International Clinical and Medical Case Reports Journal
Case Report (ISSN: 2832-5788)

What can a Wrist X-Ray Tell Us?

Merter Keçeli*

Assoc. Prof. Adnan Menderes University Medical School, Department of Pediatric Radiology, Aydın/Turkey

Citation: *Merter Keçeli. What can a Wrist X-Ray Tell Us?. Int Clinc Med Case Rep Jour. 2026;5(6):1-5.*

Received Date: 10 June 2026; **Accepted Date:** 13 June 2026; **Published Date:** 14 June 2026

***Corresponding author:** Merter Keçeli**,** Assoc. Prof. Adnan Menderes University Medical School, Department of Pediatric Radiology, Aydın/Turkey

ABSTRACT

Linear growth in children is regulated by complex interactions between the growth plate and systemic biochemical signals. In juvenile idiopathic arthritis (JIA), chronic inflammation and proinflammatory cytokines may disrupt normal growth plate maturation through both systemic and local mechanisms. Radiographic manifestations of growth plate involvement may precede overt joint destruction and provide early diagnostic clues.

An 8-year-old girl with a four-year history of joint pain and restricted movement presented for routine follow-up. She had no systemic symptoms, and laboratory studies showed no evidence of acute inflammation. Radiography of the left hand demonstrated narrowing of the fourth proximal interphalangeal joint space with subarticular sclerosis. Magnified images revealed widening of the proximal epiphysis of the fourth middle phalanx and early epiphyseal fusion compared with adjacent digits. No periosteal reaction or soft-tissue abnormality was observed. These findings, together with the clinical and laboratory data, were consistent with juvenile idiopathic arthritis involvement, resulting in accelerated bone maturation.
In JIA, proinflammatory cytokines such as IL-1, IL-6, and TNF-α can adversely affect growth plate function. Increased regional blood flow and chronic inflammation may lead to epiphyseal overgrowth or premature epiphyseal closure, which can result in growth arrest and asymmetry. Recognition of asymmetric epiphyseal maturation on imaging is essential for early diagnosis and appropriate clinical management. Growth plate abnormalities may represent an early radiographic manifestation of juvenile idiopathic arthritis. Careful assessment of epiphyseal morphology and maturation on pediatric hand radiographs can aid in identifying inflammation-related growth disturbances and help prevent long-term skeletal asymmetry.

Keywords: Juvenile idiopathic arthritis; Growth plate; Epiphyseal fusion; Pediatric radiology; Chronic inflammation

INTRODUCTİON

The growth plate is a thin layer of cartilage located near the ends of long bones and vertebrae. A hallmark of endochondral bone growth is that chondrocytes remain in a spatially fixed position within the growth plate as it differentiates through a series of maturational stages.[1] Skeletal growth during childhood is regulated by complex biochemical and physiological mechanisms. Impaired linear growth is frequently observed in children

with chronic inflammatory diseases, both at disease onset and following glucocorticoid therapy. Growth impairment in these patients is multifactorial and influenced not only by treatment but also by disease activity, nutritional and endocrinological status, and the body's response to inflammatory mediators.

Growth abnormalities in this population are often associated with altered bone development. In children with inflammatory diseases, cytokine concentrations have been shown to correlate positively with disease activity in juvenile idiopathic arthritis (JIA).[2,3] Proinflammatory cytokines—interleukin-1 (IL-1), tumor necrosis factor-α (TNF-α), and interleukin-6 (IL-6)—play a key role in JIA and may affect growth through systemic mechanisms and/or direct local effects on the growth plate of long bones.[4-6]

The purpose of this case presentation is to illustrate the effects of chronic inflammation on growth plate maturation and to describe the associated diagnostic findings on wrist radiography.

CASE PRESENTATİON

An 8-year-old girl, who had been followed for four years because of joint pain and limited range of motion, presented for routine follow-up. She had no fever or systemic complaints. Laboratory evaluation revealed no findings suggestive of acute inflammation.

