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Serologcal and histological markers of glutenic enteropathy in rheumatoid arthritis and osteoarthritis patients

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The Aim of the study was to elucidate the frequency of glutenic enteropathy in rheumatoid arthritis and osteoarthritis patients as well as to assess the clinical serological and histological symptoms of this disease.

Patients and methods. The study cohort comprised 308 subjects (181 females and 127 males) aged 18–86 years. They were divided into three groups. The main group consisted of 183 rheumatoid arthritis and osteoarthritis patients, the control group comprised of 90 healthy subjects, and in the third, comparative group there were 35 patients ill with inactive stomack and/or duodenal ulcer, chronic inactive gastritis (in remission phase) without history of other acute or chronic diseases of digestive tract or joint and bone pathology.

Blood serum IgA class gliadin and tissue transglutaminase antibody levels were determined by the immunoenzymatic (ELISA) method using IB1 – Hamburg reagents (Germany). The reaction was carried out in polystyrene hollows.

Results. In our study, in the group of rheumatic patients enhanced blood serum levels of class IgA antigliadin (AGA) antibodies were found in 42/183 (23%) and in 36/139 (25.9%) rheumatoid arthritis and in 6/44 osteoarthritis patients. In the control group, increased levels of IgA antigliadin antibodies in blood serum were found for 2/90 subjects (2.2%) and in the comparative group for 2/35 patients (5.7%) who had digestive tract diseases in remission phase. In total, enhanced blood serum levels of IgA antigliadin antibodies were found for 44/308 subjects (14.3%).

The rheumatoid arthritis patients showed the following grades of small bowel mucousa impairments: grade 3a was found in 21 (63.6%), grade 3b in 10 (30.3%) and grade 3c in 2 (6.1%) subjects according to the Marsh classification. Also in osteoarhritis patients prevailed small bowel mucosa derangements of a rather low grade: 3a in 3 (66.6%) and 3b in 1 (33.6%).

In rheumatoid arthritis patients, the frequency of class IgA antigliadin antibodies was significantly higher than in healthy subjects (25.9% *versus* 2.3%) and in subjects with digestive tract diseases in remission phase (5.7%).

In patients ill with glutenic enteropathy associated with rheumatoid arthritis, grade 3a derangement of small intestine mucosa was predominant (63.6%), as was the case also in patients in whom glutenic enteropathy was associated with osteoarthritis (66.6%).

Conclusion. The study confirms that selective examination of rheumatoid patients and subjects ill with other autoimmune diseases is a purposeful method.

Key words: glutenic enteropathy, serum indices, morphological changes

INTRODUCTION

Glutenic enteropathy is a chronic intestinal disease caused by some of cereal proteins which damage the small intestine mucous membrane in genetically predisposed subjects. A harmful effect on small intestine mucosa is exerted by gliadin (a gluten metabolite of wheat protein) and proteins of similar ce-

reals such as hordein in barley, secalin in rye and possibly avidin in oat. Glutenic enteropathy in adults is diagnosed in the presence of characteristic morpholgical changes in small intenstine mucosa and by a subsequent control of their clinical and morphological remission under a gluten-free diet. Celiac disease can be diagnosed by determining the endomysium (EmA), gliadin (AGA) and reticulin (ARA) IgA class antibodies in blood serum and their disappearance under the effect of aglutenic diet. Recently, tissue transglutaminase (tTG) has been identified as an endomysium autoantigen.

The diagnosing of the disease is complicated, as the clinical symptoms vary from those characteristic such as steatorrhea, diarrhoea, flatulence and abdomen pain, weight loss up to non-characteristic ones including anaemia, infertility, alopecia, hypertransaminasaemia, a subclinical or latent form of the disease. To these atypical symptoms also a deficiency of some microelements, digestive tract symptoms (abdomen tension, flatulence, secondary lactose intolerance, dyspepsia) are ascribed, alongside symptoms in ther organs (fatigue, depression, arthralgia, osteomalacia or osteoporosis, iron-deficiency-induced anaemia). Many of glutenic enterophaty patients before the diagnosis have for several years been suffering from prodromic extraintestinal symptoms with no pronounced abdominal indications; arthritis and arthralgia can be the first and the only sign of this enteropathy. Arthritis is most often mentioned among the extraintestinal symptoms of this enteropathy, however, definite data are lacking. There are hardly several publications to deal with an association between arthritis and glutenic enteropathy. The active form of the disease comprises only an insignificant part of all cases of glutenic enteropathy. Glutenic enteropathy is diagnosed mostly in individuals of mature age. The distribution frequency of clinically diagnosed cases in the literature varies greatly – from 1 : 1200 in the United Kingdom to 1 : 300 in West Ireland. However, these data do not reflect the actual frequency of the disease revealed by selective studies, which show a similar distribution throughout Europe. Such a great difference between the clinically determined frequency and the frequency revealed by selective studies can be explained by the predominance of low-symptomatic and latent forms of the disease, which clinically cannot be detected.

