

CROHN'S DISEASE AND DIETARY RECOMMENDATIONS

Eva Beňová¹, Mária Boledovičová², Kateřina Teplá³

¹Higher School of Health and Middle School of Health, Ústí nad Labem, Czech Republic

²University of South Bohemia in České Budějovice, Faculty of Health and Social Studies, Department of Nursing and Midwifery, České Budějovice, Czech Republic

³University of South Bohemia in České Budějovice, Faculty of Health and Social Studies, Department of Clinical and Preclinical Disciplines, České Budějovice, Czech Republic

Submitted: 2015-07-15

Accepted: 2015-10-16

Published online: 2015-12-31

Abstract

Crohn's disease is a chronic non-specific inflammatory bowel disease of any part of the digestive tract, which has a segmental character and digestive tract affects the wall transmurally in all layers. It is a disease of multifactorial etiology. One of the presumed causes of the disease is excessive immune reactions to physiologically occurring intestinal flora in genetically predisposed individuals. Incidence in the Czech Republic is around 4–6 new cases per 100,000 inhabitants per year. Clinical manifestations of the disease are varied and are dependent on localization, extent of disease and the nature of local inflammatory changes. The most common symptoms are diarrhea, abdominal pain and weight loss. The basis for the diagnosis of disease is colonoscopic examination. Treatment of Crohn's disease is symptomatic and it is a combination of conservative and surgical treatment. Nutritional therapy is a part of the conservative treatment. Nutritional counseling is one of the basic functions of nursing in which nurse holds important educational role in cooperation with the nutritional therapist. Good nutritional status has a positive effect on the overall condition of the patient who better cope with possible acute flare. Dietary recommendations have own specifics in the period of remission and in the period of relapse. Dietary measures are designed to protect the intestinal mucosa irritation. In the remission phase of disease essential dietary measures exist. Diet is no different from a rational diet, and it is very individual. The patient must self-identify foods without the risk of pain or diarrhea. In the relapse phase should be administered diet with low residue – it is necessary to completely eliminate insoluble fiber and hard to digest foods with high fat content. In the phase of relapse may be indicated by the severity of condition parenteral or enteral nutrition.

Key words: Crohn's disease; epidemiology; pathogenesis; dietary recommendations

INTRODUCTION

Crohn's disease (other names: regional enteritis, granulomatous enteritis, terminal ileitis) is a chronic unspecific inflammation of any part of the digestive tube, which has a segmental or plurisegmental character and affects

the digestive tube wall transmurally in all its layers (Dítě et al. 2010, Zbořil and Prokopová 2010). First written reports on the condition similar to Crohn's disease date back to 19th century. These were the diseases hidden behind the diagnosis of intestinal tuberculosis (Konečný and Ehrmann 2014). The disease was

first described by Crohn, Ginzberg and Oppenheimer in 1932, specifically at the site of its typical location – ileocecal location (Zbořil and Prokopová 2010). It is a chronic disease with periods of worsening (relapses) and calming (remissions) of the inflammation that is medicamentously and surgically incurable (Dítě et al. 2010). Crohn's disease occurs particularly at a younger age. Its incidence in CR ranges from 4 to 6 new cases per 100,000 persons per year; the incidence in Scandinavia and certain regions of North America is significantly higher (8–16/100,000 persons/year) (Dítě et al. 2010).

Clinical signs of the disease are various, being conditioned by the localisation and extent of the disease and by the character of local inflammatory changes. The disease can be in progress for a number of years with minute abdominal symptoms only, but also with significant extraintestinal symptomatology. Moreover, it can be manifested by unspecific symptoms only (e.g. weight loss, sideropenic anaemia and increased temperature). The picture of the disease is substantially different in various segments of the digestive tube and we can therefore differentiate several forms of the disease depending on its localisation (Dítě et al. 2010). Regarding the clinical picture, we differentiate the course and development, shape or form, activity and severity and complications and extraintestinal symptoms (Dítě et al. 2007, Zbořil and Prokopová 2010). The diagnostics of Crohn's disease is based on its clinical picture, laboratory tests, endoscopic and histological findings as well as on the results of imaging examinations. The treatment lies in the combination of medicamentous, surgery and endoscopic therapy and diet therapy.

Epidemiology

Epidemiology of this disease is subordinated to continual changes. Unspecific inflammation – Crohn's disease – occurs particularly at a younger age; the median of the incidence ranges from 25 to 28 years. The onset of the symptoms early in the childhood or adolescence was observed in 20–25% patients. There are considerable geographical differences in the incidence of the disease. The incidence in Scandinavia and certain regions of North America ranges from 8 to 16 cases per 100 000 persons per year. The

incidence of the disease in economically developed countries is constantly increasing; mainly in the population of adolescents and older children. Current estimations of the prevalence of the disease in USA range from 1 to 1.5 million cases per year (Dítě et al. 2010, Baumgart 2012). The data on race differences are interesting. A higher ratio of the incidence of Crohn's disease was observed in African-American patients than in Hispanic Americans or Asians, in whom the disease occurred at a higher age, having shorter duration and less frequent presence in the family history (Baumgart 2012). At present, the incidence of the disease in CR ranges from 4 to 6 new cases per 100 000 persons per year. The difference between genders is minimal; women are affected a little more. The ethnical subgroup most affected by Inflammatory Bowel Disease (IBD) is that of the Jewish nation – the Ashkenazi – in whom the IBD-1 locus occurs on 16th Chromosome (gene NOD-2) (Dítě et al. 2010, Zbořil and Prokopová 2010).

