Quantitative assessment of nutrition and nutritional status of patients with Crohn’s disease aged 13–18

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ABSTRACT

Introduction: Crohn’s disease (CD) is classified as an inflammatory bowel disease, with a recent increased incidence in developed countries. Treatment is based on pharmacotherapy and nutritional support. Patients in the exacerbation phase are particularly exposed to the development of malnutrition, which in the case of children may result in the inhibition of growth or delay in puberty. For these reasons, nutrition is very important in the treatment process. In the remission phase of the disease, the diet should be consistent with the recommendations for the population of healthy people in order to prevent the formation of vitamin and mineral deficiencies. The aim of the study was to determine the nutritional status of adolescent CD patients and to analyze their diet.

Materials and methods: The study was conducted on 14 children with CD, 6 girls and 8 boys with an average age of 16.25 ± 1.65 years. Their nutrition was analyzed based on information obtained from a nutritional history collected over 3 days. The collected information was entered into the Diet 5 software. The results of the menus were compared to current dietary norms. The nutritional status of the children was based on a body mass index (BMI) analysis with reference to OLA and OLAF percentiles. The additional results of morphology and the levels of sodium, potassium, total calcium and 25(OH)D from their blood were compared. All obtained results were analysed statistically using Statistica v12.0 software.

Results: The nutritional status of both the boys and the girls was normal, however the energy consumption was low (2274.83 ± 475.93 kcal and 1843.33 ± 258.4 kcal). Protein consumption was high, at 86.44 ± 29.57 g and 62.36 ± 26.51 g. In both groups, the levels of saturated fatty acids were too high (36.38 ± 14.45, 23.02 ± 9.53 mg), and in the boys’ group cholesterol was too high (427.41 ± 278.3 mg). Both sexes consumed insufficient amounts of fiber (7.32 ± 7.63 g, 19.84 ± 4.85 g) and omega-3 (1.8 ± 0.99 g, 17.4 ± 0.89 g). Iron, copper, iodine and zinc were consumed at the appropriate level. Both sexes consumed too little calcium (586.44 ± 458.11 mg, 742.47 ± 515.37 mg), potassium (2892.96 ± 2223.79 mg, 2901.62 ± 1028.56 mg) and magnesium (283.45 ± 145.26 mg, 276.71 ± 163.32 mg). Folate consumption was too low (256.44 ± 81.03 μg, 231.07 ± 81.03 μg), vitamin D (2.85 ± 1.00 μg, 0.96 ± 0.68 μg) and also vitamin E in the group of girls (7.02 mg ± 1.46 g). The blood parameters did not differ significantly between the sexes, and the concentration of 25(OH)D was within the lower limit of the norm (28.17 ± 5.91 ng/dL, 22.60 ± 3.38 ng/dL).

Conclusions: Low energy intake may adversely affect the nutritional status of CD patients. A deficiency in the diet of n-3 acids may promote the development of inflammation. Insufficient intake of calcium and vitamin D can disrupt the development of the skeletal system. The insufficient intake of dietary fiber can lead to constipation. A too low vitamin D intake and low blood levels of its metabolite indicate the need for supplementation. Additional supplementation of potassium and magnesium should be taken into consideration in the nutrition of CD patients.

Keywords: nutritional status; children; vitamin D; Crohn’s disease; inflammatory bowel disease.

INTRODUCTION

Inflammatory diseases of the intestines, such as Crohn’s disease (CD) and ulcerative colitis, are a growing challenge due to increasing morbidity. By 2015, 6155 patients had registered in the Polish Nationwide Crohn’s Disease Registry [1]. The disease can occur in any part of the digestive tract, initially in the form of an inflammation of the mucous membrane, spreading to the remaining layers of the gastrointestinal tract as the disease progresses [2].

