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Lactose Intolerance in Infants with Gluten-Sensitive Enteropathy: Frequency and Clinical Characteristics

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SUMMARY

Introduction Secondary lactose intolerance (SLI) belongs to the rarer manifestations of gluten-sensitive enteropathy (GSE). It occurs in more severe forms of the disease and its presence contributes significantly to the degree of its expression.

Objective The goal of the study was to determine the frequency of SLI in infants with clinically classic form of GSE, as well as its relationship with the duration, severity and age at the diagnosis of the basic disease and the degree of small bowel mucosa damage.

Methods The study was based on a sample of 42 infants, 30 female and 12 male, aged 7-12 months (x=9.98±1.69), with a clinically classic form of GSE. The diagnosis of GSE was established based on the characteristic pathohistological appearance of small bowel mucosa and clinical improvement of patients on gluten-free diet, while SLI on pathological lactose or milk tolerance test. The assessment of basic disease severity was based on body mass divergence in relation to the standard value, as well as on Hb and serum iron levels, while the degree of small bowel mucosa damage was determined according to the modified Marsh criteria.

Results SLI was verified in 8/42 or 19.05% of patients. In addition to the symptoms and clinical signs of GSE, all the patients with SLI also featured the problems characteristic of lactose tolerance disorders, i.e. watery diarrhoea, borborygmus and meteorism occurring after milk meals. In addition, all had perianal erythema (6 with erosive changes), as well as destructive enteropathy (5 subtotal and 3 total). The difference in the duration of the basic disease, age at diagnosis, as well as in the degree of body mass deviation from the standard value between the lactose-tolerant and lactose-intolerant infants was not found. In addition, no difference in Hb and serum iron levels or in the degree of small bowel mucosa damage was found between the two groups.

Conclusion Our findings indicate that SLI presents a relatively frequent occurrence in infants with clinically classic GSE, as well as that it occurs independently to the duration, severity and age at diagnosis of the basic disease and the degree of small bowel mucosa damage.

Keywords: lactose intolerance; infants; gluten-sensitive enteropathy

INTRODUCTION

Gastrointestinal lactose intolerance presents the most frequent nutritional disorder [1-3]. It usually develops as the result of the primary or secondary deficiency of lactase activity (hypolactasia) [2, 4]. Contrary to the primary, which presents as a developmental or genetically defined occurrence, the secondary form of lactose intolerance is caused by the damage of the small bowel mucosa [2-6]. As it disappears with the patient's improvement this form of lactose intolerance is also called transitory. Numerous diseases followed by the morphological damage of the small bowel mucosa, such as viral enteritis, intestinal lambliasis, protein-sensitive enteropathy, severe malnutrition and others, lead to secondary lactose intolerance (SLI) [2, 6, 7]. The secondary disorder of lactose tolerance also occurs as a reaction to the use of antibiotics and gastrointestinal prokinetics, as well as after gastroectomy and extensive small bowel resection [4, 8-10]. Clinical manifestation of lactose intolerance is, generally speaking, most variable and depends, not only on the severity of enzymic deficit and on the degree of its overload, but on the patient's age and compensatory capacity of the colon as well [4, 6, 7, 11-13].

One of the diseases that are relatively often followed by secondary hypoplasia is also gluten-sensitive enteropathy (GSE) [2, 4-6, 14-16]. According to the reports from the literature, in this disease a clinically manifest deficit of lactose activity, especially that of severe form, is relatively rare [2, 7, 14-18]. It mainly occurs in severe and neglected forms of the disease, and by its presence, it significantly contributes both to the severity of diarrhoeal disorder and to the degree of undernourishment of the patient [6, 7, 16]. Having in mind all these facts and milk nutritional significance, it is clear that SLI presents a serious problem (handicap) for children with GSE, and particularly those of the earliest age [7].

OBJECTIVE

The goal of the study was to assess the frequency of SLI in infants with GSE. In addition, we evaluated the relationship of this disorder with the duration and age

at GSE diagnosis, as well as with the basic clinical and laboratory nutritional parameters of patients, and the degree of small bowel mucosa damage.