A radiograph of the left hand demonstrated narrowing of the fourth proximal interphalangeal joint space and increased subarticular sclerosis (Figure 1). On magnified images, the proximal epiphysis of the fourth middle phalanx was wider than the corresponding epiphyses of the adjacent digits, and early epiphyseal fusion was observed (Figure 2). No periosteal reaction or associated soft-tissue mass was detected.

These radiographic findings were consistent with juvenile idiopathic arthritis involvement. The patient's clinical history, physical examination, and laboratory findings supported the diagnosis. In this patient, JIA involvement resulted in accelerated bone maturation. Premature epiphyseal fusion is expected to arrest longitudinal growth of the affected digit, potentially leading to digital length asymmetry.
Written informed consent was obtained from the patient's legal caregivers.

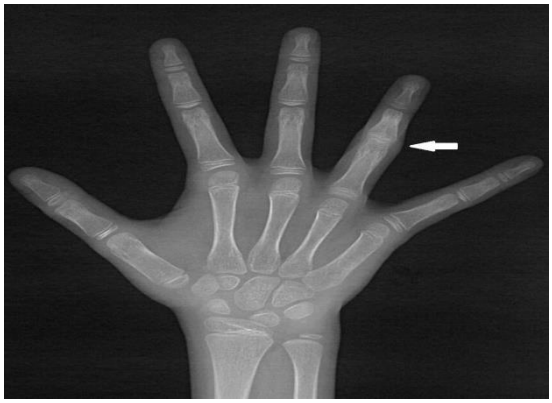

Figure 1: Anteroposterior radiograph of the left wrist and hand showed narrowing of the fourth proximal interphalangeal joint space and sclerotic subarticular density (arrow).

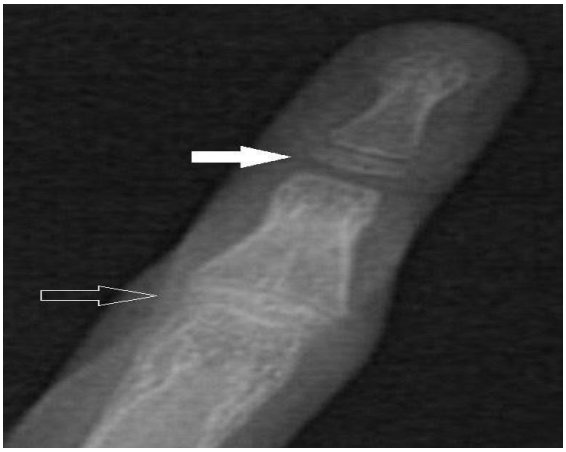

Figure 2: On magnified view, the proximal epiphysis of the fourth middle phalanx (open arrow) was larger than the other epiphyses (arrow), and epiphyseal fusion was evident.

DISCUSSION

Growth maintenance in children with JIA is a complex process influenced by multiple interacting mechanisms. Because bony erosions typically develop later in the disease course, abnormal epiphyseal growth may represent one of the earliest radiographic indicators of JIA. Systemic JIA is currently classified as an autoinflammatory disease.[7]

Cytokines involved in JIA exert both systemic and local effects on growth plates. Systemically, elevated levels of TNF-α and IL-6 are associated with growth suppression. IL-1, IL-6, and TNF-α are among the most extensively studied cytokines affecting bone and cartilage metabolism. Increased concentrations of these cytokines in serum and synovial fluid have been shown to cause local growth plate damage and inflammatory synovitis. These findings suggest that proinflammatory cytokines present in the synovial fluid may reach the adjacent growth plate and exert direct local effects.[8] However, the precise cellular mechanisms by which cytokines influence growth plate function remain incompletely understood. Alterations in intracellular signaling pathways within chondrocytes may play a critical role in growth plate dysfunction.[7]