The etiopathogenesis of CD is not fully understood yet. It is recognized that the disease is influenced by genetic, immunological, environmental and intestinal microbiota [3, 4]. The biggest genetic factor is polymorphism within the NOD2/CARD15 gene on chromosome 16. If CD is present in the closest family members, then the risk of its occurrence is 4 or even 20 times higher [5]. The most important environmental factors include smoking, environmental pollution, bacterial and viral infections, as well as incorrect nutrition. A diet rich in saturated fatty acids (SFA), refined sugar, and with a low content of fiber and omega-3 acids also increases the risk of CD [6]. In addition, studies indicate the adverse effect of processed foods on intestinal homeostasis. Popular food additives such as maltodextrin, carrageenan or xanthan gum may cause unfavorable changes in the composition of intestinal microbiota and thus contribute to inflammation of the gut [7].

Depending on the location and extent of the lesions, patients may report dysphagia if the inflammatory process includes the esophagus. Stomach pain, nausea and vomiting most often mean localization within the stomach and duodenum. Changes
in the lower part of the gastrointestinal tract may cause diarrhea and colic pain associated with fever [8]. Symptoms in children do not differ from those of adults, including the classic triad of abdominal pain, weight loss and diarrhea. However, this group of patients should be given special attention because of the low body mass and height in 80% of patients. In addition, girls experience a delay in puberty, and all pediatric patients may experience skeletal disorders and osteoporosis [9].

Diagnosis of CD is difficult due to its similarity to other diseases, and is based on endoscopic, imaging, laboratory and clinical imaging. Laboratory tests are used to determine blood morphology, albumin, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and iron and vitamin levels. However, these tests are mainly used in the disease monitoring process than for diagnostic purposes.

Treatment of CD includes both pharmacological preparations and proper nutrition. In the pediatric group, achieving remission of the disease is particularly important due to the reduced risk of developing complications and a reduction in the number of hospitalizations. In addition, in this group of patients, the restoration of growth and sexual maturation is equally important [10]. In children, the first-line treatment is exclusive enteral nutrition (EEN). Studies show that this therapy is 73% effective and in the case of corticosteroid treatment, the effectiveness is 71%. The most probable enteral nutrition (EN) mechanism consists in “resting” the intestines, facilitating the healing of the mucosa and the reorganization of intestinal microbiota [9]. Further treatment involves the use of glucocorticoids with a strong anti-inflammatory effect, 5-aminosalicylic acid derivatives, purine analogs as immunosuppressive therapy and antibiotics [11]. In recent years, biological treatments are based on the supply of antibodies obtained by genetic recombination [10].

Nutrition plays a key role next to pharmacological therapy. During exacerbation of the disease, 20% to even 85% of patients develop malnutrition [11]. The reasons for such development include a loss of appetite, exclusion of certain foods that cause pain, and anorexia [12]. In addition, inflammation, damage to the intestinal villi, or the formation of fistulas in the intestine, inhibit proper absorption of food, which may lead to deficiencies of iron, magnesium, calcium, zinc, potassium, and vitamins A, B, C, D and K [13]. In the course of the disease, a temporary lactose intolerance is also observed. The exclusion of cow milk and milk drinks from the diet aggravates the lack of calcium or vitamin D₃, which is why this group of patients is particularly exposed to the development of osteoporosis. Iron deficiencies may lead to anemia and poor energy, protein and other nutrients, inhibit weight gain, growth, sexual and intellectual maturation in pediatric patients [14].

In further stages, when oral nutrition is possible, an easily digestible diet with a reduction in fat, dietary fiber with an increased amount of protein should be entered. It is crucial that the diet is adjusted individually to each patient, respecting any tolerance to lactose or fructose. In addition, the diet should contain appropriate amounts of minerals and vitamins, and also be enriched with calcium and vitamin D₃ supplements. In the remission period, the diet should be gradually extended because during this period the nutrition should not differ from the diet of healthy people [1].

