General Nutrition and Fitness for the Child with Rheumatic Disease



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KEYWORDS

- Obesity Nutrition Growth Supplements Cardiovascular disease
- Physical fitness
 Exercise
 Bone health

KEY POINTS

- Because of new medications and aggressive treatment, most children with rheumatic diseases are no longer at high risk for growth failure.
- Children with rheumatic diseases have a risk of long-term poor bone and cardiovascular health outcomes due to both chronic disease and corticosteroid treatment.
- The duration and dose of corticosteroid therapy can often be minimized by using steroidsparing agents, such as disease-modifying antirheumatic drugs and biologic response modifiers.
- Bone health and compliance with daily calcium and vitamin D supplementation should be monitored.
- Fitness levels are often low, although functional skills are good in children with rheumatic disease. Providers should assess fitness and plan for daily physical activity with a graduated program that may include rehabilitation, group exercise and recreation, and strengthening and conditioning.

INTRODUCTION

Children with rheumatic diseases are at higher risk of poor nutritional outcomes and growth failure due to chronic inflammation. An additional consequence of uncontrolled disease is that arthritis or other manifestations limit both activities of daily living and the ability to exercise. In the long term, chronic inflammation is associated with other comorbidities, such as cardiovascular disease (CVD) and poor bone health. In the past, treatment for control of the most serious diseases, such as systemic juvenile idiopathic arthritis (sJIA), systemic lupus, and dermatomyositis, relied heavily on glucocorticosteroids (GCS), which have may metabolic side effects (also discussed in

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Dr. Gloria C. Higgins' article, "Complications of Treatments for Pediatric Rheumatic Diseases." in this issue.).

The overall outcomes of childhood rheumatic diseases have improved remarkably in the last 20 years because of more rapid recognition, better understanding of these disease processes, the availability of new medications, and a trend towards more aggressive treatment. Nonetheless, good nutrition, bone health, and physical fitness warrant close attention in these children and are best achieved through collaboration among the primary care provider, other pediatric care providers, and the rheumatologist.

WHAT PHYSIOLOGIC PROCESSES INHIBIT GROWTH IN CHILDREN WITH RHEUMATIC DISEASES?

Chronic systemic inflammation itself results in cachexia, which is associated with poor appetite, increased basal metabolic rate, loss of lean muscle, and inefficient use of stored fat for energy. Occasionally, the chronic disease state is associated with growth hormone resistance, which affects muscle mass and bone density. Tumor necrosis factor (TNF), initially named cachexin, and many other cytokines such as interferon- γ and several interleukins (IL-6, IL-12, IL-17, and IL-23) also inhibit growth through pathways that involve insulin-like growth factor 1, a mediator of growth hormone. In addition, malabsorption and medication side effects can contribute to vitamin and macronutrient deficiencies, poor muscle mass, and abnormally low bone density, respectively. 4

WHAT ARE THE EFFECTS OF RHEUMATIC DISEASE MEDICATIONS ON GROWTH?

It is well known that chronic high-dose GCS treatment decreases growth velocity. Although the best available growth data are for children with juvenile idiopathic arthritis (JIA), children with other severe rheumatic diseases often did, and sometimes still do, receive prolonged courses of high to moderate dose GCS. In the past, as a result of both their disease processes and prolonged treatment with GCS, a high proportion of children with sJIA and rheumatoid factor–positive polyarticular JIA had growth failure. In an early longitudinal study of children with "juvenile chronic arthritis" followed an average of 15 years until 1981, 10% of patients with systemic arthritis experienced severe growth failure. In a later study, 40% of patients with a history of sJIA who had received at least 2 years of treatment with GCS during childhood, had a final height of at least 2 standard deviations below the mean. In addition, 80% of this cohort failed to reach predicted final height.

The timing of GCS therapy before and during the physiologic growth spurt also affects the degree of growth retardation. Early onset of arthritis was shown to be a contributor to growth failure.⁸ In a 2011 study of growth and development in childhood lupus, growth failure determinants included age at first visit less than 13.4 years, and cumulative steroid dose greater than 400 mg/kg.⁹

GROWTH IN JUVENILE IDIOPATHIC ARTHRITIS PATIENTS IS OVERALL IMPROVING

Soon after the advent of TNF inhibitors (TNFi), improvement in the growth of JIA patients treated with these medications became evident. A cohort of patients with severe polyarticular course JIA, followed 2 years before and 2 years after treatment with the TNFi etanercept or infliximab, was described in 2006. Along with the significant decrease in disease activity after introduction of a TNFi, linear growth velocity increased in 76% of patients. The improvement in the growth velocity was the best in patients with the greatest growth retardation. Of the 77% of patients who were taking GCS at the time of starting a TNFi, all were able to significantly decrease their

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