Pathogenesis

Crohn's disease is chronic non-specific inflammation of any part of the digestive tube which has a segmental character, i.e. only certain segments are affected (skip lesions), alternating unaffected segments (skipped areas). The inflammation affects the wall of the digestive tube transmurally in all its layers, penetrating to the serose and adjacent nodes. An aphthous ulcer above the lymphatic follicle is a typical symptom. The hyperaemic erythematous mucosa is divided with deep fissures. The penetration of the fissures to the serosa causes formation of fistulas. It is possible to prove granulomas with Langhans-type cells, which never caseificate, in histological findings in many cases. Pathological changes can also be proved in microscopically normal segments (Jirásek 2002). The disease ranks among so called idiopathic Inflammatory Bowel Diseases (IBD). Current etiopathogenetic theory of IBD origin describes an inadequate reaction of the immune system to the physiological intestinal flora in genetically predisposed people. Important and modifying factors also include those from the outer environment. The genetic disposition has a 60–80% share in the etiology (Jirásek 2002, Lukáš 2012, 2014a).

Genetic factors

Genetic endowments for IBD emergence are a necessary condition of the development of the clinical symptoms of bowel inflammation in a person stigmatised in this way. For example, a similar mechanism is seen in celiac disease; its development is conditioned by the interaction between genes and the environment. At present, more than 160 IBD-associated gene loci are known, out of which 2/3 are involved in the development of Crohn's disease. The introduction of a new technique of genome testing – so called Genom-Wide Association (GWA) studies was fundamental contribution to the study of IBD genetic base; thank to these studies, a number of gene polymorphisms that could predict a risk of disease emergence and a therapeutic response were revealed (Hybenová et al. 2013, Lukáš 2014a).

Abnormal mucosal immunity

A failure in recognising of molecular markers of pathogenic and physiological intestinal microflora might be responsible for the development of chronic bowel inflammation. To maintain the intestinal mucosal barrier, antimicrobial peptides are important. Two groups of these peptides – cathelicidins and defensins – have important antimicrobial properties and communicate with the immune system. Defensins are produced particularly by Paneth cells (α -defensins) and colonocytes (β defensins). The expression of α -defensins is reduced in Crohn's disease, which is reflected in bacterial invasion into the mucosa. The main sites of the immune reaction in the bowel are Peyer's patches and lymph follicles. Specialised epithelial M cells transfer antigens into the lymphatic tissue where dendritic cells, responsible for the activation and proliferation of T and B lymphocytes, are activated. Thereafter, naïve T lymphocytes undergo differentiation into Th1, Th2 or regulatory T clones (Th3, Tr1 or CD 25⁺ CD4⁺). In this case, inflammation is mediated mainly by Th1 lymphocytes and their active substances (interleukins 12,17,23,27 and interferon γ). The ethiopathogenesis of the inflammations is, however, a substantially more complicated process and the shown scheme emphasizing Th1 and Th2 differentiation is a big simplification of the whole issue (Hindryckx and Laukens 2012, Lukáš 2014a).

Ethiopathogenesis

The key factors of outer environment, influencing the development and course of unspecific bowel inflammation, are primarily bacterial composition of the gastrointestinal system, psychical stress and smoking. A connection with vasculitis, rubeola infection and mycobacterial infection is also possible (Jirásek 2002, Lukáš 2014a). Although the infectious etiology of IBD emergence has not been confirmed yet, the presence of intestinal microbes itself is an important precondition of IBD development. A number of recent-years experiments confirmed the existence of so called dysbiosis of the intestinal microflora in the patients. These are changes in the composition and proportion of particular species of microbiots compared with healthy individuals. The presence of the representatives of *Firmicutes* family including *Lactobacilli* and *Clostridia* as well as the presence of *Bifidobacterium* family is noticeably reduced. An increased proportion is formed by *Proteobacteriae* and *Actinobacteriae*. Increased bacterial invasion into the mucous membrane is manifested by the increase in immunoglobulins formed against certain bacterial antigens. To distinguish Crohn's disease from Ulcer Colitis, the determination of anti-I2 antibodies (*Peptostreptococcus anaerobius*) or anti-purine antibody against *E. coli* membrane antigen (anti-OmpC) is, for example, used in clinical practice (Lyra and Lahtinen 2012, Yutao 2012, Lukáš 2014a). Psycho-neuro-immunologic studies brought very important proves. They suggest that "behavioural factors" (emotions and psychical stress) can significantly affect the immune system, specifically at the level of system response as well as local (mucosal) immune reaction. Unfavourable life circumstances are also included in factors stimulating disease emergence. Smoking is an important risk factor, as it causes an aggressive course of the disease, perforation complications and a worse response to immunosuppressive and biological therapy (Baumgart 2012, Lukáš 2014a). According to Baumgart (2012), breast-feeding is a protective factor of IBD emergence and development, likewise in celiac disease (Hybenová et al. 2013).

