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Review

Nutritional concerns in pediatric inflammatory bowel disease patients

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In approximately one-fourth of patients with Crohn's disease (CD) and ulcerative colitis (UC), disease onset occurs during childhood and adolescence. In addition to gastrointestinal and extraintestinal symptoms of inflammatory bowel disease (IBD), children with these conditions often experience one or more nutritional complications of their disease including growth failure, delayed puberty, osteoporosis, anemia, and micronutrient deficiencies. This article provides an overview of the epidemiology, pathophysiology, evaluation, and management of selected nutritional complications in pediatric IBD.

Keywords: Inflammatory bowel disease / Nutritional complications / Pediatrics

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1 Introduction

Crohn's disease (CD) and ulcerative colitis (UC), collectively known as inflammatory bowel disease (IBD), are idiopathic and chronic inflammatory disorders of the gastrointestinal tract. Approximately 25% of cases begin during childhood or adolescence, and the incidence of these conditions appears to be increasing. CD, which is characterized by transmural inflammation, can affect any region of the gastrointestinal tract from the mouth to the anus and may be discontinuous. In contrast, the inflammation observed in UC is limited to the mucosa of the large intestine. Disease in patients with UC typically begins in the rectum and extends, in a continuous fashion, to include more proximal portions of the colon. While proctitis and left-sided colitis are common in adult patients with UC, children most often present with pan-colitis. Despite these differences, both conditions share a common pathophysiology and many of the same clinical features including abdominal pain, diarrhea, rectal bleeding, weight loss, and, in pediatric populations, growth failure. A number of other systemic and/or extraintestinal symptoms may also be present.

The key principles of therapy for patients with UC and CD are identical: induction and maintenance of remission, monitoring and correction of nutritional deficiencies, and prevention of complications. Physicians caring for children with IBD have the added tasks of restoring normal or near-normal linear growth, pubertal maturation, and psychosocial development. This article reviews the scope of nutritional complications in pediatric IBD, discusses the pathophysiology of these complications, and provides an overview of the evaluation and management of nutritional status in affected youth.

2 Nutritional complications in pediatric IBD: Overview and pathophysiology

In addition to gastrointestinal and extraintestinal symptoms of IBD, children with CD and UC will often experience one or more nutritional complications of their disease including growth failure, delayed puberty, osteoporosis, anemia, and micronutrient deficiencies (Table 1). Importantly, these nutritional complications can also contribute to the psychosocial impairment associated with chronic IBD.

In general terms, the pathophysiology of these nutritional complications is related to the type of disease (CD *versus* UC), anatomic location(s) of disease, severity of disease, and age of the patient. Nutritional complications are far

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Abbreviations: CD, Crohn's disease; **IBD,** inflammatory bowel disease; **IGF-1**, insulin-like growth factor 1; **IL**, interleukin; **UC**, ulcerative colitis

Table 1. Nutritional complications in pediatric IBD

Growth failure
Delayed puberty
Osteopenia and osteoporosis
Anemia
Micronutrient deficiencies: iron, folate, B_{12}, vitamin E, vitamin A, beta-carotene, magnesium, selenium, and zinc

Table 2. Mechanisms of growth failure in children with IBD

Inadequate caloric intake
Gastritis, esophagitis, and other symptoms
Cytokine-mediated anorexia
Fear of worsening GI symptoms
Malabsorption
Carbohydrate
Fat
Protein
Minerals
Fat-soluble vitamins
Increased energy expenditure from chronic inflammation
Pro-inflammatory cytokines and hormonal imbalances
Decreased IGF-1
Exogenous steroids

more common in patients with CD than UC, and in patients with active disease rather than in remission. Micronutrient deficiencies (*i. e.,* vitamin B_{12}) and/or fat malabsorption are more likely in patients with CD and ileal inflammation and/or resection rather than colonic disease. Finally, malnutrition is more likely to present as slowed growth and/or short stature in prepubertal children whereas postpubertal children and adults typically will have symptoms of weight loss [1].

2.1 Growth failure

Impairment of linear growth is common in children with CD. In fact, growth failure may precede the intestinal symptoms of CD in childhood [2]. Depending on the population under study and the definition of growth impairment, the proportion of children with this complication has varied from 32 to 88% [2–5]. Weight loss, or failure to gain weight appropriately for age, will often precede a decrease in height velocity. Additionally, pubertal development is often delayed. As a consequence of growth failure during childhood, 19–37% of patients with pediatric-onset CD fail to reach their expected adult height [6, 7].

