in concentration camps caused insufficiency of the circulatory system and neurological disturbances such as coma or seizures. Hypophosphataemia during intensive nutritional therapy was observed for the first time in 1940 among Japanese soldiers. These reports were published by Schnitker *et al*. [10] not before 1951. In 1946 Stein and Fenigstein published a study concerning cardiological-neurological disturbances after long-term starvation in persons from the Warsaw ghetto in 1942 [11]. 'Refeeding syndrome' has often been described since the 1970s as a complication of intensive nutritional treatment, especially parenteral [12]. According to Keys *et al*., refeeding syndrome occurs after 6 months of malnutrition [6].

Reports concerning 'refeeding syndrome' in Polish literature are not numerous; most often they are review articles. A case of severe hypophosphataemia was characterized by Słodkowska *et al*. in 2004. This report concerned a 53-year-old patient after operative treatment of a pancreatic tumour. Similarly to the patient described by us, he was fed with a low-protein and low-calorie diet, but he was not under systematic medical control. After admission to the hospital, parenteral feeding was introduced due to intolerance of enteral feeding and severe general state. Similarly as in our patient, symptoms occurred during the third day of total parenteral nutrition in this case. Phosphorus level was 0.25 mmol/l; consciousness disorders, hypotonia and tachycardia occurred [12].

The patient described by us was malnourished probably due to social causes. Differential diagnostics excluded malnutrition as the result of a severe form of underlying disease. Gastroesophageal reflux is characteristic for infantile cerebral palsy, often with concomitant oesophagitis. Regurgitations, nausea, and particularly odynophagia cause aversion to food. The patient described by us suffered from cerebral palsy, but no symptoms from the gastrointestinal tract were noted. Also endoscopy of the upper part of the gastrointestinal tract did not reveal any organic lesions in the oesophagus that could result in poor appetite. Malabsorption syndrome was excluded as a cause of malnutrition and anaemia on the basis of histopathological examination of the biopsy specimen of the small intestine.

According to Patrick [13] and Manary *et al*. [14] 'refeeding syndrome' occurs in malnourished children whose body mass is 45-75% of standard proper body mass. The presented patient had an even lower body mass, 44.4% of the proper body mass for her age. Such a significant degree of malnutrition and similar complications of nutritional treatment are described in patients suffering from anorexia nervosa [15-17]. Kohn *et al*. [15] analysed 48 patients and found hypophosphataemia in three of them, but they also presented cardiological and neurological symptoms. Body mass of these patients was below 70% of proper body mass. Ornstein *et al*. consider that hypophosphataemia is found in 7% of patients suffering from anorexia nervosa during nutritional treatment. Reports concerning low phosphorus level during nutritional treatment in girls with anorexia nervosa are also present in Polish literature. Three cases of anorexia nervosa in teenagers, observed by Bukowska *et al*. [18], developed electrolyte disturbances after application of total parenteral nutrition. The disturbances were probably caused by 'refeeding syndrome'.

Extreme anaemia was noted in the presented girl, who needed urgent transfusion of erythrocyte concentrate and subsequently iron supplementation. The blood iron level was impossible to measure at the day of admission due to the level being below the limit. Data obtained during anamnesis revealed that the child was fed mainly with milk preparations. This diet, poor in iron and vitamins, predisposes to anaemia [19]. However, there are no reports in the literature about such a low haemoglobin level during malnutrition. After admission, the patient was fed mainly enterally, but she also received a standard dose of vitamins. Initial aversion to food (predominantly impossibility to eat solid food), very low haemoglobin values, and decreased protein

concentration, meant that additionally she received parenterally (25 kcal/kg/day) the entire provision of necessary vitamins and micro and macroelements.

On the basis of a literature review, it is possible to conclude that 'refeeding syndrome' is characteristic for intensive parenteral treatment, but only a few reports describe it during enteral nutritional treatment [17].

Basal energetic requirement in our patient was calculated according to Schofield (REE) and it was 75.5 kcal/kg/day. Considering increased energetic requirement of about 50-80% in the case of malnutrition treatment, recommended caloric intake in the child was 113-136 kcal/kg/day. A diet rich in calories and proteins, used as standard in malnourished children, was initially introduced. It was not expected that the child would develop such serious metabolic disturbances. However, simultaneous, systematic application of vitamin and mineral supplementation prevented sudden life-threatening hypophosphataemia. There was no necessity to abandon the recommended diet when proper phosphorus supplementation was applied.