**MATERIALS AND METHODS**

The study group consisted of 14 patients diagnosed with CD, under the supervision of the gastroenterological counseling center at the Independent Public Clinical Hospital No. 1 of the Pomeranian Medical University in Szczecin. The study comprised 8 boys and 6 girls, aged 13–18 years. Patient diagnosis included endoscopic and radiologic examination and evaluation of histopathological sections – performed during their first diagnosis. The patients were in clinical remission as ascertained by the attending physician when study was performed. The average duration of CD in the studied group was 5 years (min. duration of illness was 1 year). Patients did not receive Modulen therapy or EN. The aim of the study was to determine the nutritional status and nutrient intake in the diet in the children in CD remission phase.

Among the patients and their parents/carers, a questionnaire was carried out covering information about the disease, medications and dietary supplements, as well as physical activity. In addition, a nutritional interview over 3 days was carried out, using the “Album of portions of products and dishes” [17]. Data obtained from the interview were introduced and analyzed using the Diet 5 software package, recommended by the Food and Nutrition Institute. Thanks to this, information was obtained on the amount of energy consumed, proteins, fats, carbohydrates and nutrients. These data were then compared to the currently valid Nutrition Standards for the 2017 Polish Population [18]. The nutritional status of patients was analyzed in relation to BMI applied to OLA and OLAF growth charts. In addition, the patient’s blood results were analyzed. For this purpose, the results of blood counts and the level of potassium, sodium, total calcium and 25(OH)D in the serum were used. All obtained results were subjected to statistical analysis using the Statistica v12.0 software (Statsoft, Tulsa, Oklahoma). The Student’s t-test was used for samples independent of variables.

**RESULTS**

The average age of the group was 16.25 ± 1.65 years, average body weight was 58.35 ± 17.18 kg and average height was 170.25 ± 11.11 cm. The anthropometric data of the groups are shown in Table 1.
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TABLE 1. Characteristics of the studied boys and girls

| Parameters       | Average of group | Average of boys (n = 8) | Average of girls (n = 6) |
|------------------|------------------|------------------------|-------------------------|
| Age (years)      | 16.25 ±1.65      | 16.25 ±1.83            | 16.25 ±1.50             |
| Body weight (kg) | 58.35 ±17.18     | 62.91 ±19.16           | 49.25 ±7.80             |
| Height (cm)      | 170.25 ±11.11    | 172.93 ±12.25          | 164.87 ±6.68            |

The questionnaire sought information on the medicines and dietary supplements taken by the patients. Over 80% of respondents were supplemented with vitamin D₃, over 60% with potassium ions, about 40% with calcium carbonate (Fig. 1). In most cases, they consumed immunosuppressants and 5-aminosalicylic acid derivatives, and less than 20% consumed glucocorticoids (Fig. 2).

Analysis of the nutrition status based on BMI showed that the boys were well nourished – average BMI was at the 50th percentile. However, after applying BMI to the growth chart it is shown that only 3 had a normal body mass, 3 were underweight, 1 oscillated between the border of the 50th percentile and overweight, and 1 was above the 95th percentile (Fig. 1). In the group of girls, the average BMI was at the lower limit of the norm. Three girls had normal body weight, and 1 was severely underweight (Fig. 3).

The content of individual ingredients in the diet varied between the girls and boys. For both, the energy consumption was insufficient. The dietary content of protein, sucrose and SFA was too high. The boys ate too much cholesterol. There was too little intake of omega-3 fatty acids and fiber. Consumption of carbohydrates and fat was at normal values (Fig. 4).

The intake of phosphorus, zinc, iron, iodine and copper was at normal values. Vitamins B₁, B₂ and B₁₂ were consumed in correct amounts. Too much vitamin A was consumed by both groups, and pyridoxine and niacin by the boys (Tab. 2).

The patients consumed too little calcium, potassium and magnesium. The girls consumed a significantly lower amount of vitamin E and D₃ than the boys. And for both sexes, the consumption of vitamin D₃ was abnormally low compared to nutritional standards, with the consumption also insufficient for folate. The consumption of vitamin C by the boys was twice lower than by the girls (Tab. 2).