In contrast to patients with CD, those with UC are less likely to experience pubertal delay [4] and/or a reduction in linear growth. In a prospective study which defined growth failure as a height Z score ≤ -1.64, the reported prevalence of growth failure in children with UC was 9%, compared to 38% in children with CD [5]. In another population-based study of growth in children with IBD, the reported prevalence of growth failure, defined as a height velocity SD score of <2.0 during at least one period between age 3 and the end of puberty, was 34% in UC patients compared to 65% in CD patients [4]. Consequently, 90% of children with UC will reach their expected adult height [7].

The pathophysiology of growth disturbance in children with IBD is likely multifactorial (Table 2). Inadequate caloric intake leading to chronic malnutrition has long been implicated as the primary cause for growth failure in children with IBD [8]. Caloric deficiency may be due to a combination of factors including symptoms of esophagitis and gastritis, cytokine-mediated anorexia, and fear of worsening gastrointestinal symptoms. Stool losses, including protein-losing enteropathy and steatorrhea, occur secondary to mucosal damage and may also account for nutrient deficiency. Not only do children with IBD have inadequate caloric intake, they also have increased energy expenditures resulting from fever and chronic inflammation [9].

In addition to protein–energy malnutrition, direct growth-inhibiting effects of proinflammatory cytokines released from the inflamed intestine may also contribute to growth failure in children with IBD. Using a rat model of colitis, Ballinger *et al*. [10] have recently reported decreased growth velocity in rats with colitis compared with diet-matched controls. Levels of insulin-like growth factor 1 (IGF-1) were also decreased in the colitis group. Correction of nutritional balance alone did not restore linear growth; administration of IGF-1 to the colitic group increased plasma IGF-1 concentrations and linear growth by approximately 44–60%. They concluded that growth failure occurs as a direct result of the inflammatory process, mediated by a decrease IGF-1, which is independent of malnutrition [10]. This and other studies suggest that decreased IGF-1 may be mediated by an increase in circulating proinflammatory cytokines such as interleukin-6 (IL-6) [11].

Finally, corticosteroid treatment is another mechanism that contributes to growth failure in pediatric IBD patients. Retrospective studies have described a positive association between corticosteroid treatment and growth restriction [7]. However, this association has not been consistently observed [3, 5, 6]. Indeed, the independent effects of corticosteroid treatment and growth are difficult to establish because of confounding by severity, anatomic location, and other clinical factors. Furthermore, it is unclear what daily dose is necessary to suppress growth. The growth suppressive effects of glucocorticoids are multifactorial, including (i) central suppression of growth hormone release, (ii) decreased hepatic transcription of growth hormone receptor, such that production of IGF-1 is decreased, and (iii) decreased IGF-1 binding in cartilage. In essence, the use of exogenous corticosteroid may result in a state of functional growth hormone deficiency [12].

Taken together, the implication of these mechanisms of growth failure in children with IBD is that restoration of

normal linear growth velocity can be maximized through a multipronged approach: (i) control of active intestinal inflammation, (ii) provision of adequate nutrition, and (iii) minimization of corticosteroid exposure.

2.2 Osteopenia and osteoporosis

Bone mineralization is another important consideration in the care of the growing child with IBD, particularly as peak bone mass, which is attained during adolescence, is the most important determinant of lifelong skeletal health. High rates of osteopenia and osteoporosis, as defined by the World Health Organization as >1 SD and >2 SD below the mean, respectively, have been reported as complications of pediatric IBD [13–15].

Similar to growth failure, decreased bone mineral density is also more common among children with CD than UC [13–15]. In a recently published study of 58 children with CD, 18 with UC, and 49 healthy controls, Sylvester *et al.* [13] reported the prevalence of osteopenia was 43%, 39%, and 29% respectively. Osteoporosis was present in 12% of CD patients, compared to 6% of UC patients and 2% of controls. In another study, Gokhale *et al*. [15] observed osteopenia (lumbar spine Z scores < –1) in 35% of CD patients, compared to 22% of UC patients. Osteoporosis (lumbar spine Z scores < –2) was observed in 18% of children with CD compared to 3% with UC. As in adult populations, decreased bone density has also been associated with compression fractures in pediatric patients with IBD [8].