The early diagnosis and treatment of the subclinical forms of the disease is important, as these patients are exposed to a higher risk of remote complications such as anaemia, osteoporosis, infertility, tumours. Now that autoimmune patients undergo more frequent morphological examiantions as well as the testing for AGA, EmA, ARA and tTG anibodies, it is probable that the low-symptomatic and latent forms of the disease in Lithuania will be diagnosed more often.

The aim of the work was to elucidate IgA class antigliadin and tissue transglutaminase antibodies in the blood serum of rheumatoid arthritis and osteoarthritis patients and the control group members and to examine the small intestine mucosa for morphological changes in the individuals in whom IgA class antigliadin and tissue transglutaminase antibodies have been found.

The object of the study. The study cohort comprised 308 subjects (181 females and 127 males) aged 18–86 years. They were divided into three groups. The main group consisted of 183 rheumatoid arthritis and osteoarthritis patients, the control group comprised 90 healthy subjects, and in the third, comparative group there were 35 patients ill with inactive stomack and/or duodenal ulcer, chronic inactive gastritis (in remission phase) without the history of other acute or chronic diseases of digestive tract or a joint and bone pathology.

All study groups were matched as regards their age; their mean age statistically did not differ. The recruited subjects were asked to fill a questionnaire.

METHODS

Serum markers of glutenic enteropathy. The enzyme-linked immunosorbent assay (ELISA) is one of the most sensitive immunochemical methods. The essence of this method is formation of an antigen–antibody immune complex, which is revealed by secondary antibodies. The latter are marked with an enzyme which desintegrates the substrate, and a coloured reaction occurs.

Peripheral venous blood for the enzymatic ELISA analysis was taken from all study participants. The blood samples were centrifuged and kept at a temperature of –20 °C until use. Blood serum IgA class gliadin and tissue transglutaminase antibody levels were determined by the immunoenzymatic (ELISA) method using IBL – Hamburg reagents (Germany). The reaction was carried out in polystyrene hollows.

Peroral aspiratory small intestine biopsy and the morphological examination of the bioptates. Morphological investigation of small intestine mucosa is the basic diagnostic method for glutenic enteropathy. The biopsy substrate was taken with a Crosby capsule from the proximal part of the jejunum or from the distal part of the duodenum behind the Treitz's band, or from the distal part of the duodenum with a fibroduodenoscope. The capsule was introduced *per os* and its localization was adjusted by X-ray. The biopsy substrate, fixed in 10% formalin, was embedded in paraffin, cut into histological sections

5 μm thick, stained with hematoxylin and eosin and examined on a light microscope.

Statistical analysis. To process the data and calculate the results, the EpiInfo 2000 ver. 1.1.2 and EpiInfo 6 ver. 6.04 d software packages were used (Centers for Disease Control and Prevention (CDC), U.S.A. and World Health Organization, Geneva, Switzerland).

RESULTS

Comparison of GE serum markers in the study groups. GE serum markers were investigated in 183 subjects ill with joint diseases. Of them, 139 (75.9%) were rheumatoid arthritis and 44 (23%) osteoartritis patients. In the control group, elevated blood serum levels of IgA class antigliadin antibodies were found for 3/90 (2.2%) and in the comparative group for 2/35 (5.7%) subjects. In total, elevated blood serum levels of IgA class antigliadin antibodies were found for 44/308 (14.3%) subjects. The difference between data for the rheumatoid arthritis patients and the control group members for whom IgA class antigliadin antibodies had been found in blood serum was statistically reliable ($p < 0.0001$), as was the difference between data for osteoarthritis patients and members of the control group ($p < 0.05$). Data for rheumatoid arthritis patients and members of the comparative group for whom IgA class antigliadin antibodies in blood serum had been found also differed statistically reliably, whereas the difference between the data for asteoarthritis patients and members of the comparative group was statistically not reliable ($p > 0.05$) (Figs. 1 and 2).