In the case of the blood, morphology and biochemical tests, no significant differences were found between the sexes.
TABLE 2. Comparison of energy, protein, fat, carbohydrate, mineral and vitamin intake between the boys and girls

| Ingredient                          | Norm EAR | Average intake boys (n = 8) | Average intake girls (n = 6) | p   |
|-------------------------------------|----------|-----------------------------|-----------------------------|-----|
| Energy (kcal)                       | 2500/3400* | 2274.830 ±75.932            | 1843.333 ±258.447           | NS  |
| Protein (g)                         | 44/54*   | 86.448 ±29.575              | 62.361 ±26.513              | NS  |
| Animal protein (g)                  | –        | 57.144 ±26.618              | 40.314 ±20.017              | NS  |
| Plant protein (g)                   | –        | 26.375 ±7.922               | 21.506 ±7.559               | NS  |
| Fat (g)                             | 48–86/67–117* | 80.635 ±31.557             | 58.830 ±19.536              | NS  |
| Saturated fatty acids (g)           | 13.9–16.7/19.3–23.1* | 30.380 ±14.458             | 23.024 ±13.536              | NS  |
| Monounsaturated fatty acids (g)     | –        | 31.904 ±14.715              | 21.258 ±7.638               | NS  |
| Linolenic acid (g)                  | –        | 8.387 ±3.638                | 8.102 ±4.264                | NS  |
| α-linolenic acid (g)                | –        | 1.735 ±0.939                | 1.702 ±0.854                | NS  |
| EPA acid (g)                        | –        | 0.0087 ±0.009               | 0.0142 ±0.012               | NS  |
| DHA acid (g)                        | –        | 0.0635 ±0.050               | 0.0337 ±0.024               | NS  |
| Polysaturated fatty acids (g)       | –        | 10.453 ±6.444               | 9.907 ±5.001                | NS  |
| Cholesterol (mg)                    | to 300   | 472.426 ±278.31             | 192.780 ±99.733             | NS  |
| Carbohydrates (g)                   | 130*     | 315.379 ±77.808             | 283.995 ±55.527             | NS  |
| Saccharose (g)                      | 60       | 92.837 ±81.803              | 86.966 ±53.247              | NS  |
| Lactose (g)                         | –        | 5.367 ±7.292                | 9.021 ±6.012                | NS  |
| Starch (g)                          | –        | 149.984 ±66.341             | 116.028 ±23.359             | NS  |
| Digestible carbohydrates (g)        | –        | 294.617 ±85.186             | 264.165 ±50.827             | NS  |
| Fiber (g)                           | 21       | 17.328 ±7.638               | 19.843 ±4.858               | NS  |
| Ash (g)                             | –        | 15.275 ±6.184               | 12.929 ±5.422               | NS  |
| Sodium (mg)                         | 1500**   | 3076.2 ±1206.3              | 2143.665 ±1099.65           | NS  |
| Potassium (mg)                      | 3500**   | 2892.96 ±1223.7             | 2901.627 ±1028.56           | NS  |
| Calcium (mg)                        | 1100     | 586.44 ±458.112             | 762.471 ±515.371            | NS  |
| Phosphorus (mg)                     | 1050     | 1320.207 ±437.8             | 1088.699 ±581.561           | NS  |
| Magnesium (mg)                      | 300/340* | 283.460 ±145.26             | 276.712 ±163.325            | NS  |
| Iron (mg)                           | 8        | 10.248 ±2.634               | 8.856 ±3.799                | NS  |
| Zinc (mg)                           | 7.3/8.5* | 9.576 ±2.913                | 8.376 ±4.124                | NS  |
| Copper (mg)                         | 0.7      | 0.978 ±0.322                | 1.133 ±0.555                | NS  |
| Manganese (mg)                      | 1.6/2.2*; ** | 5.181 ±2.673               | 4.179 ±2.931                | NS  |
| Iodine (µg)                         | 95       | 111.995 ±80.583             | 98.477 ±33.861              | NS  |
| Vitamin A                           | 490/630* | 1734.62 ±1124.2             | 2184.428 ±1264.940          | NS  |
| Vitamin E (mg)                      | 8/10*    | 10.530 ±2.531               | 7.0215 ±1.463               | 0.029 |
| Tiamin (mg)                         | 0.9/1*   | 1.275 ±0.753                | 0.933 ±0.548                | NS  |
| Riboflavin (mg)                     | 0.9/1.1  | 1.607 ±0.707                | 1.415 ±0.628                | NS  |
| Niacin (mg)                         | 11/12*   | 23.070 ±12.999              | 12.965 ±9.284               | NS  |
| Vitamin B6 (mg)                     | 1/1.1*   | 2.007 ±1.052                | 1.951 ±1.398                | NS  |
| Vitamin C (mg)                      | 55/65*   | 58.220 ±42.877              | 113.529 ±47457              | 0.068 |
| Folic acid (µg)                     | 330      | 256.445 ±81.026             | 231.074 ±81.037             | NS  |
| Vitamin B12 (µg)                    | 2        | 2.937 ±2.102                | 2.606 ±0.652                | NS  |
| Vitamin D (µg)                      | 15**     | 2.855975 ±1.002             | 0.964250 ±0.688             | 0.0072 |