The etiologies for osteopenia and osteoporosis in IBD are multifactorial and include hypovitaminosis D, decreased calcium intake and calcium malabsorption from steatorrhea, corticosteroid effects, and cytokine-mediated bone resorption. Vitamin D (serum 25-hydroxy-vitamin D (25OHD)) deficiency is high among children and adolescents with IBD. Pappa *et al*. [16] recently reported that 25OHD deficiency, defined as a serum concentration ≤15, was present in 34.6% of pediatric IBD patients, compared to 24.1% of healthy New England controls. Mechanisms for hypovitaminosis D may include decreased exposure to sunlight, decreased intake, and protein losing enteropathy which could lead to a deficiency of vitamin D binding protein [8].

Calcium malabsorption occurs secondary to steatorrhea, as calcium binds to the increased intraluminal fat. Of note, this same process also predisposes CD patients to developing hyperoxaluria and renal stones. Under normal circumstances, intraluminal calcium binds oxalate facilitating oxalate excretion. When free calcium is decreased in the setting of steatorrhea, the absorption of oxalate is increased [1].

In addition, corticosteroids reduce calcium absorption, down-regulate calcitriol synthesis, decrease gene expression of calcium-binding protein, inhibit osteoblast proliferation, and stimulate osteoclastic bone resorption [17]. Indeed, the cumulative exposure to corticosteroids is negatively associated with lumbar spine bone mineral density [14, 15]. Finally, there is some evidence to suggest that proinflammatory cytokines including IL-6 and tumor necrosis factor-alpha released from an inflamed gut may inhibit osteoblastic activity [18].

2.3 Anemia

Anemia in IBD may occur from blood loss, chronic inflammation, micronutrient deficiency (iron, folate, B_{12}), myelosuppression, and hemolysis. Iron deficiency and the anemia of chronic inflammation are the most common causes of anemia in CD. These two conditions are inter-related. It is now recognized that the inflamed intestine in IBD releases proinflammatory cytokines such as IL-6 into the intestine. These systemically released cytokines in turn trigger the release of the hormone hepcidin from the liver. The hepcidin then impairs iron absorption from the intestinal enterocyte, and sequesters iron within the cells of the reticuloendothelial system, resulting in impaired erythropoiesis and anemia [19]. Children with active CD have evidence of increased IL-6, increased urinary hepcidin, and impaired intestinal iron absorption [19, 20].

Laboratory features include microcytosis and low levels of serum iron and ferritin. Iron deficiency anemia may be difficulty to diagnose in affected patients because serum ferritin can be elevated as part of the acute phase response. Other etiologies of anemia in IBD patients include folate or vitamin B_{12} deficiency (particularly in patients with ileal CD), impaired utilization of iron, or myelosuppression caused by medications including 6-mercaptopurine [1]. Folate deficiency may be particularly important, not only for its contribution to anemia, but also because low folate levels may result in an elevation of homocysteine and hence contribute to the hypercoagulable state observed in affected patients [21].

2.4 Other micronutrient deficiencies

Although clinically relevant micronutrient deficiencies in pediatric IBD patients are very rare and largely limited to case reports, subclinical micronutrient deficiencies have been commonly reported in patients with IBD. Deficiency of both vitamin A and E has been reported in 16% of children with IBD, and the risk of these vitamin deficiencies is positively correlated with disease activity [22]. Other nutrient deficiencies that have been reported in IBD patients include beta-carotene, magnesium, selenium, and zinc [23]. Zinc is an essential mineral which is an essential cofactor for a number of human enzymes (including alkaline phosphatase), and is also necessary in the "zinc finger" proteins that regulate gene transcription. Symptoms of severe zinc deficiency, as seen in acrodermatitis enteropathica, include skin rashes, perianal inflammation, irritability, hypogeusia (decreased taste), and growth failure

[24]. While such severe symptoms of zinc deficiency are rarely reported in CD patients, up to 65% of CD patients in remission may have low plasma zinc levels [25]. Griffin *et al*. [26] evaluated intestinal zinc absorption, and urinary and fecal zinc excretion in a cohort of 15 adolescents with CD compared to 15 healthy controls; the CD patients had both lower plasma zinc levels and decreased intestinal absorption, but normal excretion [26]. While zinc supplementation has not been proven to prevent relapse or reduce disease activity, a pilot study suggested zinc supplementation might normalize intestinal permeability in CD patients [27]. Taken together, the available literature suggests that zinc deficiency is common in patients with CD, and that patients should be periodically screened for zinc deficiency. At least one study suggests that UC patients also may have zinc deficiency [23]. However, the clinical response and benefits of zinc supplementation in IBD patients remain a topic of study.