Diagnostics

The diagnosis of Crohn’s disease must be comprehensive, based on a thorough analysis of the anamnestic data that are focused on extraintestinal symptoms (uncertain febrile states, uncertain anaemia or weight loss). Accurate determination of the extent of digestive tube affection by means of radiodiagnostic methods (eneroclysis, MR enteroclysis) is of substantial significance. The assessment of the status includes the digestive tube wall, surrounding organs, mesentery, lymph nodes and retroperitoneum. At present, MR enteroclysis is a standard and least patient-burdening examination. Out of endoscopic methods, colonoscopic examination with terminal ileus examination followed by histomorphological evaluation of samples has a dominant position in the diagnostics of Crohn’s disease. It is used in the phase of diagnosis determination, at patient follow-up or therapeutic response monitoring and in the post-surgery period. Endoscopy can be used for the therapy of stenosis. In case of upper GIT affection, esophagogastroduodenoscopy is of the same importance. In exceptional cases, a modern method of capsule enteroscopy with a low informative value can be used. Standard examination of patients with Crohn’s disease in the segment of terminal ileum is based on ultrasound examination. The

laboratory diagnostics is focused on testing the inflammatory activity (sedimentation, C reactive protein and leucocyte and thrombocyte levels). The serological test of antibodies (anti-*Saccharomyces cerevisiae* (ASCA) and anti-neutrophil-cytoplasmic-antibodies (ANCA) as well as the determination of faecal calprotectin (Zbořil 2013, Konečný and Ehrmann 2014) are of significance in differential IBD diagnosis.

Clinical picture

The clinical symptoms of Crohn’s disease are very diverse and are conditioned by the extent and localisation of the disease as well as by the character of local inflammatory changes. The disease can only be present with minute abdominal symptoms for a number of years but also with significant extraintestinal symptomatology. The disease can also be manifested by unspecific symptoms only. The clinical pictures enables distinguishing its course and development, shape or form, activity and severity, complications and extraintestinal symptoms (Dítě et al. 2007, Zbořil and Prokopová 2010). From a view of its course, there can be a first attack, remission, relapse or reactivation of the disease. According to Greenstein’s classification, the patients are divided into 2 types by the disease development (Table 1).

Table 1. Course and development of Crohn’s disease (Zbořil and Prokopová, 2010)

Type		Characteristics
A	Agressive-fistulizing	Aggressive course, tendency to formation of fistulas and abscesses
B	Indolent-fibrosenotic	Less frequent attacks, tendency to stenosis formation
C	Inflammatory	Combination of groups A and B with fluctuating intensity

From a view of disease shape or form, ileitic and ileocolic forms are prevalent (30–40%); the perianal affection forms approx. 20% and the oral segments affection is rare (0.5–2%). Each of these affections has its particularities (Table 2).

To evaluate the disease activity, a clinical index is used, either the CDAI (Crohn’s Disease Activity Index or the Harvey-Bradshaw Index) (Freeman 2008). The occurrence of complications and extraintestinal symptoms is typical for this disease. The intestinal complications include intestinal obstruction, subileous and ileous conditions and formation

of fistulas or abscesses. The life-threatening complications include intestinal perforation and massive haemorrhage. The extraintestinal symptoms occur approx. in 1/3 patients. They are divided in symptoms connected with high inflammatory activity of the disease (skin, joint or ocular complications). They soothe as the intestinal inflammation is being managed. Another group of the extraintestinal complications includes coincident diseases independent on the activity of the intestinal inflammation; the therapy of the intestinal inflammation does not have any effect on them (Dítě et al. 2010).

Table 2. Symptoms of Crohn's disease forms (Dítě et al. 2010)

Form	Symptoms
Ileitic and ileocolic	Pain in right abdomen, diarrhoea, increased temperature, weight loss, bloating, flatulence, burping
Perianal (anorectal)	Formation of abscesses and fistulas (perianal, anovaginal, anorectal, anovulvar, rectovaginal, rectoscrotal, rectovesicular and entero-enteral)
Affection of large intestine	Diarrhoea, spasmodic abdominal pain dependent on emptying, tenesmus and rectal bleeding, purulent complications in perianal area
Affection of small intestine	Lack of appetite, bloating, burping, excessive gas, rumbling, signs of malnutrition, intermittent abdominal pain, weight loss, anaemia, malnutrition, growth retardation in children
Atypical location	Duodenum – weight loss, stomach – sense of fullness and pain after meal, nausea and vomiting oesophagus – dysphagia

