



Review Article

Juvenile Idiopathic Arthritis: A Deep Dive into Pediatric Autoimmune Arthritis

Sanika Ramchandra Malusare*

Post Birwad, Tal. Mahad, Dist. Raigad, Maharashtra, India.

*Corresponding author's E-mail: malusaresanika5@gmail.com

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ABSTRACT

Arthritis is an autoimmune disease that causes inflammation of the joints, with seven major types identified. One of these is Juvenile Idiopathic Arthritis (JIA), which begins before the age of 16 years. JIA is further classified into six subtypes based on clinical and laboratory findings: systemic, oligoarticular, polyarticular with or without rheumatoid factor, enthesitis-related arthritis (ERA), psoriatic arthritis, and undifferentiated arthritis. Among these, polyarticular JIA is the most common type seen in children. Although the exact cause of JIA remains unknown, it cannot be completely prevented; however, adopting a healthy lifestyle, preventing infections, early diagnosis, and proper management can help reduce its severity and complications. Diagnosis varies depending on the subtype, for example, anti-CCP antibodies are used for polyarticular JIA, while HLA-B27 testing and MRI of joints aid in diagnosing ERA. Management strategies have evolved significantly: initially, non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids were the primary treatments, but the introduction of biologic medications has greatly improved outcomes. Agents such as abatacept and tocilizumab have shown particular benefit in polyarticular JIA, while methotrexate, TNF inhibitors, and abatacept are effective in treating JIA-related uveitis. Looking ahead, the future of JIA treatment is moving towards more targeted, personalized, and less toxic therapies, including JAK inhibitors, IL-17/23 blockers, stem cell therapy, microbiome-based interventions, and nanomedicine. Early diagnosis and comprehensive management are crucial for preventing joint damage and improving long-term outcomes and adopting a healthy lifestyle, preventing infections, early diagnosis, and proper management can help reduce its severity and complications. Continued research on immunogenetics and novel biologics holds promise for personalized and more effective therapies in the future.

Keywords: Juvenile Idiopathic Arthritis, Management strategies, inflammation.

INTRODUCTION

HISTORY OF ARTHRITIS

A detailed study investigated the long-term effects of arthritis in men with idiopathic haemochromatosis. This research tracked 18 male patients for a full decade. The focus was on how joint problems developed and progressed. Researchers looked for chondrocalcinosis, a condition where calcium crystals build up in cartilage. They used X-rays to check for this. During the study, seven men developed chondrocalcinosis in their wrists. Another seven saw it appear in their knees. Three patients developed it in their hips. One man showed it in the pubic symphysis, a joint in the pelvis. Two patients had it in their spine. A key finding was that the chondrocalcinosis never lessened or disappeared in any of the patients. Treating the excess iron in the body did not change the outcome. Iron overload was managed by venesection, which involves drawing blood. This treatment did not prevent the development or worsening of chondrocalcinosis. The study

also found no link between chondrocalcinosis and the patient's age. It didn't matter if they were younger or older at the start or end of the study. The amount of iron removed through venesection also had no effect.

Arthritis itself, marked by joint space narrowing, cysts, and damaged joint surfaces, was also examined. This type of arthritis often came with other symptoms and signs. Eight patients had hand arthritis at the study's start. Thirteen others developed it later. Arthritis affecting larger joints was less common. Importantly, none of the men in this group had severe, destructive arthritis of their hips or knees. This observation contrasted with earlier findings. A review of 93 patients with idiopathic haemochromatosis seen at King's College Hospital since 1967 revealed a different pattern. In that larger group, seven patients did suffer from destructive arthritis affecting their hips. This suggests a potential difference in how joint disease manifests in idiopathic haemochromatosis patients.^{1,2}

Types of Arthritis

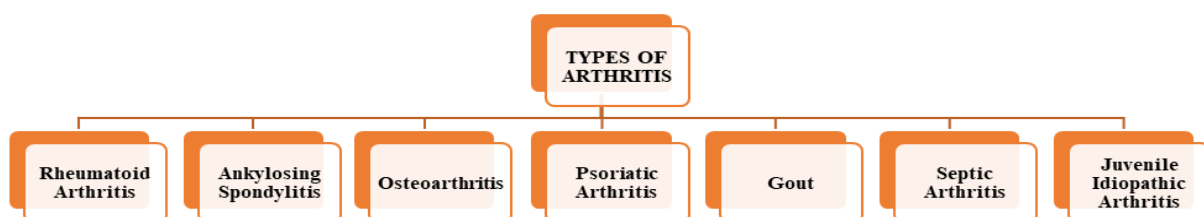


Figure 1: Types of arthritis



INTRODUCTION OF ARTHRITIS

The term arthritis originates from ancient Greek. It translates to "disease of the joints." This condition is fundamentally an inflammation of one or more joints. This inflammation can target a single joint or affect many throughout the body. The most common age group for arthritis is adults over 65. However, arthritis can appear in children, teenagers, and young adults. Data shows arthritis impacts women more often than men.³