Low phosphorus level (0.60 mmol/l) occurred in the described patient during the third day of feeding. Both intravenous (0.47 mmol/kg/day) and oral (2.30-2.63 mmol/kg/day) supplementation was applied, together in the amount of about 3 mmol/kg/day. The girl received orally breast milk fortifier (BMF) appropriate for neonates and infants with low birth weight. This preparation, apart from its standard use, is a rich source of phosphorus and calcium. We apply 22 mg of phosphorus after dissolution of bag contents in 20 ml of water, which makes a triple concentration higher than milk mixture and more than twice as high as the standard milk hospital diet for small children. Daily requirement for inorganic phosphorus in a child from 4 to 6 years old is 0.2 mmol/kg/day. Despite intake that exceeds tenfold the normal value for the age, the phosphorus level was still decreased during the next day. Normalization occurred during the sixth day of treatment. Despite the fact that observed and intensively normalized phosphorus levels in the patient did not threaten her life directly, these levels fulfilled the criteria of 'refeeding syndrome'. It is not clear to properly assess the clinical features in the child. Signs concerning the circulatory system have not been observed, but those concerning the neural system (which occur earlier and are pathognomonic for the syndrome) would not have been possible to detect because of the basic illness (infantile cerebral palsy).

According to Marik *et al*., 'refeeding syndrome' during nutritional treatment can be diagnosed when the level of inorganic phosphorus is 0.16-0.65 mmol/l. This decrease occurs most often after 48 h from the beginning of therapy [20]. Crook *et al*. consider that severe hypophosphataemia means the serum value of 0.32 mmol/l. Symptoms from the circulatory system and the central nervous system appear at such a low phosphorus level [21]. Afzal *et al*. describe a 14-year-old girl with symptoms of chronic diarrhoea that persisted for 2 months during Crohn's disease. Significant heart rate acceleration and phosphorus level decrease occurred after introduction of nutritional therapy that comprised 2500 kcal/day and constituted 120% of caloric requirement in the described patient. Phosphorus level decreased during the fourth day to the level of 0.63 mmol/dl, during the fifth day to the level of 0.23 mmol/l. Phosphorus was initially supplemented at the value of 1.25 mmol/kg/day [9]. Considering the lack of improvement during the subsequent 24 h, caloric intake was limited to 1800 kcal/day, and she received 5 mmol/kg/day of phosphorus. Gradually phosphorus supplementation (3 mmol/kg/day) was withdrawn after obtaining normal phosphorus level during the seventh day. Fan *et al*. analysed 158 patients with disturbances of the gastrointestinal tract and observed phosphorus level decrease during nutritional treatment as early as during the first day (the average level of inorganic phosphorus was 0.56 ±0.19 mmol/l); then during the two subsequent 24 h it was 0.42 and 0.43 ±21 mmol/l. Normalization occurred during about the eighth day. Decrease of the calcium level down to 2.13 ±0.15 mmol/l was present also during the third day [22]. Also Kohn and Beumont found hypophosphataemia in patients during the first week after introduction of nutritional therapy [15, 16]. Severe cases of hypophosphataemia during treatment of anorexia nervosa are also described in the literature. Pertschuk *et al*. observed 11 patients, including 2 of them in whom refeeding syndrome occurred, but one patient died [23]. Other authors, such as Kohn *et al*. [15] and Fischer *et al*. [14], observed arrhythmia in patients suffering from anorexia nervosa, but Beumont *et al*. [16] found neurological disorders. Other reports concern 'refeeding syndrome' of kwashiorkor type in malnourished children. Manary *et al*. [3] during a retrospective analysis concerning the course of nutritional treatment of children in Malawi ascertained that 5 of 8 children with 'refeeding syndrome' died.

The presented case is evidence of the difficulties of management concerning patients with extreme malnutrition. Knowledge of possible complications during nutritional therapy can limit complications of cardiovascular and neurological disorders, and can even prevent deaths.

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