3 Indications for nutritional therapy in children with CD

Adequate nutritional support is an essential part of the long-term management of IBD. Total parenteral nutrition has long been recognized as a useful adjunct to medical therapy in the severely ill, hospitalized patient with either CD or ulcerative colitis. Preoperative parenteral nutrition is effective in reducing surgical complications in malnourished children with CD or UC undergoing resection [28–30]. In addition, parenteral nutrition has been effectively used to control and even close postoperative fistulas [31].

In extreme cases of gut failure from extensive disease and/or bowel resection, therapy with home parenteral nutrition can result in nutritional repletion, reduced requirement for corticosteroids, and improvement in quality of life. However, home parenteral nutrition has the potential risks of catheter blockage, catheter-related sepsis, dehydration, and electrolyte imbalance [31].

Enteral supplementation is also an important adjunct to medical therapy in hospitalized patients with less severe disease. Additional indications for enteral nutritional therapy in the treatment of CD include: (i) exclusive enteral nutrition for active disease, (ii) supplemental enteral nutrition to maintain disease remission, (iii) nutritional support to promote adequate weight gain and facilitate catch-up growth, and (iv) supplementation of specific vitamins and minerals [32].

3.1 Efficacy of enteral nutrition *vs.* steroids in the induction of remission in active CD

Although rarely used in the United States, enteral nutritional therapy is considered a front-line therapy for the induction of remission for patients with active CD. Although a number of meta-analyses conducted in the 1990s concluded that enteral nutrition as a primary therapy for CD is not as effective as corticosteroids [33, 34], a more recent meta-analysis which included only pediatric trials indicated that enteral nutrition might be of comparable efficacy to corticosteroid therapy [35]. This apparent discrepancy might be due to differences in the study populations (*i. e.*, age, severity, disease location), methodologies, interventions, and outcome assessments of included studies.

More recently, Zachos *et al*. [36] reported a meta-analysis of six trials which included 192 patients treated with enteral nutrition and 160 treated with steroids. The pooled odds ratio was 0.33, favoring steroid therapy (95% CI 0.21–0.53). Of the six studies included, the two studies which were rated as high quality had conflicting, but not statistically significant results. Inclusion of only these two studies in the meta-analysis yielded an odds ratio of 1.24 favoring elemental therapy (95% CI 0.44–3.45). In the first study, Gonzalez-Huix *et al*. [37] performed a randomized, controlled trial of polymeric enteral nutrition administered by nasogastric tube compared to steroids (1 mk/kg/day). Fifteen of 17 patients (88%) given steroids and 12 of 15 patients (80%) given enteral nutrition achieved clinical remission. A pediatric randomized trial comparing polymeric formula alone to oral corticosteroids found that the proportion of patients achieving clinical remission was comparable between the two groups: 15 of 19 (79%) for those treated with polymeric formula and 12 of 18 (67%) for those treated with steroids) [38]. Taken together, the results of these studies and meta-analyses suggest that steroids are likely to be more effective than enteral nutrition for the induction of clinical remission in adults with active CD; however, it should be noted that the pediatric trial and meta-analysis both failed to find significant differences between these two treatment modalities. Thus, it is possible that the benefits of enteral nutrition may differ between children and adults.

3.2 Composition of enteral formula

A meta-analysis of ten trials which included 199 adult patients with active Crohn's treated with an elemental diet and 146 patients treated with a nonelemental diet demonstrated no statistically significant differences among diet formulations (OR 1.10; 95% CI 0.69–1.75). Similarly, low-fat *versus* high-fat formulas appeared to be equally effective in inducing remission [36].

3.3 Route of administration

In the majority of published studies, enteral feedings were administered through a nasogastric tube. Some trials, including the pediatric study, allowed subjects to take the feed orally with nasogastric feeding instituted if the children were unable to tolerate the prescribed volume orally.

The use of polymeric formulas, such as the Modulen used in this study, improves the palatability of enteral treatment regimens; however, a significant number of patients are unable to tolerate the prescribed volume. Approximately 23.5% of children in the enteral nutrition group eventually required nasogastric feedings. Furthermore, two of these children dropped out of the study due to inability to tolerate nasogastric tube feedings [38]. Although palatability and/or the nasogastric route of feeding may be viewed as barriers to enteral nutritional therapy, Afzal *et al*. [39] have demonstrated improvements in quality of life for children after treatment with exclusive enteral nutrition.

