A case of pseudotumor from overgrowth of the fibula
Effect of growth hormone treatment of juvenile arthritis

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Case report

A 13-year-old girl with polyarticular destructive juvenile chronic arthritis (JCA) after systemic onset at the age of 1 year was referred to us for a suspect tumor in the left medial calf and some lateral bowing of the lower leg (Figure 1). It had grown slowly for the past 6 months and was solid on palpation but not tender or painful. The girl had been chair-bound for several years; she had 70° flexion deformity of both hips and knees as well as a slight outward rotational deformity of both lower legs. She had pronounced general growth retardation, due to early treatment with massive doses of corticosteroids. Treatment with growth hormone (GH) had been started at the age of 11, and resulted in accelerated longitudinal growth of tubular bones, evident in the left fibula as well as the right tibia and fibula. Due to premature closure of the left proximal and distal tibial growth plates, there was an S-shaped deformation of the rapidly growing left fibula (Figure 2), which explained the tumor-like appearance on the medial side of the left calf.

The GH treatment, which had gone on for 22 months, was discontinued and a reconstruction plan made up. This included correction of the flexion deformity of the knees in combination with lengthening and angular correction of the left lower leg, followed by soft tissue releases of the hips and subsequent endoprosthetic replacement of both hips and knees. Treatment started on the left leg with correction of the knee flexion contracture in combination with correction and lengthening of the lower leg with an Ilizarov frame. The flexion deformity of the knee was initially treated with distension, followed by extension, and full correction was obtained after 4 weeks. 4 cm of tibial lengthening had been obtained after 6 weeks. The frame was removed after 4 months, and was followed by plaster for another 2 months. At follow-up after 6 months she had 20–70° motion of the left knee with no rotational or angular deformity of the lower leg. The curved fibula was partly straightened and the tumor had disappeared.

Discussion

Growth disturbance is common in JCA, especially in those with a low age at onset of the disease. Stunted general growth may be caused by the disease per se, but also by treatment with systemic corticosteroids. Excessive local growth is probably due to the stimulation of growth plates by the hyperemia caused by chronic synovitis. It is most commonly seen in the knee, causing leg length discrepancy and flexion deformity. It is also common in the hip causing increased valgus and anteversion of the femoral neck, and in the foot with an equino-varus deformity of the hindfoot, cavus deformity of the middle foot, and adductus deformity of the forefoot (Rydholm 1990).

GH has been used for treating general growth retardation, but so far no effect on final length has been proven in JCA, and the possible side effects are numerous and often difficult to anticipate. If GH treatment is considered for a child with JCA, it is an absolute prerequisite to make a map of the maturity of the different growth plates in the long tubular bones. The timing of physeal closure is not always symmetric in these children, as the intensity of the local joint synovitis determines the behavior of the growth plates. Local hyperemia accelerates physeal growth and may also result in premature closure. Starting GH treatment in the absence of radiographs of the tubular bones may result in considerable asymmetric local growth disturbance which is more troublesome for the patient than the initial one. The Ilizarov method seems to be a good instrument for correction of deformities caused by both the disease itself (Hägglund et al. 1993) or by treatment with GH.
Figure 1. The left lower leg with lateral bowing and the medial "tumor".

Figure 2. S-shaped deformation of the left fibula due to growth stimulation by GH.

Figure 3. Clinical appearance after treatment.

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References