Therapy of Crohn's disease

The therapy of Crohn's disease is a combination of conservative and surgical therapy. It is a symptomatic therapy, not a causal therapy. The conservative therapy is based on medicamentous treatment and nutritional therapy. The surgical solution is indicated in cases of localised disease that does not react to the medicamentous therapy or in case of the formation of intra-abdominal complications (stenosis, abscesses, fistulas, cancerisation and acute conditions – fulminant clinical course, haemorrhage, toxic megacolon or perforation). Surgical intervention is required in 70–90% patients in a certain phase of therapy. The endoscopic therapy is, for example, used in the dilation of intestinal stenoses (Dítě et al. 2007, Kala et al. 2014). The aim of the conservative therapy is inducing of remission, prevention of relapse and formation of complications and maintenance of long-term remission (Červený 2014). The therapy of the disease depends on its location, severity and complications. Thus, the therapeutic approach must be individualised regarding the symptomatic response and treatment tolerability (Kučela and Zakuciová 2012).

The medicaments for inducing of remission are aminosalicylates-mesalazine/sulphasalazine (they do not have a substantial effect on the disease, however) and corticoids. They are used in mild to moderately active Crohn's disease. Oral or parenteral corticoids are used in severally active disease. The biological therapy is used in complicated patients who do not respond to standard

therapy. However, it can be used as the first choice. It is the newest therapy with significant anti-inflammatory effect, based on the administration of monoclonal antibodies against the inflammatory cytokine TNF α [tumour necrosis factor (TNF α)]. At present, two formulations are registered – infliximab (for intravenous administration) and adalimumab (subcutaneous administration). A new biological formulation – vedolizumab – has an anti-inflammatory effect in the area of the gastrointestinal tract only; thus, it is not associated with the systemic immunosuppressive effect and a risk of infectious complications. The positive response (improvement of the state or inducing of remission in therapy-naïve patients) is reached in more than 90% patients. The remission of the disease is reached approx. in 60% patients. The so called primary non-response to biological therapy ranges from 10 to 15%. The secondary loss of the therapeutic response during long-term administration occurs in 25–30% patients who reacted to the therapy very well at first. The reason for potential failure of biological therapy after a certain period of time might be unknown etiology and incompletely known pathogenesis of the disease without a possibility of causal therapy, various and substantially different phenotypic IBD symptoms and different mechanisms of inflammation in a specific patient occurring during the disease lasting for a number of years (Lukáš 2009, 2011, 2012, 2013, 2014b). The maintenance therapy is based on the administration of mesalazine/sulfasalazine, immunosuppressants and, in

the recent years, also on biological therapy. A higher efficacy expressed by a higher number of remissions and mucosa healing has been proved for combination therapy (immunosuppressant + biological), yet with a higher incidence of adverse events (opportunistic infections) and undesirable effects of immunosuppressants. Antibiotics (nitroimidazole chemotherapeutics and quinolone antibiotics) are administered in cases of infectious or septic complications and after resection surgery (Dítě et al. 2007, Lukáš 2012, 2013, Červený 2014). Other medicaments include prebiotics and probiotics. Prebiotics are indigestible food components administered orally. They stimulate growth and activity of natural GIT microflora. Their representative is lactulose which is disintegrated by action of bacterial flora which results in pH reduction. The reduced pH in the gut induces the reduction of the growth of specific bacterial species; on the contrary, the numbers of *Lactobacilli* and *Bifidobacilli* increase. Probiotics – e.g. non-pathogenic *E. coli strain Nissle 1917* enhances the function of the intestinal barrier and modulates immune responses (Abdulmir et al. 2012, Červený 2014). Selected foreign studies also describe the use of adjuvant therapy and alternative medicine in patients with Crohn's disease (e.g. massages, physiotherapy, acupuncture, homeopathy or phytotherapy, vitamins and dietary supplements) (Bernstein 2015).

Nutritional therapy

The nutritional therapy in patients with Crohn's disease is part of conservative therapy and is as important as medicamentous or surgical therapies. A good nutritional state has a favourable effect on the general condition of the patient who copes better with potential acute flare-up (Zbořil 2015). Nutritional support and education of the patient on dietary measures and possibilities of artificial nutrition are inseparable parts of comprehensive therapy of a patient with this type of unspecific intestinal inflammation. The diet must meet the criteria of rational diet, respecting the patient's current state of health with regard to the grade and severity of malnutrition, phase of the disease and associated complications (Vrzalová et al. 2011). One of the basic functions of nursing is