3.4 Mucosal healing

Current treatment paradigms call for mucosal healing in addition to symptom control and clinical remission. Although many studies suggest that corticosteroids fail to induce mucosal healing in a large number of patients, several trials have suggested that enteral nutritional therapy may lead to endoscopic as well as clinical remission in pediatric patients with active CD [38–40]. In the Borrelli paper described above, 14 of 19 children (74%) randomized to enteral therapy demonstrated mucosal healing, compared to 3 of 18 (33%) in the corticosteroid group. Further work is necessary to confirm the effects of enteral nutrition on mucosal healing as well as the importance of mucosal healing as a clinically significant endpoint.

3.5 Mechanisms of enteral nutrition

The mechanisms by which enteral nutrition improves disease activity and promotes endoscopic healing in patients with CD are not fully understood. Nutrients are a major component of the intestinal ecosystem, and along with bacteria may be involved in the regulation of mucosal immunity. Thus, it has been suggested that enteral therapy may alter the intestinal microenvironment, removing disease triggers. In addition, suboptimal levels of micronutrients may also participate in the pathogenesis of CD due to impaired host defense. Restoration of these micronutrients might improve mucosal functioning. Finally, enteral nutrition may have direct antiinflammatory effects. Fell *et al*. [40] demonstrated decreased interferon gamma and increased TGF beta mRNA in the ileum and decreased IL-8 mRNA in the colon after treatment with a polymeric diet. Thus, enteral nutrition may exert its therapeutic effects through a variety of mechanisms which are only now being described.

3.6 Summary of enteral nutritional therapy in the treatment of active CD

Enteral nutritional therapy is a safe and effective treatment option for patients with active CD. Although probably not as effective as corticosteroids in inducing clinical remission, some evidence suggests that children may respond better than adults to nutritional therapy. The protein and fat composition of different nutritional therapies does not appear to influence the effectiveness of enteral nutrition. Although a major barrier to the use of enteral therapy may be patient tolerance (*i.e.*, palatability and/or nasogastric tube placement), there is evidence that health-related quality of life actually improves after the institution of enteral therapy. In addition to the above considerations, the steroid sparing effects of enteral therapy as well as the additional benefits of improved weight and growth status make enteral therapy a reasonable consideration in selected patients with CD. Further work is necessary to further elucidate the mechanisms by which enteral therapy works and to define clinical and demographic factors (*i.e.*, age, severity, disease duration, and location) that may predict response to therapy.

3.7 Other indications for nutritional therapy in pediatric IBD

A limited number of studies suggest that long-term enteral nutrition prolong remission in patients with CD. In a retrospective study of 65 children and adolescents with CD who were successfully brought into remission with exclusive enteral nutrition, Wilschanski *et al*. [41] found that those patients who continued supplementary enteral nutrition (nocturnal tube feedings) had a longer time to recurrence than those not taking supplements. Similar results have been described in adult studies of oral nutritional supplementation [42]. Larger, prospective studies are necessary to fully evaluate the efficacy of long-term enteral supplementation for the maintenance of remission in CD.

Nutritional supplementation *via* nocturnal tube feedings may also be used to support adequate weight gain and facilitate catch-up growth in children with severe growth failure. The advantage of nocturnal supplementation is that a child can eat normally during the day, receive an additional 1000–1500 calories overnight, and the tube can be removed before school in the morning. In a study of children with CD aged 8–15 years, intermittent nocturnal nasogastric infusion of an elemental formula resulted in a mean weight gain of 6.9 kg and height gain of 7.0 cm over a 1-year period [43].

An additional indication for nutritional supplementation is to prevent and correct specific micronutrient deficiencies as discussed in Section 4.