* girls/boys; **Al – adequate intake; DHA – docosahexaenoic acid; EPA – eicosapentaenoic acid; EAR – estimated average requirement

Although the boys had higher values, hematocrit was low for both sexes, as was hemoglobin in the girls. Monocytes were also slightly elevated in the girls (Tab. 3). The concentration of 25(OH)D for both sexes was at the lower limit of the normal range, while the sodium level in the girls oscillated around the min. level (Tab. 4).
TABLE 3. Comparison of morphology between the boys and girls

| Parameter                          | Reference values   | Average boys (n = 8)     | Average girls (n = 6)    | p    |
|------------------------------------|--------------------|-------------------------|--------------------------|------|
| Leukocytes (10^3/µL)               | 4.23–9.07          | 7.830 ±3.359            | 4.822 ±1.736             | NS   |
| Erythrocytes (10^6/µL)             | 4.63–6.08          | 4.928 ±0.295            | 4.617 ±0.449             | NS   |
| Hemoglobin (g/dL)                  | 13.7–17.5          | 13.7875 ±1.479          | 13.350 ±0.443            | NS   |
| Hematocrit (%)                     | 40.1–51.0          | 39.625 ±3.070           | 38.800 ±0.989            | NS   |
| Mean Corpuscular Volume (fl)       | 79.0–92.2          | 80.725 ±8.619           | 84.550 ±7.345            | NS   |
| Mean Corpuscular Hemoglobin (pg)   | 25.5–32.2          | 28.125 ±3.902           | 29.075 ±2.728            | NS   |
| Mean Corpuscular Hemoglobin        |                    |                         |                          |      |
| Concentration (g/dL)               | 32.3–36.5          | 34.737 ±1.291           | 34.425 ±0.590            | NS   |
| Platelets (10^3/µL)                | 150.0–400.0        | 328.500 ±104.946        | 269.00 ±89.327           | NS   |
| Red Cell Distribution Width (%)    | 11.6–14.4          | 13.900 ±1.117           | 13.900 ±0.734            | NS   |
| Mean Platelet Volume (fl)          | 9.4–12.6           | 10.312 ±0.842           | 9.700 ±0.909             | NS   |
| Platelet Large Cell Ratio (%)      | 19.2–47.0          | 26.725 ±6.944           | 21.500 ±8.007            | NS   |
| Procalcitonin (%)                  | 0.17–0.35          | 0.332 ±0.093            | 0.2550 ±0.075            | NS   |
| Neutrophils (%)                    | 4.0–8.00           | 5.590 ±12.83            | 6.175 ±11.227            | NS   |
| Lymphocytes (%)                    | 20.0–40.0          | 29.862 ±11.267          | 22.525 ±11.386           | NS   |
| Monocytes (%)                      | 2.0–10.0           | 10.037 ±3.243           | 13.550 ±3.382            | NS   |