also nutritional consulting, at which a nurse, cooperating with a nutritional therapist, holds an important educational function. The diet at this type of unspecific inflammation is specific in the way of the non-existence of any established dietary recommendations. An individual approach based on the tolerability of particular meals is preferred (Kohout 2004). It is necessary to count in the occurrence of negative nitrogen balance, hypoproteinaemia, protein malnutrition and episodes of diarrhoea with a risk of dehydration and ion dysbalance in the patients. There is also a usual problem of a lack of fat-soluble vitamins, iron and folates. Secondary lactose intolerance is also frequent (Novotná 2013a). The dietary measures are different in acute exacerbation (the relapse phase) and in a symptom-free period (the remission phase). The dietary measures are not substantial in idiopathic bowel inflammation in quiet periods. General recommendations are based on the intake of high quality proteins, restriction of the intake of saturated fats and sufficient amounts of vitamins and trace elements. It is necessary to avoid flatulent foods, spirits and also insoluble fibre. The recommendations include a sufficient intake of liquids (2–2.5L/day), food rich in calcium (cheese, yogurts, and cottage cheese), consumption of food with increased content of iron and vitamins and increased consumption of proteins. It is suitable to adjust energy recommendations individually; in general the diet should consist of 144–192kJ/kg/day and 1.2–1.5g protein/kg/day (Vrzalová et al. 2011). The food should be non-irritant; it is necessary to pay attention to small amounts of food divided in six portions a day. Individual tolerability of particular foods plays an important role. In general, legumes, raw vegetables, vegetable and fruit juices, citrus fruits, sauerkraut, onion, fatty and sour foods and milk are poorly tolerated. On the contrary, boiled meat, fish, poultry, potato, rice, pasta and easy-to-digest fruit and vegetables are well tolerated. It is necessary to avoid consumption of fibre if stenosis occurs on the gut. Balancing of the amounts of proteins, carbohydrates and fat with sufficient amounts of vitamins is also necessary. It is necessary to administer folic acid, vitamin B₁₂ and iron during the therapy with sulfasalazine, after resection in the area of terminal ileum and in chronic diarrhoea,

respectively (Zbořil 2015). The aim of the dietary measure in acute stage of the disease is reducing of burden of the bowel affected by disease. In the relapse phase, a so called low-residue diet must be administrated – complete avoiding of insoluble fibre and hard-to-digest foods with high contents of fat is necessary; reduction of the amount of soluble fibre is not required; adding of probiotics is appropriate. An easy-to-digest carbohydrate diet is preferred – with a high content of starches (pasta), increased content

of proteins, iron and calcium and a sufficient amount of vitamin C and B-complex – Table 3 (Šachlová 2011). It is recommended to separate the intake of solid food from the intake of liquids. Regarding technological preparation, the appropriate methods are boiling, braising, steaming and baking in a hot air oven. In patients with a stenotic form of Crohn's disease, it is necessary to administrate a low-residue diet even in the remission phase (Kohout 2011, Šachlová 2011).

Table 3. Appropriate and inappropriate foods in relapse period (Šachlová 2011)

	Appropriate	Inappropriate
Meat	Turkey, rabbit, chicken, veal, lean pork	Marinated meat, smoked goods, oily fish and fat pork
Smoked goods	Lean ham, children's ham, ham mousse	Intensely spiced and fat smoked goods
Diary	Fermented products, cottage cheese, natural cheese, yoghurt milk with probiotics	Fresh milk, blue cheese, aromatic cheese
Bakery products	White bread, white bakery products, sponge biscuits	Freshly leavened, whole-meal
Side dishes	Potato, mashed potato, gnocchi, rice, pasta	Leavened dumplings, fried side dishes
Drinks	Still water, weak fruit tea, diluted fruit juice	Alcohol, strong coffee, strong tea, hot chocolate
Spice Condiments	Green herbs, little salted foods	Spiced foods, meals with sodium glutamate
Fruit Vegetables	Diluted fruit juice, vegetable purée, mashed fruit	Fresh fruit and vegetables, seeds, husks, nuts

In the relapse phase, parenteral or enteral nutrition can be indicated for a period of several days or weeks until the symptoms disappear, depending on the severity of the condition.

The enteral nutrition (EN) is based on the administration of pharmaceutically prepared nutritive solutions into the digestive tract that are intended for sipping or for administration by probe into the stomach or small intestine. The easiest method of the application of enteral nutrition is oral administration in a form of sipping. The correct use of this method lies in sipping the nutrition in the intervals between meals, ideally cooled, in small portions (up 50ml/4times daily) at the beginning and observing individual tolerability (Šachlová 2011). Another way of the administration of enteral nutrition is percutaneous endoscopic gastrostomy (PEG), intended for long-term application.

For temporary administration of enteral nutrition, the application of pharmaceutically prepared formulations by jejunal probe is preferred; they are applied by enteral pump (accurate dosage in mL/hour secures better tolerance of the nutrition). This method is performed in selected patients who are able to cooperate, are properly informed and have signed the informed consent. The insertion of the nasojejunal probe and education on the use of enteral pump is performed at short hospitalisation during which the patient's reaction to artificial nutrition is evaluated and its dosage is established. The patient is informed about the care of the probe (washing, fixation exchange, hygiene) and receives a phone contact on the nutrition team securing the therapy together with a gastroenterologist. This method is used advantageously just in home care. The enteral nutrition is applied either during 12 hours at night or continuously

