1. INTRODUCTION

One of the major somatic consequences and psychological burdens of chronic renal failure (CRF) is the disturbance of body growth. Since the start of dialysis improves renal clearance, it was the hope of patients and clinicians that the improved renal clearance may also result in a better growth rate. Unfortunately, those expectations have not been fulfilled. Furthermore, the time of dialysis is usually too short to correct for a previous height deficit. As a consequence, treatment of growth failure in renal patients should be diagnosed and treated early. Since the majority of children with CRF have congenital renal disorders, the problem of prevention and treatment of growth failure starts immediately after birth.

This chapter outlines etiology, clinical presentation and treatment modalities of growth disturbance in CRF prior to and during dialysis treatment.

2. CLINICAL PRESENTATION

The regulatory mechanisms of statural growth during childhood differ in the successive stages of development. During the first 2 years of life, growth is mainly driven by nutritional factors, particularly the intake of energy and protein. In later childhood, growth appears to depend mainly on the somatotropic axis, with nutrition exerting a more permissive influence. During puberty, the growth process is dominated by the gonadotropic hormone axis, which stimulates and finally terminates body growth by a direct action on the growth cartilage and by modulation of the somatotropic axis. In view of these differences in growth regulation, growth in renal disorders will be described separately for the periods of infancy, midchildhood and puberty.
2.1. Infancy

Untreated CRF during early infancy is usually associated with severe growth retardation. The loss in relative height is greatest during the first year of life, particularly during the first 6 months. A detailed analysis of the early infantile growth pattern according to the Infancy–Childhood–Puberty model of Karlberg revealed that the infancy growth phase, starting in intra-uterine life and ending during the second year of life, is affected in 50% of patients with CRF. Height SDS was already slightly reduced at birth, decreased further during the first 3 postnatal months, stabilized between 3 and 9 months and decreased again between 10 and 12 months of life. After a transient stabilization of growth rate, a further loss in relative height apparently occurred between 0.75 and 1.5 years of age. In the mechanistic Infancy–Childhood–Puberty model, this period reflects the transition from the infancy to the childhood growth phase (Figure 1). The height deficit acquired during this period may be due either to a delayed onset of the childhood growth phase or to a temporary “offset” of the childhood growth phase. In unselected patients studied by Karlberg et al., a loss of height SDS of nearly 4 SD was observed at the end of the third year of life. The reasons for this secondary deterioration of growth in infancy, which may occur despite adequate nutritional and medical supplementation, are still poorly understood. If the hypothesis is correct that the childhood growth component is mainly driven by the somatotrophic axis, the growth patterns during this transitional period could represent changes between periods of normal (infancy and childhood components operative) and impaired GH action (only infancy component intact). With regard to early postnatal life, anorexia, water and electrolyte imbalances caused by uremia, recurrent vomiting, catabolic responses to infections and metabolic acidosis have been cited as the main factors compromising this period of growth.

![Figure 1](image-url)