TABLE 4. Comparison of electrolyte concentration and vitamin D between the boys and girls

| Parameter                          | Reference values   | Average boys (n = 8)     | Average girls (n = 6)    |
|------------------------------------|--------------------|-------------------------|--------------------------|
| Sodium (mmol/L)                    | 135–145            | 140.375 ±1.922          | 138.750 ±0.957           |
| Potassium (mmol/L)                 | 3.50–5.50          | 4.489 ±0.283            | 4.090 ±0.404             |
| Calcium (mmol/L)                   | 2.09–2.54          | 2.394 ±0.090            | 2.278 ±0.099             |
| Vitamin D 25(OH) (ng/mL)           | 20–60              | 28.171 ±5.917           | 22.600 ±7.034            |

DISCUSSION

This study was limited by the relatively low number of patients with CD disease in the 13–18 age group studied. Therefore, the discussion focuses on analyzing intake against current nutrition standards without addressing gender differences.

ECCO/ESPGHAN guidelines recommend EEN as a first-line treatment to induce remission in children with active CD. The duration of such a treatment is usually 6–8 weeks. The reintroduction of usual foods should not occur abruptly at the end of EEN treatment. It is suggested to gradually reintroduce foods while reducing the volume of the mix every 2–3 days over a period of 2–3 weeks [19].

Malnutrition in pediatric patients with CD is common in those newly diagnosed, but it can remain despite enteral treatment. Poor nutrition in children with inflammatory bowel disease (IBD) can cause growth disorders [20]. Aurangzeb et al. and D’Souza et al. showed that children suffering from CD have a lower BMI compared to the healthy controls [21, 22]. Our own research showed the appropriate BMI in both sexes. Long et al. pay attention to the growing problem of overweight and obesity also among patients with CD [23].

The energy intake was too low in relation to the norm, other studies also show lower intake of calories in such a group of children, and a low intake reduces body mass [24, 25]. According to ESPEN guidelines, demand for energy is different depending on the course of CD. In remission, energy intake should be the same as in the healthy population [26].

In this study, the intake of protein by CD patients was too high in relation to current nutrition standards for healthy people. Research by Pons et al. indicates an optimal dietary intake of protein, while Hartman et al., to the contrary, promote an increased supply in the diet [24, 27]. Protein intake should be higher in active phases of IBD, but in remission intake should be similar to healthy population: 1 g/kg/day. In the group of children in our study a lower lean body mass and increased risk of obesity over time. Steroid treatment can especially cause a higher loss of protein in children. Anthropometry provides insights into which patients develop relative deficiencies in lean body mass and would therefore benefit from nutritional supplementation. There is no good evidence that the daily protein requirements of IBD patients should differ from those of the healthy controls [26].

Insufficient consumption of carbohydrates in CD patients has been shown in many studies [24, 27, 28]. Our study showed a higher intake of carbohydrates in the diet. Guerreiro et al. emphasize too high a share of carbohydrates in the diet [29]. Researchers also report an excessive intake of simple carbohydrates [30]. Opstelten et al. show a higher intake of carbohydrates but lower fiber consumption compared to healthy controls [31]. This may confirm the higher intake of simple sugars in this group of patients. Attention should be paid to the...
wide range of the norm for fat consumption and the norm for carbohydrates, which refer only to the min. amount. The caloric value of the diet for both sexes in a given age range is specific, not a range, even with an adequate supply of fats, carbohydrates and an excessive protein intake, the caloric value of the diet may be too low as observed in our own study. According to Pons et al., patients with CD consume too little dietary fiber, which correlates with the results in this work [24]. Studies by Hartman et al. also indicate deficiencies of fiber in the diet [27]. It is believed that a high fiber diet has a positive effect on gastrointestinal function in CD patients [32] and 24.3 g/day of dietary fiber has been shown to reduce the risk of CD development by 40% [33]. In addition, it is observed that with a lower intake of dietary fiber, sucrose intake is higher. Sakamoto et al. points out a too high sucrose intake in CD patients [30].