In the group of rheumatoid patients, enhanced blood serum levels of transglutaminase (tTG) antibodies weer found for 6/78 (7.7%) subjects: 6/50 (12%) were rheumatoid arthritis patients, whereas no such antibodies were detected in osteoarhritis patients. In the control group, an enhanced ETG concentration in blood serum was found in 2/35 subjects (5.7%) from the comparative cohort, and no such antibodies were present in the healthy subjects. There was no statistically reliable difference between the data for the rheumatoid arthritis patients and the subjects from the comparative group for whom class IgA tissue tTG were found in blood serum (Fig. 3).

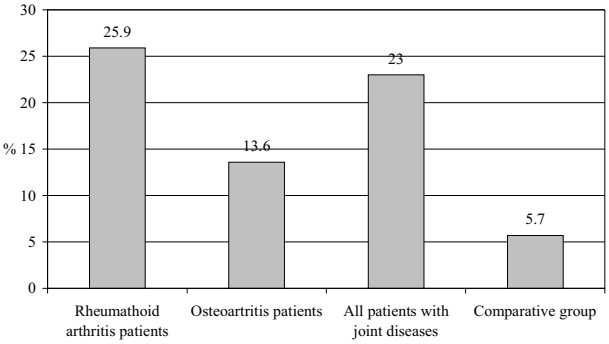

Fig. 2. Frequency of elevated blood serum levels of class IgA antigliadin antibodies in subjects with joint diseases and in comparative group ($p < 0.05$)

Comparison of morphological changes in small intestine mucosa in the study groups. Small intestine mucosa biopsy was performed and the bioptates were morphologically studied for 44 subjects, of them 42 (95%) rheumatoid patients and 2 (5%) patients with digestive tract diseases in remission phase. Of the 42 rheumatoid patients, small intestine mucosa biopsy was made and the bioptates were morphologically studied for 36 (85%) rheumatoid arthritis and 6 (15%) osteoarthritis patients. The grades of small bowel mucous membrane impairment in rheumatoid arthritis patients were distributed ar follows: grade 3a in 21 (63.6%), grade 3b in 10, and grade 3c in 2 (6.1%) according to Marsh's classification. Rheumatoid arthritis patients showed no statistically reliable differences between the morphological changes and arthritis activity level. The degree of small bowel mucosa impairment in rheumatoid arthritis patients was not

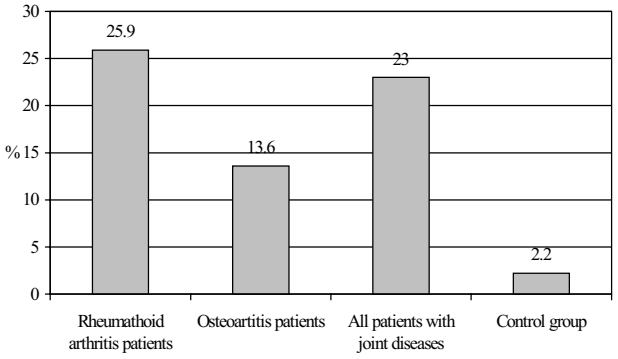

Fig. 1. Frequency of elevated blood serum levels of class IgA antigliadin antibodies in subjects with joint diseases and in control group ($p < 0.05$)

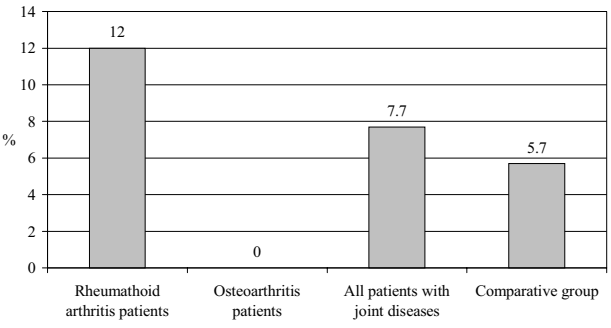

Fig. 3. Frequency of elevated blood serum levels of class IgA transglutaminase antibodies in subjects with joint diseases and in comparative group ($p < 0.05$)