METHODS

We retrospectively analyzed a sample of 42 infants, 30 female and 12 male, aged 7-12 months (X=9.98±1.60), with a clinically classic type of GSE, i.e. the type of the disease followed by chronic diarrhoea (>2 weeks) and disordered development. The diagnosis of GSE was based on the characteristic pathohistological appearance of the small bowel mucosa and clinical improvement of the patient on gluten-free diet [19]. The diagnosis was preceeded by a detailed illness history, a complete physical examination and relevant laboratory investigations.

All the patients with the history of watery, explosive and foamy stools after milk intake and/or the presence of perianal erythema with marked meteorism, underwent lactose tolerance test (LTT) or milk tolerance test (MTT). The confirmation of lactose intolerance was based on pathological LTT or MTT findings, i.e. the presence of watery diarrhoea, meteorism, as well as positive Clini test findings (>0.5%) and a low stool Ph (<5.5) after the intake of 10% of lactose solution in the dosage of 2 g/*kg* body mass or 200-220 ml of highly adopted cow's milk [6, 7, 20, 21]. None of the patients was on antibiotics, and none had gastrointestinal infection or some other condition followed by lactose intolerance.

In addition, all the patients were investigated in detail for history data at the onset and duration of the basic disease, while during clinical examination in each a precise body length and body weight were measured and compared to the referent values for the corresponding age and gender [22]. The body length values were expressed in percentiles, and body weight deviations in relation to ideal values in percentages. Hb and serum iron levels, as the laboratory parameters of the nutritional status, were determined by standard methods from a blood sample taken in the morning before breakfast. The diagnostic criteria for anaemia was serum Hb level below 110 g/L, and for sideropenia serum iron concentration below 10.7 μmol/L [23-25]. Hb values ranging from 100-109 g/L indicated mild, from 70-99 g/L moderate, and below 70 g/L severe anaemia [23].

Small bowel mucosa samples were obtained by aspiration or endoscopic enterobiopsy. By the former method biopsy was performed from the initial part of the jejunum or duodenum, and by the latter from the postbulbous (descending) part of the duodenum. Using aspiration enterobiopsy, we obtained two, and by endoscopic enterobiopsy three to five samples of the mucosa. Immediately after the biopsy and adequate orientation, each specimen was stereomicroscopically analyzed in detail. After the stereomicroscopical evaluation and a precise description, the mucosa specimens were immersed in a standard formalin solution and were then sent for a pathohistological analysis. The classification of the degree of small bowel mucosa damage was made according to the modified Marsh criteria, dividing it into inflammatory damage of infiltrative (I), infiltrative-hyperplastic (II), destructive (III) and hypoplastic (IV) type [26, 27]. Depending on the degree of villous degeneration, destructive enteropathies were additionally differentiated into partial (IIIa), subtotal (IIIb) and total (IIIc) [27, 28].

The difference between the lactose intolerant and tolerant infants in the duration and age at GSE diagnosis, as well as in the degree of body weight deficit and Hb and serum iron levels were analyzed by the Student's t-test, and in the severity of small bowel mucosa damage by the Mann-Whitney test.

RESULTS

Basic data related to the whole group of patients are presented on Table 1. Among the total 42 patients, 35 infants were on maternal milk; of these, only 4 were concurrently introduced to gluten containing diet. However, none of the infants was breast fed at the time of GSE diagnosis. Except for one infant whose body weight was below the low limits of the referent value for the corresponding age and gender, the remaining patients had normal longitudinal growth. All the infants had body weight deficit; in 19 (45.24%) it was above 20%. Anaemia was registered in 30 (71.43%) patients, of whom in 18 it was mild and in 12 moderate, while sideropenia was detected in 34 (80.95%). All the patients had enteropathy of the most severe degree, of whom in one only it was partial.