for 20 or more hours daily. The feeds for enteral nutrition and sets are prescribed by a physician licensed for artificial nutrition and intensive metabolic care. There are polymeric, hypercaloric, modified or oligomeric feeds at disposal. Their spectre is supplemented with module dietetics to enrich either oral dieter EN formulations. There are various dietary supplements based on milk protein or enzyme modified starches or amino acid formulations with glutamine precursors that have also immunomodulative effect (Lochs et al. 2006, Kohout 2011, Novotná 2013b). Parenteral nutrition (PN) is part of the therapy during relapse, in perioperative period, in severe form of malnutrition and in the occurrence of intestinal complications. It is administrated into the central or peripheral vascular bed in dependence on the severity of patient's state and presumed duration of its application. At present, bags in "all in one" system are used for PN, containing fats, carbohydrates, amino acids, vitamins, minerals and trace elements. This system secures better utilisation of

nutrients, lower occurrence of metabolic complications and lower risk of infection (Van Gossum et al. 2009, Vrzalová et al. 2011, Novotná 2013c). This type of nutrition can be administrated even in home environment in long-term therapy. Regarding increased loss of water in diarrhoea, fluid intake is important. The following return to normal diet must be gradual. Parenteral/enteral nutrition together with easy-to-digest food (tea, white bakery products) is primarily administrated. Boiled vegetables, potato, rice, pasta, lean meat, low-fat cheese, poultry and fish are subsequently added, depending on the tolerance. Afterwards, this diet is followed with the switching to normal diet; the patient must avoid food and ways of preparation that trigger undesirable symptoms just in him/her (Zbořil 2015). The diet in remission phase does not differ from rational diet (or from the diabetic diet in patients with diabetes); the diet regimen is completely individual. The patient himself/herself must select foods without a risk of pain or diarrhoea – Table 4 (Šachlová

Table 4. Appropriate and inappropriate foods in remission period (Šachlová 2011)

	Appropriate	Inappropriate
Meat	Lean, poultry, fish with skin removed	Fatty, fried, smoked, marinated meat, pork scratchings, bacon, spicy cans
Smoked goods	Turkey and rabbit roulades, ham	Hot sausages
Soups	Vegetable and fat-free broths	Legume, fatty and spiced soups
Side dishes	Pasta, rice, everything from potatoes, dumplings with baking powder	Fried, leavened
Sauces	Tomato, chives, root vegetables sauces	Hot paprika, pepper, curry in sauce
Vegetables	Root vegetables, peeled tomatoes, spinach, young kohlrabi, squash	Mushrooms, cauliflower, broccoli, legumes
Fruits	Peeled fruit, apricots, peaches, fruit purée and mousse	Pears, fresh fruit with peels and grains, nuts and seeds
Dough	Croquembouche, semolina, whipped, sponge doughs	Leavened
Desserts	Less sweet and less fatty	To fatty and too sweet
Meatless meals	Fruit dumplings, charlotte, rice soufflé	Fried and fatty
Fat	Fresh butter, olive oil	Bacon, pork scratchings
Diary	Sour milk products, processed and firm cheeses, natural cottage cheese, drinks with probiotics	Fresh milk
Spices	Green haulms – celery, lovage, chives, parsley, dill; onion, garlic, mushroom and cumin broth, if tolerated	Hot spice

2011). However, it is still suitable to avoid bloating meals, spirits and fat meat in this phase of the disease. Drinking regime is also important (2–2.5L liquids/day). A frequent question of the patients refers to alcohol consumption (e.g. toast at various social events when they do not want to advertise their disease). Quality red wine (e.g. Bikavér) in a small amount (2dL at most) or quality cognac (0.5dL at most) can be recommended. The energy and protein intakes must cover the daily physical activity. Special dietary measures are required in patients after repeated small intestine resections. The diet must contain a sufficient amount of energy (144–192kJ/kg/day) and proteins (1.2–1.5g/kg/day). It is recommended to separate the administration of liquids and solid food and eat smaller portions. The reduction of lactose amount is also appropriate. Following the terminal ileum resection, the supplementation with vitamin B₁₂ might be necessary (Kohout 2011).

CONCLUSION

The dietary measures in Crohn's disease cannot influence the disease itself. However, the same emphasis is put on the nutritional therapy as on the medicamentous or surgical therapies. The state of the diet therefore affects patient's general condition and the way he/she copes with the disease. A dietary measure is a permanent part of the therapy. It has significance for the prevention of relapse and emergence of complications, for the maintenance of remission and in the perioperative period. Although there are no established dietary recommendations, the diet has its particularities in the phases of relapse as well as remission, unlike that in the diseases directly dependent on the diet. It includes the possibilities of enteral or parenteral nutrition. From a dietetic point of view, individual approach of a physician, nurse or nutritional therapist is always necessary.

CONFLICT OF INTEREST

The authors have no conflict of interest to disclose.