high: among grade I° arthritis patients in 5 (6.1%) only the density of intraepithelial lymphocytes (IEL) in lamina propria was enhanced, two patients (6.1%) showed partial atrophy of small intestine mucous villi; no cases of total atrophy were stated; among grade II° arthritis patients, in 10 (30.3%) an increased IEL density in the lamina propria was found, five patients (15.2%) showed partial atrophy of villi in small intestine, and one patient (3%) developed a total atrophy of mucous membrane villi; among grade III° arthritis patients, six (18.1%) showed an increased IEL density in the lamina propria, three (9.1%) developed partial and one (3%) total atrophy of small intestine mucosa villi. In osteoarhritis patients the level of small intestine mucous membrane impairment was also not high, reaching grade 3a in two (66.6%) and grade 3b in one (33.3%) patient (Table 1).

A comparison of small bowel mucous membrane derangement degree and changes in glutenic enteropathy serum markers in rheumatic patients showed that a moderate increase in class IgA antigliadin antibody levels (>11<50 V/ml) was present in 42 (91.3%) subjects; of them, only a higher IEL density in the lamina propria was found in 29 (63%) and partial villous atrophy of small intestine mucosa in eight (17.4%) subjects, and in 5 (10.9%) the small intestine mucosa was normal. A significant increase of class IgA antigliadin antibody levels (> 50 V/ml) was detected in 4 (8.7%) patients, of them three (6.5%) showed partial villous and one (2.2%) total atrophy of small bowel mucosa; no grade 3a small intestine mucosa derangements were found. A moderate increase in class IgA tTG antibody level (>11<50 V/ml) was found in six (100%) subjects, of them two (33.3%) showed only an increased IEL level in the lamina propria, three (50%) developed partial and one (16.6%) total villous atrophy of small intestine mucosa. None of the study subjects showed a significant increase (>50 V/ml) in class IgA tTG antibody level. Subjects with a moderate increase of class IgA antigliadin antibody levels more often showed a grade 3a impairment of small bowel mucosa; in the presence of a significant increase of the level of these antibodies, more frequent was grade 3b impairment, as was also the case in subjects that showed a moderate increase of class IgA tTG antibody levels. The statistical reliability was not determined, possibly because of the small number of cases (Table 2).

Table 1. **Comprison of morphological changes in small intestine mucosa and rheumatoid arthritis activity**

Activity of disease	Morphological changes		
	IEL* density Marsh 3a (No/%)	Subtotal atrophy Marsh 3b (No/%)	Total atrophy Marsh 3c (No/%)
Grade I° RA**	5/15.2	2/6.1	0
Grade II° RA**	10/30.3	5/15.2	1/3
Grade III° RA**	6/18.1	3/9.1	1/3
All patients with RA** n = 33	21/63.6	10/30.3	2/6.1
All patients with OA*** n = 3	2/66.6	1/33.3	

* Intraepithelial lymphocytes. ** Rheumatoid arthritis. *** Osteoarthritis.

DISCUSSION

The pathophysiology, causes, clinical features of glutenic enteropathy and its relation to other diseases have been under study for more than a century; however, variations in clinical manifestations of this enteropathy, the problem of the increasing frequency of the disease and

Table 2. **Comparison of glutenic enteropathy serum markers and small bowel mucous membrane derangement in patients with joint disease**

Morphologic changes of small bowel mucous membrane	Moderate increase of AGA-A* levels (No/%)	Significant increase of AGA-A levels (No/%)	Moderate increase of tTG-A** levels (No/%)	Moderate increase of tTG-A levels (No/%)
IEL* density Marsh 3a	29/63%	–	2/33.3%	–
Subtotal atrophy Marsh 3b	8/17.4%	3/6.5%	3/50%	–
Total atrophy Marsh 3c	–	1/2.2%	1/16.6%	–
Normal	5/10.9%	1/2.2%	–	–
Total	42/91.3%	4/8.7%	6/100%	–

* Class IgA antigliadin antibodies. ** Class IgA transglutaminase antibodies.

its subclinical forms remain urgent, providing an incentive to search for the new clinical peculiarities and connections with other diseases in order to supplement our knowledge, which in practice could be helpful in revealing the early stages of the disease and reducing the risk of remote complications.