Of total 42 patients with the classic form of GSE, SLI was confirmed in 8 or 19.0%. Beside the symptoms and clinical signs of GSE, all SLI patients also had additional problems, i.e. watery diarrhoea, borborygmus and meteorism after milk meal. In addition, all had perianal erythema, of whom with erosive changes in 6. None of the patients had gastrointestinal infection or any other pathological conditions followed by lactose intolerance, and none showed allergy to cow's milk proteins. Nutritional lactose restriction, with gluten-free diet, resulted in a rapid recovery of the patients followed by improvement in the consistency and number of stools, as well as in the loss of perianal erythema. In none of the infants, SLI concomitant with GSE did not last over 2-3 weeks after the introduction of the diet.

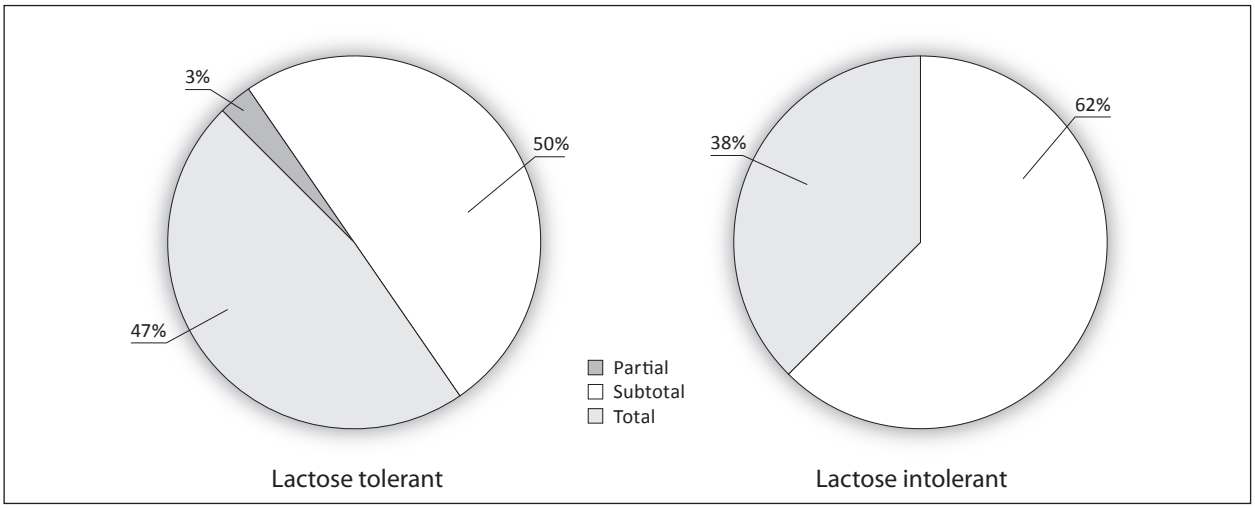
Table 1. Basic data in infants with GSE (n=42)

Data	Values	Range
Age at introduction of gluten-free diet (months)	3-6	4.50±0.90
Age at onset of first symptoms of GSE (months)	4-11	7.49±1.75
Duration of problems until GSE diagnosis (months)	1-5	2.49±1.39
Age at diagnosis (months)	7-12	9.98±1.60
Body weight deficit (%)	-5 to -40	-18.70±9.44
Body length (P)*	5-95	40±25
Blood Hb (g/l)	71-126	102.07±19.21
Serum iron (μmol/l)	2.2-19.5	7.06±4.12
Enteropathy (IIIa:IIIb:IIIc)	1:22:19	

* One patient below P_3

Table 2. Difference in age, duration of problems, clinical and laboratory parameters of nutritional status and the degree of small bowel mucosa damage between the lactose-intolerant and lactose-tolerant infants with GSE (n=42)

Assessed features	Lacotose-intolerant		Lactose-tolerant		Statistical significance	
	Value	Range	Value	Range		
Age (months)	8-12	9.94±1.47	7-12	9.99±1.65	t=0.075	p=0.941
Duration of problems (months)	1-5	2.25±1.49	1-5	2.54±1.38	t=0.534	p=0.597
Percent in BW divergence	-17 to -33	-22.13±5.19	-20.5 to -40	-17.91±10.08	t=1.140	p=0.261
Blood Hb (g/l)	102.5-126.0	100.75±16.5	97.0-125.0	102.4±20.05	t=0.214	p=0.831
Serum Fe (μmol/l)	3.30-12.2	5.71±3.32	2.2-19.5	7.38±4.29	t=0.889	p=0.381
Enteropathy IIIa:IIIb:IIIc (%)	0.0:62.5:37.5		2.9:50.0:47.1		Z= -0.385	p=0.700