4 Monitoring of nutritional status in children with IBD

Screening and assessing the nutritional status of children with IBD are an essential component of medical care (Table 3). This includes, at a minimum, body weight for age

Table 3. Monitoring of nutritional status in children with IBD

Anthropometrics
Height for age
Weight for age
Body mass index
Plotted on longitudinal growth curves
Monitoring for anemia
Complete blood count, if hematocrit low then check:
Reticulocyte count
Serum iron, total iron binding capacity, and serum ferritin
Folate and B_{12}
Hemoglobin electrophoresis
Monitoring for malabsorption
Serum albumin
Zinc
Monitoring for bone health (patients with severe disease)
Bone densitometry
Serum 25-OH vitamin D and parathyroid hormone

and height for age, with calculation of body mass index. These data should be plotted and followed longitudinally on appropriate growth charts. Obtaining a dietary history (often with the assistance of a clinical nutritionist or registered dietician) and reviewing recent weight changes should be part of each clinical encounter [9]. A decline in any of these nutritional parameters should prompt further evaluation of disease activity along with dietary counseling to increased caloric consumption. If there is evidence of ongoing intestinal inflammation, more aggressive pharmacologic management should be considered. If nutritional status remains suboptimal despite treatment of the underlying disease, nocturnal supplementation *via* nasogastric tube, as reviewed in Section 3.7, should be discussed with the child and his/her family.

In addition to these macronutrient considerations, we recommend that children be counseled to take a daily multivitamin given the many studies documenting micronutrient and antioxidant deficiencies discussed previously. Most such vitamins contain 400 mg of folic acid, but in patients receiving sulfasalazine and/or methotrexate, 1 mg is recommended. We also recommend that patients consume 1200–1500 mg of total daily calcium along with 400 IU/day of vitamin D, either through their diet or as supplements. Finally, patients with chronic ileitis and/or a history of ileal resection should be monitored for vitamin B_{12} deficiency, and if necessary, supplemented with oral or intramuscular vitamin B_{12}.

Evaluation of a patient with persistent anemia should include complete blood count, reticulocyte count, erythrocyte sedimentation rate, C-reactive protein, serum iron, total iron binding capacity, serum ferritin, and hemoglobin electrophoresis (if a hemoglobinopathy is suspected). Persistent iron deficiency in a patient with IBD suggests poorly controlled disease, especially if acute phase reactants (ESR, CRP) are elevated. In such patients, additional therapy (including immunomodulators, infliximab, or surgery) may be indicated to induce a remission. Alternatively, iron supplementation may be of benefit, with intravenously administered iron being more effective and better tolerated than iron given orally [44].

5 Optimizing and monitoring of skeletal health in children with IBD

As discussed previously, children with IBD are at increased risk for osteopenia and osteoporosis. However, no established guidelines exist for the monitoring of bone health in children with IBD. In addition, it is unclear what medical interventions are appropriate in children with osteopenia diagnosed by bone densitometry. Nevertheless, we suggest monitoring patients at high risk for osteopenia (severe disease, prolonged corticosteroid use, *etc*.) with bone densitometry as well as following serum levels of 25-OH vitamin D and parathyroid hormone.

As with growth failure, effective therapy of the underlying disease is the most powerful means to prevent (and treat) osteoporosis [9]. Maintaining physical activity, encouraging full participation in sports, and minimizing bed rest are also important components of treatment, as is minimizing further corticosteroid exposure. Finally, it is important to ensure adequate intake of both calcium and vitamin D as discussed above.

If osteopenia is recognized, patients should be treated with vitamin D and calcium. For patients with vitamin D deficiency, supplementation of vitamin D in the form of ergocalciferol (D_2) or cholecalciferol (D_3) may be necessary. Recent evidence suggests that vitamin D_3 may be more efficacious than vitamin D_2 [45].

Options for the pharmacologic management of osteopenia/osteoporosis include calcitonin and bisphosphonates; however, there is not enough data on the safety and efficacy of these medications in pediatric populations to make definitive recommendations at this time. Therefore, for patients with osteoporosis with or without fractures, we suggest referral to an expert in pediatric skeletal health and consideration of clinical trial participation.

6 Conclusions

CD and ulcerative colitis are chronic IBDs which often begin during childhood or early adulthood. Nutritional complications are common in patients with IBD, and include growth failure, osteopenia/osteoporosis, anemia, and vitamin malabsorption. The frequency and type of these complications are related to disease type, severity, anatomic location, and age of onset. Clinicians caring for

children with IBD should be aware of and screen for these complications. The general principles for managing nutritional complications include: (i) pharmacologic and/or surgical intervention to reduce or remove chronic intestinal inflammation, (ii) minimization of corticosteroid use, (iii) supplementation of macronutrients to reduce protein–energy malnutrition, and (iv) supplementation of micronutrients when appropriate. Furthermore, exclusive enteral nutrition may have a role in the treatment of active CD.

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