The main signs of untreated glutenic enteropathy are changes of small intestine: ciliar atrophy, crypt hyperplasia and an enhanced IEL density (1). An increased number of chronic inflammatory cells is found in lamina propria (2). Also, a reduced ECH (enterocyte height) is observed (3). Under aglutenic diet these histological changes become restored and accompanied by clinical recovery, however, they may reappear if gluten-containing products are consumed (4). When diagnosing glutenic enteropathy, it is particularly important to indicate that the damage of small intestine depends on gluten, since villose atrophy and intraepithelial lymphocytosis can be also evoked by other factors such as allergy to cow's milk, giardiasis and postenteritic syndrome (5). On the other hand, in developed countries these derangements are more rare in adults. In the case of glutenic atrophy, small intestine mucous membrane atrophy more often is manifested in the proximal part of the intestine, while the hip intestine mucous membrane can be not damaged (6). Based on the data on 11 adults with untreated glutenic enteropathy, it has been stated that the distinctness of the symptoms correlates with the damaged area of small intestine and does not depend on the degree of ciliar atrophy (7, 8, 9).

Pathogenetic aspects of glutenic enteropathy. Today the influence of the immunological mechanisms on the development on mucosa derangement in glutenic enteropathy patients is commonly recognized. Untreated patients exhibit signs of activation of mucous membrane cellular and humoral immune system (11). The basic environmental (external) factor is the consumed gluten. Gluten-specific are T cells related to HLA DQ2 and DQ8, found in damaged mucous mebrane of small intestine (12). Recently W. Dietrich and co-authors have established that the serum EmA, a specific indicator of active glutenic enteropathy, can be recognized by tissue transglutaminase. This enzyme is the basic, though not the only one, autoantigen in the case of glutenic enteropathy. In the case of active glutenic enteropathy tTG expression is enhanced, in HLA DQ2 and DQ8 molecules glutamine deamination is activated, thus gliadin can be bound to them (13). On this peptidic bond depends T cell bonding affinity and reactivity (14). Secretion of γ-interferon and other inflammatory cytokines in the activated cells of the lamina propria can damage small intestine mucous membrane (15). Besides, there is the opinion that antibodies against tissue transglutaminase can be directly involved in glutenic enteropathy pathogenesis. In an *in vitro* model, one can see that tGT suppresses epitelial differentiation on the crypt–villi axis (16). Whether tGT plays a role in the derangements of small intestine mucosa *in vivo* is not exactly known.

Glutenic enteropathy and rheumatic diseases. According to data published by G. T. Cooper and co-authors, among 314 celiac disease patients in 63 (20%) a disease of immune origin was diagnosed (17). There is an opinion that the immune complexes formed in the small intestine mucosa of glutenic enteropathy patients accumulate in other organs and trigger the development and progress of autoimmune diseases (18). The literature provides a description of nearly 100 diseases related to glutenic enteropathy, rheumatoid arthritis included (19). There are reports showing that glutenic enteropathy is related to rheumatic diseases in 0.4% to 2% of cases (20). In rheumatological practice, when performing differential diagnostics some of the clinical peculiarities of glutenic enteropathy should be borne in mind. There are facts to show that arthralgia and arthritis can be the first and only symptom of this enzymopathy (21).

Early diagnosis and treatment of the subclinical forms of the disease are very important, as these patients are at a higher risk of late complications such as anaemia, osteoporosis, infertility, tumours. Now that the gliadin, endomysium and tissue transglutaminase antibody tests are finding increasing application for examining autoimmune patients, a more frequent diagnosis of the low-symptomatic and latent forms of the disease can be expected in Lithuania.

Glutenic enteropathy and oxidative stress. Many of the diseases, digestive tract pathology (22) and arthritis among them, are connected with oxidative stress. Oxidative stress in the organism is induced by free radical excess in the cells. The indices characterizing the peroxidative and antioxidative status of the organism undergo changes. Free radicals are the chemical compounds that are able to exist independently with one or more unpaired electrons. One of the targets for free radicals to attack are lipids present in the membrane, or more precisely fatty acids present in their structure (24). Glutenic enteropathy, a disease ranked among intestine enzymopathies, is related to changes in the oxidative stress indices (25). Changes in the antioxidative system can be evoked by malabsorption (26).

In the present work, IgA class antigliadin and tissue transglutaminase antibodies were determined in the blood of patients with rheumatoid arthritis, osteoarhritis and digestive tract diseases in remis-

sion phase and of healthy subjects; morphological changes were examined in small intestine mucous membrane of the subjects in whom antibodies had been found; dependence of serum markers and their distribution on rheumatoid arthritis activity, disease duration, patients' age and sex was assessed.

