

Juvenile Chronic Arthritis: Growing versus Developing (Dys) function in Joints of Children

Kumar Senthil P.*, Bhat Kamalakshi, Kumar Vijaya K.***, Adhikari Prabha****, Jaganathan*******

*Professor, Maharishi Markandeshwar Institute of Physiotherapy and Rehabilitation (Maharishi Markandeshwar University), Mullana- Ambala, Haryana, India.

**Professor, Dept of Pediatrics, Kasturba Medical College (Manipal University), Mangalore, Karnataka, India.

***Associate professor, PhD Candidate, Dept of Physiotherapy, Kasturba Medical College (Manipal University), Mangalore, Karnataka, India.

****Professor, Dept of Medicine, Kasturba Medical College (Manipal University), Mangalore, Karnataka, India.

*****Professor, Department of Physiology, Kasturba Medical College (Manipal University), Mangalore, Karnataka, India.

Abstract

The objective of this review paper was to summarize the existing evidence for juvenile chronic arthritis (JCA) from an overview-based search of PubMed. Evidence from existing studies provides information on differential diagnosis of JCA based upon immunogenetic, etiopathogenetic, antibody-based and autoimmune mechanisms for JCA. Management studies were on medical, surgical, orthopedic and rehabilitation of JCA. Studies also suggested a multidimensional biopsychosocial model suitable for evaluating medical and psychosocial empirical findings which acts as a frame of reference for a multi-professional team approach in the care of rheumatic children and their families.

Keywords: Juvenile chronic arthritis; Juvenile arthritis; Pediatric rheumatology.

The objective of this review paper was to summarize the existing evidence for juvenile chronic arthritis (JCA) from an overview-based search of PubMed.

Juvenile chronic arthritis (JCA) is actually a collection of conditions that together constitute the major forms of chronic arthritis in childhood.[1] JCA has multifactorial etiology and multidimensional manifestations with an unpredictable progressive course.[2]

Classification of JCA should incorporate criteria to delineate homogeneous populations in incorporating advances in disease knowledge. Broad umbrella term such as juvenile or childhood arthritis should be used only for communicating with the lay public,

and medical nomenclature should reflect homogeneous subgroups of arthritis, without artificially proscribing a relationship between paediatric and adult disease.[3]

Immunogenetic differential diagnosis (Based upon DRw13-DRw18, DQw6-DQw18 and HLA-B27) of JCA include Juvenile rheumatoid arthritis, juvenile psoriatic arthritis, and juvenile spondyloarthropathies.[4] The immunological alterations in juvenile chronic arthritis play a major role in patient management and classification as well as their possible etiopathogenetic manifestations. [5] Autoantibody-based categorization of JCA into: presence of IgM rheumatoid factor and have erosive polyarthritis resembling adult

Corresponding Author: Senthil P. Kumar, Professor, Maharishi Markandeshwar Institute of Physiotherapy and Rehabilitation (Maharishi Markandeshwar University), Mullana- Ambala, Haryana, India. E-mail: senthilparamasivamkumar@gmail.com

rheumatoid arthritis, which occurs in minority; and the majority (90%), who are seronegative by conventional means.[6]

Woo[7] described the autoimmune pathogenetic mechanisms of JCA as follows: "Cytokines are important mediators of the immune response as well as the inflammatory response. Those concerned primarily with cell growth, differentiation and activation of cells within the immune system are called interleukins, of which there are now 18. Exposure to antigenic and environmental stimuli causes T cells to differentiate and polarise into Th1 or 2-like cells with different cytokine profiles, and requiring different cytokines for differentiation (IL-12 for Th1 and IL-4 for Th2). Homeostasis is usually restored as these cells are mutually inhibitory. Autoimmune diseases have been associated with a persistent imbalance with more Th1-like cells, which are thought to contribute to pathology. With regard to juvenile chronic arthritis (JCA), there is some preliminary evidence of this imbalance in the oligoarticular subgroup. Imbalance of pro-inflammatory cytokines, IL-1 and TNF with their natural inhibitors has also been shown to contribute to persistence of inflammation. In the case of JCA, there has been some evidence that these imbalances could account for some of the disease phenotypes."

Vandvik and Høyeraal[8] presented a multidimensional biopsychosocial model suitable for evaluating medical and psychosocial empirical findings in juvenile chronic arthritis (JCA), which acts as a frame of reference for a multi-professional team approach in the care of rheumatic children and their families.

Pain underestimation in juvenile chronic arthritis is common in Children since they often don't verbalize their pain adequately. On a pain scale they usually grade pain lower than adults. Pain perception depends on child's stage of development, with younger children showing more nonverbal pain expressions. Interference with the child's activities and its associated biobehavioural

changes finally lead to developmental disturbances.[9]

Radiological investigations

Radiological investigations in JCA include conventional radiographs (for staging various arthritides on the basis of distribution and pattern of joint space changes), Ultrasonography (for detection of joint effusions and guiding fluid aspiration), Doppler USG (for the evaluation of synovial hypertrophy and activity), Arthrography (for changes in synovium), and MRI (for changes in articular cartilage, joint effusion, synovial hypertrophy, cortical and medullary bone, cartilage and bone perfusion, and fibrocartilaginous structures).[10]

Treatments using intravenous gamma globulin, Sulfasalazine, and Methotrexate were effective for addressing different manifestations of immunogenetic mechanisms in JCA.[11] Surgical management of a child with JCA involves a multidisciplinary team composed of a pediatric rheumatologist, an orthopedic surgeon, an anesthetist, and a physiotherapist.[12] Orthopedic management of JCA should consider the involvement of all connective tissue and the multiple organ affections in this disease, especially the kidney-function and the hematopoietic system. Surgeries are often indicated for soft tissue contractures, synovial adhesions and joint deformities.[13]

Early start of a comprehensive rehabilitation programme is necessary to restore loss of function and prevent permanent handicap. It comprises of physiotherapeutic regimen (pain relief, movement expansion, training of muscular coordination and movement re-integration), occupational therapy (joint protection and self-care training), assistive devices (splinting, adapted footwear and walking aids), psychosocial (small children for restoring normal development) and vocational (adolescents for restoring productivity and self-care support) and parental education and integration of the whole family into the

rehabilitation programme.[14]

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