Solitary epiphyseal overgrowth or accelerated epiphyseal closure is most commonly associated with chronic inflammation or sustained increases in regional blood flow. Inflammatory conditions and malignancies can both result in increased local perfusion, leading to excessive or accelerated bone growth.[1-3] Inflammatory trauma occurring during the healing process may also contribute to premature epiphyseal closure. Osteomyelitis and hemophilia can produce similar imaging appearances through comparable mechanisms. Additionally, arteriovenous malformations that increase regional blood flow may result in analogous findings; therefore, careful evaluation of the surrounding soft tissues is essential.[7-10]

A rare differential diagnosis is dysplasia epiphysealis hemimelica (Trevor disease), which is characterized by epiphyseal overgrowth and osteocartilaginous lesions resulting from idiopathic benign cellular proliferation.[11]

Although clinical diagnosis of JIA is usually established before radiologic confirmation due to evident inflammatory symptoms, imaging plays a crucial role in evaluating joint, bone, and soft-tissue involvement. In pediatric patients, epiphyseal changes caused by recurrent or prolonged inflammation—mediated through increased local blood flow—should be assessed separately. Early recognition of these changes may alert clinicians to the risk of growth asymmetry resulting from accelerated epiphyseal closure and prompt investigation of underlying local inflammatory processes.[9-10]

CONCLUSİON

When asymmetry in epiphyseal size or premature accelerated epiphyseal closure is detected on radiographs or cross-sectional imaging, inflammatory conditions associated with increased regional blood flow adjacent to the affected bone should be carefully investigated.

REFERENCES

1. Kronenberg, HM. Developmental regulation of the growth plate. Nature. 2003;423:332–6.
2. Grom AA, Murray KJ, Luyrink L, et al. Patterns of expression of tumor necrosis factor alpha, tumor necrosis factor beta, and their receptors in synovia of patients with juvenile rheumatoid arthritis and juvenile spondylarthropathy. Arthritis Rheum. 1996; 39:1703–10.
3. Yetgin S, Ozen S, Saatci U, et al. Evaluation of tumour necrosis factor alpha, interferon gamma and granulocyte-macrophage colony stimulating factor levels in juvenile chronic arthritis. Rheumatology. 1999;38:468–71.
4. Kutukculer N, Caglayan S, Aydogdu F. Study of pro-inflammatory (TNF-alpha, IL-1 alpha, IL-6) and T-cell-derived (IL-2, IL-4) cytokines in plasma and synovial fluid of patients with juvenile chronic arthritis: correlations with clinical and laboratory parameters. Clin Rheumatol. 1998;17:288–92.
5. Wilkinson N, Jackson G, Gardner-Medwin J. Biologic therapies for juvenile arthritis. Arch Dis Child. 2003;88:186–91.
6. Yilmaz M, Kendirli SG, Altintas D, Bingol G, Antmen B. Cytokine levels in serum of patients with juvenile rheumatoid arthritis. Clin Rheumatol. 2001;20:30–5.
7. MacRae VE, Farquharson C, Ahmed SF, The pathophysiology of the growth plate in juvenile idiopathic arthritis. Rheumatology. 2006;45:11–9.
8. de Hooge ASK, van de Loo FAJ, Bennink MB et al. Growth plate damage, a feature of juvenile idiopathic arthritis, can be induced by adenoviral gene transfer of oncostatin M—a comparative study in gene-deficient mice. Arthritis Rheum. 2003;48:1750–61.
9. Sheybani EF, Khanna G, White AJ, Demertzis JL. Imaging of juvenile idiopathic arthritis: a multimodality approach. Radiographics. 2013;33(5):1253-73.
10. Tsujioka, Y, Nishimura G, Sugimoto H. et al. Imaging findings of juvenile idiopathic arthritis and autoinflammatory diseases in children. Jpn J Radiol. 2023;41:1186–1207.

11. Degnan AJ, Ho-Fung VM. More Than Epiphyseal Osteochondromas: Updated Understanding of Imaging Findings in Dysplasia Epiphysealis Hemimelica (Trevor Disease). AJR Am J Roentgenol. 2018;4:910-9.