Graph 1. Distribution according to the severity of enteropathy in lactose-tolerant and lactose-intolerant infants with GSE (n=42)

Among the features set in the study objectives, differences between the lactose intolerant and lactose tolerant infants are presented on Table 2 and Figure 1. As evident, there were no significant differences between these two groups of patients, either in the basic disease duration, age at diagnosis, or in the deficit of body weight, and Hb and serum iron levels. In addition, there was also no significant difference in the severity of small bowel mucosa damage.

DISCUSSION

Lactose is the basic milk carbon hydrate in most mammals [29]. It consists of glucose and galactose molecules interlinked by the β-1.4 glucoside configuration. In addition to energetic significance, lactose stimulates the resorption of calcium, magnesium and iron, as well as the colonization of the large bowel with Bifidobacterium and Lactobacillus bacteria [2, 30]. Lactose hydrolysis, which presents a precondition for its absorption, is promoted by lactase (β-galactosidase), a specific hydrolase that is linked with its C-terminal ending to the luminal side of the erythrocyte membrane in the proximal small bowel segment [2, 7, 31]. After being released, glucose and galactose molecules are by active co-transport with sodium transferred into the enterocyte, which then exits it to be easily diffused throughout the portal bloodstream. By phosphorization processes, transfer to uridine-diphosphate and epimerization occurring in the liver under the activity of galactokinase, galactose 1-phosphate uridyl transferase and uridine diphosphate-4-epimerase, galactose is transformed into glucose [33, 34]. Therefore, according to the level of development lactose tolerance disorders are classified into two groups of clinical entities, of which the former occur due to hypolactase, gastrectomy or glucose and galactose malabsorption, and the latter due to the deficit of galactokinase, galactose 1-phosphate uridyl transferase or uridine diphosphate-4-epimerase [2, 35]. Except for the deficiency of lactase activity, other causes of lactose intolerance are rare [2, 35].

GSE belongs to the diseases often followed by hypolactasia [2, 4, 6, 14-18]. The deficiency of lactase activity occurs as the result of small bowel mucosa inflammation, i.e. the reduction of its functional surface and epithelial immaturity [36]. Although the changes are most prominent in the small bowel segment, where also lactase activity is highest owing to its remaining fraction and compensatory role of the colon, clinically manifest hypolactasia is relatively rare and is mostly seen in the severe forms of the disease [2, 4, 7, 14, 17, 18, 37, 38]. In the group of our SLI patients, it was disclosed in 8/42 or 19.05%. All had a severe form of GSE and destructive small bowel mucosa damage; in 5 subtotal and in 3 total. In addition, the patients were of infantile age, which is characterized by a relatively high lactose overload, physiologically more vivid peristalsis and a lower compensatory capacity of the colon [4, 6, 7].

Clinical features of lactose intolerance are quite typical. Problems occur immediately after milk meals and depend, not only on the enzymatic deficiency and on the degree of its overload, but also on the patient's age, as well as the compensatory capacity of the colon [4, 37-39]. More severe forms of the disorder, particularly in infants and small

children, are characterized by osmotic diarrhoea, periodically of such intensity that, not only does it disturb water and electrolyte balance, but also the nutritional status of the child, while in milder forms and at older age the basic symptomatology involves abdominal pains of colic type, meteorism and increased flatulence [6, 7]. All our patients with SLI, beside the symptoms and clinical signs of GSE, also had the problems characteristic of lactose maldigestion, i.e. watery diarrhoea, borborygmus and meteorism after milk meals. In addition, in all we disclosed perianal erythema associated with erosive changes in 6 of them.

