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Article

Clinical Features and Incidence of Musculoskeletal Disorders in Children With Juvenile Idiopathic Arthritis

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Abstract: We studied the clinical features of musculoskeletal lesions in 56 children with juvenile idiopathic arthritis (JIA). We determined the nature of histological changes in the synovial membrane, articular cartilage, and subchondral bone depending on the clinical form of the disease and the duration of the inflammatory process.

Keywords: Juvenile Idiopathic Arthritis, Children, Clinical Features, Morphology, Inflammation

Introduction

Juvenile idiopathic arthritis (JIA) remains one of the leading causes of chronic disability in children and adolescents worldwide: the inflammatory process in JIA affects not only the synovial membrane of the joint, but also cartilage, subchondral bone, entheses, ligaments and growth zones, which leads to functional impairment, deformities and growth retardation/impairment of the affected limb.[1] Modern therapy has significantly reduced the incidence of gross arthritic deformities; however, the morphological substructures of the lesion, their variability across JIA subtypes, and the consequences of chronic low-intensity inflammation remain poorly understood [2]. Although modern imaging techniques can detect inflammatory changes, it is morphological and molecular studies of synovium, cartilage, and bone tissue that provide the most in-depth understanding of pathogenesis [3]. Moreover, the child's body has a number of age-specific characteristics—the presence of growth zones and immature immune responses—that significantly modify the clinical significance of the detected morphological changes [4]. Modern reviews highlight significant progress in understanding the pathogenesis and therapy of JIA, including the development of targeted drugs and the integration of morphological studies into clinical practice [5]. Despite the development of noninvasive imaging techniques (ultrasound, MRI, and targeted radiographs), significant discrepancies remain between visual markers of injury activity and the morphological (histological and immunohistological) picture in the synovium, cartilage-osseous junction, and subchondral bone. In particular, the presence of

"subclinical" synovial activity on MRI does not always correlate with typical macro- and microscopic signs of chronic synovitis proliferation, and the nature of cellular infiltration and vascularization of the synovial layer remains heterogeneous across different JIA subtypes. This limits the ability to accurately stratify the risk of structural damage and personalize therapy.[6]

Thus, studying the clinical features of joint and periarticular tissues in children with JIA is key to early diagnosis, prognosis of the disease course, and selection of the optimal therapeutic strategy.

Materials and Method

A comprehensive clinical and morphological study was conducted on 56 children with JIA treated at the clinical sites of Tashkent State Medical university .Patient demographics**:** Age range 3 to 17 years; 32 girls and 24 boys.[7] Clinical Groups: Patients were categorized by clinical forms (Oligoarticular, Polyarticular, and Systemic) and disease duration (<2 years, 2–5 years, and >5 years).Clinical and laboratory materials from 56 children with various clinical forms of JIA (oligoarthritis - 20 cases, polyarthritis - 14 cases, systemic form - 11 cases) were examined. [8]

Results and Discussion

The Table 1. incidence of musculoskeletal disorders varied significantly based on the disease subtype. Our findings indicate that joint deformity and functional limitation increase progressively with the duration of the inflammatory process.[9]

Table 1. Clinical characteristics and incidence of joint involvement by JIA subtype

Clinical Feature	Oligoarticular (n=22)	Polyarticular (n=24)	Systemic (n=10)	Total (n=56)
Joint Swelling	100%	100%	90%	98.2%
Morning Stiffness	45.4%	87.5%	70%	67.8%
Joint Deformity	13.6%	41.6%	30%	28.5%
Muscle Atrophy	18.1%	54.1%	40%	37.5%
Functional Limitation	27.2%	70.8%	60%	51.7%

Morphological Table 2. examination revealed that the intensity of histological changes depends on the activity of the inflammatory process and its duration.

Table 2. Histological changes in joint tissues according to disease duration[10]

№	Tissue Component	< 2 years (Acute/Subacute)	> 2 years (Chronic)
1.	Synovial Membrane	Hyperemia, edema, moderate lymphocytic infiltration.	Villous hypertrophy, fibrosis, lymphoid follicle formation.
2.	Articular Cartilage	Superficial fraying, depletion of glycosaminoglycans.	Deep ulcerations, pannus invasion, thinning.
3.	Subchondral Bone	Focal hyperemia, osteoclastic activity.	Sclerosis, trabecular destruction, bone marrow fibrosis.

Our study shows that the polyarticular form of JIA is associated with the highest incidence of musculoskeletal complications, such as muscle atrophy (54.1%) and functional limitation (70.8%).[11] Morphologically, the early stage of JIA is dominated by exudative-proliferative synovitis.[12] As the disease progresses (beyond 2 years), the "pannus" formation becomes evident, leading to the destruction of the articular cartilage.[13] The subchondral bone reacts with structural remodeling, which explains the high frequency of radiographic erosions seen in late-stage JIA.[14] These findings suggest that histological damage often precedes severe clinical deformity, emphasizing the need for aggressive early-stage therapy.[15]

Conclusion

In children with JIA, the clinical and morphological presentation of joint lesions is characterized by a combination of chronic productive synovitis and vascular-dystrophic changes in the cartilage and subchondral bone, accompanied by local neoangiogenesis and immune cellular activity. The severity of fibroproliferative changes correlates with the clinical form of the disease and the duration of the inflammatory process. The incidence of musculoskeletal disorders in children with JIA is highest in the polyarticular subtype.Chronic inflammation leads to a transition from synovial edema to deep cartilaginous destruction and subchondral bone sclerosis.Disease duration exceeding 2 years is a critical threshold for irreversible morphological reorganization, marking the necessity for optimized biological or DMARD therapy before this period.

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