REFERENCES

1. Abdulmir AS, Zaman MZ, Hafidh RR, Bakar FA (2012). The Role of Diet, Prebiotic and Probiotic in the Development and Management of Inflammatory Bowel Disease. In: Karoui, S et al. *Inflammatory Bowel Disease – Advances in Pathogenesis and Management*. New York: Springer, pp. 250–274. [online] [cit. 2015-01-18]. Available from: <http://www.intechopen.com/books/inflammatory-bowel-disease-advances-in-pathogenesis-and-management/the-role-of-diet-and-probiotic-prebiotic-bacteria-in-the-development-protection-and-treatment-of-inf>
2. Baumgart DC (2012). *Crohn's Disease and Ulcerative Colitis. From Epidemiology and Immunology to a Rational Diagnostic and Therapeutic Approach*. New York: Springer.
3. Červený P (2014). Farmaceutická péče u pacientů s idiopatickými střevními záněty [Pharmaceutical care in patients with idiopathic intestinal inflammation]. *Prakt. lékař. 10/1*: 26–29 (Czech).
4. Bernstein CN (2015). Treatment of IBD: Where We Are and Where We Are Going. *Am J Gastroenterol. 110/1*: 114–126. [online] [cit. 2015-01-20]. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/25488896>
5. Dítě P. et al. (2007). *Vnitřní lékařství [Internal medicine]*. 2nd ed. Praha: Galén (Czech).
6. Dítě P, Lukáš M, Lata J (2010). Idiopatické střevní záněty [Idiopathic intestinal inflammation]. In: Češka R et al. *Interna [Internal medicine]*. Praha: Triton, p. 400–404 (Czech).
7. Freeman HJ (2008). Use of the Crohn's disease activity index in clinical trials of biological agents. *World J Gastroenterol. 14/26*: 4127–4130. [online] [cit. 2015-01-18]. Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2725371/>
8. Hindryckx P, Laukens D (2012). Intestinal Barrier Dysfunction: The Primary Driver of IBD? In: Karoui, S et al. *Inflammatory Bowel Disease – Advances in Pathogenesis and Management*. New York: Springer, pp. 24–40. [online] [cit. 2015-01-18]. Available from: <http://www.intechopen.com/>

- books/inflammatory-bowel-disease-advances-in-pathogenesis-and-management/intestinal-barrier-dysfunction-the-primary-driver-of-ibd-
9. Hybenová E, Štöfirová J, Mikulařová A (2013). Celiac disease and gluten-free diet. *Potravinářstvo*. 7/1: 95–100. [online] [cit. 2015-02-17]. Available from: <http://dx.doi.org/10.5219/276>
 10. Jirásek V (2002). Crohnova nemoc [Crohn's disease]. In: Jirásek V, Brodanová M, Mareček Z. *Gastroenterologie a hepatologie [Gastroenterology and hepatology]*. Praha: Galén, pp. 92–99 (Czech).
 11. Kala Z, Marek F, Válek V, Bartušek D (2014). Chirurgická léčba Crohnovy choroby [Surgical treatment of Crohn's disease]. *Vnitř Lék.* 60/7–8: 617–623 (Czech).
 12. Kohout P (2004). Výživa u pacientů s idiopatickými střevními záněty [Nutrition in patients with idiopathic intestinal inflammation]. Praha: Maxdorf (Czech).
 13. Kohout P (2011). Výživa u pacientů s idiopatickými střevními záněty [Nutrition in patients with idiopathic intestinal inflammation]. *Postgraduální med.* 13/2: 122–127 (Czech).
 14. Konečný M, Ehrmann J (2014). Pokroky v diagnostice a léčbě nespecifických střevních zánětů [Progress in diagnosis and treatment of non-specific intestinal inflammation]. *Vnitř Lék.* 60/7–8: 625–629 (Czech).
 15. Kužela L, Zakuciová M (2012). Racionálna liečba chronických nešpecifických zápalov čreva. Štandardný diagnostický a terapeutický postup. Metodický list racionálnej farmakoterapie a liekovej politiky MZ SR [Rational treatment of chronic non-specific intestinal inflammation. Standard, diagnosis and therapeutic process. Methodological list of rational pharmacotherapy and drug policy of the Ministry of Health of the Slovak Republic.]. 15/5–7: 2–8. [online] [cit. 2015-02-13]. Available from: <http://www.sgssls.sk/files/publikacie/metodicke-listy/ml-zapaly-creva-2012-final.pdf> (Slovak).
 16. Lochs H, Dejong C, Hammarqvist F, Hebuterne X, Leon-Sanz M, Schütz T et al. (2006). ESPEN Guidelines on Enteral Nutrition: Gastroenterology. *Clinical Nutrition*. 25/2: 275–284. [online] [cit. 2015-03-14]. Available from: <http://espen.info/documents/ENGastro.pdf>
 17. Lukáš M (2009). Farmakoterapie idiopatických střevních zánětů [Pharmacotherapy of idiopathic intestinal inflammation]. *Prakt. lékařn.* 5/4: 164–167 (Czech).
 18. Lukáš M (2011). Možnosti medikamentózní léčby u Crohnovy nemoci a ulcerózní kolitidy [Possibilities of medicamentous treatment of Crohn's disease and ulcerous colitis]. *Interní med.* 13/11: 422–426 (Czech).
 19. Lukáš M (2012). Idiopatické střevní záněty [Idiopathic intestinal inflammation]. In: Klener P et al. *Vnitřní lékařství [Internal medicine]*. Praha: Galén, pp. 611–619 (Czech).
 20. Lukáš M (2013). Současnost a budoucnost v léčbě Crohnovy nemoci [Present and future in treatment of Crohn's disease]. *Gastroent Hepatol.* 77/4: 306–312 (Czech).
 21. Lukáš M (2014a). Etiologie a patogeneze idiopatických střevních zánětů [Etiology and pathogenesis of idiopathic intestinal inflammation]. *Vnitř. Lék.* 60/7–8: 640–644 (Czech).
 22. Lukáš M (2014b). Perspektivy biologické léčby u idiopatických střevních zánětů [Perspectives of biological treatment of idiopathic intestinal inflammation]. *Gastroenterol Hepatol.* 68/3: 225–229 (Czech).
 23. Lyra A, Lahtinen S (2012). Dysbiosis of the Intestinal Microbiota in IBS. In: Lule G. *Current Concepts in Colonic Disorders*. New York: Springer, p. 262–276. [online] [cit. 2015-04-11]. Available from: <http://www.intechopen.com/books/current-concepts-in-colonic-disorders/dysbiosis-of-the-intestinal-microbiota-in-ibs>
 24. Novotná H (2013a). Dieta při zánětlivých chorobách střevních [Diet in inflammatory intestinal diseases]. In: Holubová A, Novotná H, Marečková J. *Ošetrovatelská péče v gastroenterologii a hepatologii [Nursing care in gastroenterology and hepatology]*. Praha: Mladá fronta, pp. 215–216 (Czech).
 25. Novotná H (2013b). Podávání enterální výživy – péče o pacienta [Providing enteral nutrition – patient care]. In: Holubová A, Novotná H, Marečková J. *Ošetrovatelská péče v gastroenterologii a hepatologii [Nursing care in gastroenterology and hepatology]*. Praha: Mladá fronta, pp. 193–201 (Czech).
 26. Novotná H. (2013c). Podávání parenterální výživy – péče o pacienta [Providing parenteral nutrition – patient care]. In: Holubová A, Novotná H, Marečková J. *Ošetrovatelská péče v gastroenterologii a hepatologii [Nursing care in gastroenterology and hepatology]*. Praha: Mladá fronta, pp. 203–208 (Czech).