The phenomenon of coexistence of various autoimmune diseases is well known. Patients ill with an immune disease are more prone to fall ill with another HLA system-related disease. As mentioned in the review of the literature, this enteropathy is linked with a number of rheumatic diseases as well as with rheumatoid arthritis. However, there are only several publications to describe a connection between glutenic enteropathy on the one hand and rheumatoid arthritis and osteoarthritis on the other, its clinical peculiarities.

Reliable data on the distribution of this disease in Lithuania are lacking. Approximate evaluations give us 0.24 : 1000 newborns (27), *versus* 2.9 : 1000 in Sweden. Such a great difference reflects the poor diagnostics of this disease rather than its actual occurrence, as comprehensive studies in The Netherlands and Italy have shown that the ratio of known and unknown cases of celiac disease is 1 : 7. The disease is somewhat more frequent in females; they comprise 52% to 71% of the patients (28).

Our study showed that the frequency of IgA class antigliadin antibodies in rheumatoid arthritis patients was 25.9% *versus* 2.2% in healthy subjects and 5.7% in patients with digestive tract diseases in remission phase, *i.e.* it was rather high. In rheumatoid arthritis patients, IgA class tissue tTG antibody frequency was 12% and in patients with digestive tract diseases in remission phase 5.7%. The frequency of glutenic enteropathy in rheumatoid arthritis patients was 23.7%, osteoarthritis patients 13.6% and in patients with digestive tract diseases in remission phase 5.7%.

Studies made in other countries showed the frequency of IgA class antigliadin antibodies for juvenile rheumatoid arthritis patients to be 3.1% (29), for type I diabetes mellitus patients 9–31% (Carlson et al., 1999), among blood donors in the U.S.A 2.2% (30) and in Brazil 3.03% (31), among healthy children of Estonia 3.1% (32), in North Spain general population 15%. The frequency of glutenic enteropathy among type I diabetes mellitus patients is 2–5%, autoimmune tyroiditis 4%, Sjögren syndrome patients 15% (33). Thus, data reported by various authors vary, the variations most possibly being dependent on the antibody determination technique, the homogeneity of the study cohorts and on other factors.

Data obtained in our study and reported in the literature allow to maintain that the frequency of IgA class antigluadinantibodies in rheumatic patients is reliably high compared with the healthy subjects. Therefore, selective examination of autoimmune disease patients seems justifiable. Serum (noninvasive) tests seem to serve the purpose best.

Results of small bowel mucous membrane morphological studies showed grade I° mucous membrane derangements according to Marsh's classification (infiltrative-hyperplastic variant) to be most frequent. In cases of moderate (<50 V/ml) IgA class antigliadin antibody levels, grade I° mucous membrane derangements prevailed, whereas grade II° derangements were predominant in cases of significantly increased antibody concentrations (>50 V/ml). Grade II° small intestine mucous membrane impairment prevailed also in patients to whom a moderate increase of IgA class tTG antibody levels had been diagnosed. We suppose that the development and intensity of the clinical symptoms of glutenic enteropathy depend on the damaged area of the small intestine mucous membrane rather than on the degree of the derangement. Glutenic enteropathy is characterized by an impaired absorptive function of small intestine mucosa induced by gluten. Which gene influences the development of the extraintestinal symptoms of this enteropathy is not exactly known. Malabsorption in glutenic enteropathy patients can be either generalized or limited to only several nutritive substances. This is the reason for the diversity of clinical manifestations of glutenic enteropathy in adults. Among the extraintestinal manifestations of glutenic enteropathy, arthritis, though rare, can be one of the primary symptoms in adults.

CONCLUSIONS

In rheumatoid arthritis patients, the frequency of IgA class antigliadin antibodies (25.9%) was statistically reliably higher than in healthy subjects (2.3%) and in patients ill with digestive tract diseases in remission phase (5.7%).

In patients ill with glutenic enteropathy associated with rheumatoid arthritis, grade 3a impairment of small bowel intestine mucosa was prevailing (63.6%), as was also the case in patients with glutenic enteropathy associated with osteoarthritis (66.6%).