Beside a strict diet, the treatment of SLI concomitant with GSE also involves a contemporary elimination of lactose in the patient's nutrition [3, 6, 7, 40-42]. With this goal, the infant on artificial diet is fed on some of milk lactose-free formulas, and the older child on yogurt or some other fermented dairy product (sour milk, kefir yogurt and cheese) [4, 6, 43-45]. As all our patients with SLI were infants, beside strict gluten-free diet, all were fed on lactose-free cow's milk formulas. The application of these dietetic-therapeutic measures resulted in the decreased number and improvement of stool consistency, withdrawal of perianal erythema and increase in the patients' body weight. In our sample of patients, lactose elimination from meals was necessary to be used for only 2-3 weeks.

However, the comparison of the basic characteristics of GSE that could influence the clinical expression of hypolactasia did not indicate the presence of significant differences. It showed that in this relation the studied sample was homogenous, but concurrently a question also emerges as to the basic clinical expression of hypolactasia in 8 of 42 patients. As none of these infants, except for GSE and associated malnutrition, had no other cause of lactose intolerance, it can be concluded that the explanation for this could be found in different extensities, i.e. in the degree of small bowel mucosa involvement as a whole, as well as in the individual variations of the compensatory capacities of the colon and the level of lactose activity [4, 6, 33, 35, 46].

CONCLUSION

The results of our study indicated that there was a relatively high incidence of SLI in infants with a classic form of GSE. Beside symptoms and clinical signs of such form of GSE, all the infants with SLI had problems characteristic of lactose intolerance. As the sample of subjects was quite homogenous, both regarding the age and the severity of the basic disease, the presence of lactose intolerance in one group of patients could be explained by the difference in the extensiveness of small bowel mucosa disorder, as well as in the individual variations in the compensatory capacity of the colon and the level of lactose activity.

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Секундарна интолеранција лактозе код одојчади са глутен-сензитивном ентеропатијом: учесталост и клиничке одлике

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КРАТАК САДРЖАЈ

Увод Секундарна интолеранција лактозе (СИЛ) је ретка манифестација глутен-сензитивне ентеропатије (ГСЕ). Јавља се у тежим облицима болести и битно доприноси степену њеног испољавања.

Циљ рада Циљ рада је био да се утврди учесталост СИЛ код одојчади с клинички класичном ГСЕ, као и њен однос са трајањем, тежином и узрастом дијагностиковања основне болести и степеном оштећења слузнице танког црева.

Методе рада Истраживање је обухватило 42 одојчета (30 женског и 12 мушког пола), узраста од седам до 12 месеци (просечно 9,98±1,69 месеци), с клинички класичним обликом ГСЕ. Дијагноза ГСЕ је постављена на основу типичног патохистолошког изгледа слузнице танког црева и резултата клиничког опоравка болесника на дијети без глутена, а дијагноза СИЛ на основу патолошког налаза теста којим се испитивало подношење лактозе или млека. Процена тежине основне болести заснивала се на одступању телесне масе у односу на стандардну вредност, као и на нивоима хемоглобина и гвожђа у крви, док су за одређивање степена оштећења слузнице танког црева коришћени модификовани Маршови (*Marsh*) критеријуми.

Резултати СИЛ је потврђена код осам испитаника (19,05%). Осим симптома и клиничких знакова ГСЕ, код свих болесника са СИЛ су се испољавале и сметње типичне за поремећај подношења лактозе: водена дијареја, борборигми и метеоризам после оброка млека. Такође, код свих су уочени перианални еритем (код шест с ерозивним променама) и деструктивна ентеропатија (код пет суптотална, а код три тотална). Разлика у трајању основне болести, узрасту у којем је постављена дијагноза и степену одступања телесне тежине у односу на стандардну вредност између одојчади која подносе лактозу и оне која је не подносе није забележена. Такође, између ове две групе испитаника није било разлике ни у нивоима хемоглобина и гвожђа у крви, нити у степену оштећења слузнице танког црева.

Закључак Резултати овог истраживања показују да је СИЛ релативно честа појава код одојчади с клинички класичном ГСЕ, те да се јавља независно од трајања, тежине и узраста дијагностиковања основне болести и степена оштећења слузнице танког црева.

Кључне речи: интолеранција лактозе; одојчад; глутен-сензитивна ентеропатија

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