27. Šachlová M (2011). Jakou volit stravu u pacientů s Crohnovou chorobou? [Which diet in patients with Crohn's disease?] *Interní Med.* 13/10: 403–405 (Czech).
28. Van Gossum A, Cabre E, Hébuterne X, Jappesen P, Krznaric Z, Messing B et al. (2009). ESPEN Guidelines on Parenteral Nutrition: Gastroenterology. *Clinical Nutrition.* 28/4: 415–427. [online] [cit. 2015-04-21]. Available from: <http://espen.info/documents/0909/Gastroenterology.pdf>
29. Vrzalová D, Konečný M, Ehrmann J (2011). Enterální a parenterální výživa u pacientů s nespecifickými střevními záněty [Enteral and parenteral nutrition in patients with non-specific intestinal inflammation]. *Med. Praxi.* 8/7–8: 337–338 (Czech).
30. Yutao Y (2012). Pathogenesis of Inflammatory Bowel Disease. In: Karoui S et al. *Inflammatory Bowel Disease – Advances in Pathogenesis and Management.* New York: Springer, pp. 111–134. [online] [cit. 2015-01-30]. Available from: <http://www.intechopen.com/books/inflammatory-bowel-disease-advances-in-pathogenesis-and-management/advance-in-the-pathogenesis-of-inflammatory-bowel-diseases>
31. Zbořil V (2013). Endoskopické metody v racionální diagnostice a terapii idiopatických střevních zánětů [Endoscopic methods in rational diagnosis and therapy of idiopathic intestinal inflammation]. *Čas. lék. čes.* 152/6: 280–283 (Czech).
32. Zbořil V (2015). Nutriční postupy [Nutrition procedures]. [online] [cit. 2015-02-01] Available from: <http://www.crohnovanemoc.cz/zakladni-lecebne-metody/nutricni-postupy/dieta.html> (Czech).
33. Zbořil V, Prokopová L (2010). Idiopatické střevní záněty – Crohnova nemoc [Idiopathic intestinal inflammation – Crohn's disease]. In: Bureš J, Vaňásek T et al. *Gastroenterologie [Gastroenterology].* Praha: Galén, pp. 99–102 (Czech).

 **Contact:**

Mária Boledovičová, University of South Bohemia in České Budějovice, Faculty of Health and Social Studies, Department of Nursing and Midwifery, U Výstaviště 26, 370 05 České Budějovice, Czech Republic
E-mail: boledovicova.maria@gmail.com