To summarize, we can conclude that glutenic enteropathy cannot be ranked among rare diseases and that its clinical manifestations have changed. Thus, absence of many of the symptoms cannot exclude the possibility of glutenic enteropathy. Our study confirms that selective examination of rheumatoid arthritis patients and subjects with other autoimmune diseases is a purposeful method.

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SERUMINIAI IR HISTOLOGINIAI GLIUTENINĖS ENTEROPATIJOS ŽYMENYS SERGANT REUMATOIDINIU ARTRITU IR OSTEOARTRITU

S a n t r a u k a

Tikslas. Darbo tikslas buvo nustatyti gliuteninės enteropatijos dažnį sergantiesiems reumatoidiniu artritu ir osteoartritu bei įvertinti klinikinius, serologinius ir histologinius šios enteropatijos požymius.

Ligoniai ir metodai. Gliuteninės enteropatijos tyrimai iš viso atlikti 308 pacientams 18–86 metų amžiaus, tarp jų 181 moteriai ir 127 vyrams. Tirtieji buvo suskirstyti į tris grupes. Pagrindinėje tirtųjų grupėje 183 sirgo reumatoidiniu artritu ir osteoartritu. Kontrolinę grupę sudarė 90 sveikųjų. Trečią, palyginamąją, grupę sudarė 35 pacientai, sirgę neaktyvia skrandžio ir/ar dvylikapirštės žarnos opalige, lėtiniu neaktyviu gastritu (remisijos fazė), tačiau nesirgę kitomis ūminėmis ar lėtinėmis virškinimo sistemos ligomis, neturėję sąnarių-kaulų sistemos patologijos.

IgA klasės gliadino ir audinių transgliutaminazės antikūnų koncentracija kraujo serumuose nustatyta imunofermentiniu (ELISA) metodu naudojant IBL – Hamburg reagentus (Vokietija). Reakcija atlikta polistirolo plokštelės duobutėse.

Rezultatai. Mūsų tyrimo duomenimis, sergančiųjų sąnarių ligomis grupėje padidėjusi Ig A klasės antigliadino (AGA) antikūnų koncentracija kraujo serume nustatyta 42 atvejais (23%) iš 183: 36 (25,9%) asmenims iš 139 ligoniams, sergantiems reumatoidiniu artritu, taip pat 6 (13,6%) asmenims iš 44 ligoniams, sergantiems osteoartritu. Kontrolinėje grupėje padidėjusi IgA klasės antigliadino antikūnų koncentracija kraujo serume nustatyta dviem (2,2%) asmenims iš 90, palyginamojoje grupėje – dviem (5,7%) iš 35 ligonių, sirgusių virškinimo trakto ligomis (remisijos fazė). Iš viso padidėjusi IgA klasės antigliadino antikūnų koncentracija kraujo serume nustatyta 44 (14,3%) asmenims iš 308 ištirtųjų.

Sergantiesiems reumatoidiniu artritu nustatyti tokie plonosios žarnos gleivinės pažeidimo laipsniai: 21 (63,6%) asmeniui – 3a laipsnio, 10-iai (30,3%) – 3b laipsnio ir dviem (6,1%) – 3c laipsnio pagal Marsh klasifikaciją. Sergantieji osteoartritu taip pat turėjo nedidelio laipsnio plonosios žarnos gleivinės pažeidimus, tai yra 3 (66,6%) – 3a laipsnio ir vienas (33,6%) – 3b laipsnio pagal Marsh klasifikaciją.

Sergantiesiems reumatoidiniu artritu statistiškai reikšmingai ($p < 0,0001$) dažniau nustatyti IgA klasės antigliadino antikūnai (25,9%) negu sveikiems asmenims (2,3%) ir sergantiesiems virškinimo trakto ligomis (remisijos fazė) (5,7%).

Tarp sergančiųjų gliutenine enteropatija, asocijuota su reumatoidiniu artritu, vyravo 3a laipsnio plonosios žarnos pažeidimas (63,6%); tarp sergančiųjų gliutenine enteropatija, asocijuota su osteoartritu, taip pat vyravo 3a laipsnio plonosios žarnos pažeidimas (66,6%).

Išvada. Mūsų tyrimas patvirtina, kad yra tikslinga vykdyti sergančiųjų reumatoidiniu artritu ir kitomis autoimuninėmis ligomis atrankinį tyrimą.

Raktažodžiai: gliuteninė enteropatija, seruminiai žymenys, morfologiniai pokyčiai