The studied test group consumed an appropriate amount of fat. Other studies indicate a reduced intake of this nutrient, but special attention should be paid to the low levels of omega-3 fatty acid [28]. A Dutch study showed a lower intake of unsaturated fats in patients with CD than in healthy controls [31]. ECCO/ESPGHAN guidelines do not recommend omega-3 supplementation in remission [19].

Patients with IBD can develop microelement deficiencies due to diarrhea and a low dietary intake. Deficits can occur even in seemingly well-fed patients. These observations emphasize the need for routine monitoring (maybe every year) to detect deficiencies. A daily multivitamin supplement may correct most deficiencies, but is not a guarantee of adequacy, even in the long run. In the current study over 80% of the children supplemented with vitamin D, over 60% with potassium and 40% with calcium [26]. Consumption in the diet of minerals such as phosphorus, copper, iodine, iron and zinc was normal. Other studies indicate a higher intake of zinc and iron, but this was due to additional supplementation and not the diet itself [28]. The results by Pons et al. show a higher intake of zinc and a correct intake of iron [24]. In contrast, a cross-sectional study on 62 patients with CD showed an insufficient intake of calcium, zinc and magnesium [34].

The deficiency in the diet concerning calcium, zinc and magnesium was obtained by other researchers [24, 27]. A low intake of calcium and zinc as well as glucocorticoid use are risk factors of bone tissue loss in CD patients [34]. The reason for the deficiency of calcium in the diet is seen from the elimination of milk and milk products, as a result of lactose intolerance. It should be mentioned that the intolerance of this disaccharide in patients with IBD is not higher compared to the healthy population [35].

The intake of vitamin E in the boys’ group was normal but in the group of girls was significantly lower and below the norm. The results of Pons et al. show a lower dietary intake of vitamin E in CD patients, too much vitamin A consumed, and a lower intake of retinol [24]. In our study, correct contents in the diet were observed for vitamins C, B1, B2 and B12, similarly to Hartman et al. [27].

The level of 25(OH)D was within the lower limit of the normal range among the studied children with CD. In other studies, similar results are observed and in some cases serum levels are even lower [27, 36, 37]. The morphological parameters of the blood were mostly normal, with the exception of low hemoglobin and hematocrit, and elevated monocyte counts in girls. Studies by Cho and Yang also indicate reduced parameters of hemoglobin and hematocrit in newly diagnosed patients [38]. Elevated monocytes were also observed in the study by Szczuko et al. [39]. It should be noted that monocytes and activated lymphocytes are cells responsible for the production of proinflammatory cytokine TNF-α, which contributes to the development of inflammation in the gastrointestinal tract [10].

CONCLUSIONS

1. Too low a caloric intake, along with the disease, contributes to the abnormal nutritional status of patients with CD. Crohn’s disease does not exclude the possibility of overweight and obesity among patients.

2. Insufficient intake of fiber can lead to constipation and changes in intestinal microbiota. More SFA than polyunsaturated fatty acids (PUFA) may exacerbate inflammation through the production of proinflammatory mediators. A low intake of antioxidant vitamins E and C may result in reduced protection against oxidative stress, which is increased in the disease.

3. Deficiencies in the diet of vitamin D3 and calcium may lead to abnormal mineralization of bone tissue and increase the risk of osteopenia and osteoporosis.

4. In remission, magnesium and potassium supplementation should be considered.

LIMITATIONS

Due to the limited number of children with CD that were studied, it is necessary to perform additional research over a larger number of children.

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