1) Rheumatoid Arthritis

Rheumatoid arthritis (RA) was first described in 1800 by Dr. Augustin Jacob Landré-Beauvais, and the term was later coined in 1859 by Sir Alfred Baring Garrod. It is a chronic autoimmune disease that primarily affects synovial joints such as those in the hands, wrists, and knees. In RA, the immune system mistakenly attacks healthy joint tissues, leading to inflammation, pain, and swelling. The disease often affects joints symmetrically and can also impact other organs like the heart, lungs, eyes, and skin. Over time, RA can cause joint damage, affecting cartilage, bone, and ligaments.⁴⁻⁶



Figure 2: Rheumatoid Arthritis

2) Ankylosing Spondylitis

Ankylosing spondylitis (AS), with evidence found in ancient Egyptian mummies from 1559 B.C., is a chronic inflammatory disease that mainly affects the spine and sacroiliac (SI) joints connecting the spine to the pelvis. It belongs to the group of disorders called spondyloarthropathies (SA), which involve inflammation of the axial skeleton and sometimes the peripheral joints of the arms and legs.^{5,7}



Figure 3: Ankylosing Spondylitis

3) Osteoarthritis

Osteoarthritis (OA), first described in the 1800s by physician William Heberden, is a chronic joint disease that commonly affects the knees, hands, hips, and spine. It involves inflammation and cartilage breakdown, causing pain, stiffness, and swelling as bones rub together. Though there

is no cure, treatment focuses on symptom control and improving mobility through exercise, physical therapy, and medication.^{4,8}



Figure 4: Osteoarthritis

4) Psoriatic Arthritis (PsA)

Psoriasis, first identified by British dermatologist Robert Willan in 1809, is a chronic inflammatory skin disease that affects the whole body, often causing red, scaly patches on the elbows and knees. About 30% of patients develop psoriatic arthritis (PsA), an inflammatory joint condition linked to psoriasis. It affects men and women equally and usually appears between ages 40 and 50.^{9,10}



Figure 5: Psoriatic Arthritis

5) Gout Arthritis

Gout, described by Hippocrates in the 5th century BC as the "unwalkable disease," is a painful condition caused by uric acid crystal buildup in the joints. It leads to swelling, redness, and stiffness, most often affecting the big toe, but can also involve the ankles and knees. Hippocrates linked gout to diet and lifestyle, noting it was more common in men and postmenopausal women.¹¹



Figure 6: Gout Arthritis

6) Septic Arthritis

Septic arthritis is a serious joint infection usually caused by *Staphylococcus aureus*, though viruses, fungi, or mycobacteria can also be responsible. It causes severe inflammation and can lead to permanent joint damage. The infection often spreads through the bloodstream, or enters via injury, surgery, or needle use. Large joints like the knee and hip are commonly affected, but any joint can be

involved. Sudden joint pain, swelling, and fever require immediate medical evaluation.^{12,13}



Figure 7: Septic Arthritis

7) Juvenile idiopathic arthritis

Juvenile idiopathic arthritis (JIA) is a group of autoimmune disorders causing joint pain and swelling in children under 16. The immune system mistakenly attacks healthy joints, leading to symptoms that can range from mild to severe. While primarily a childhood condition, JIA can occasionally persist into adulthood.¹⁴



Figure 8: Juvenile idiopathic arthritis

ETIOLOGY OF ARTHRITIS

- **Autoimmune**

Autoimmune conditions target the body's own joint tissues. The immune system mistakenly attacks healthy cells. This leads to inflammation and joint pain.

- **Metabolic Cause**

Metabolic causes involve body chemistry issues. Metabolism can go wrong. This leads to crystals forming in joints. These crystals cause irritation. Gout is a common example of this.

- **Post-traumatic**

Some joint problems arise after an injury. Trauma can damage joint structures. This damage can lead to long-term issues. A past fracture can affect joint health later.

- **Genetic Factors**

Genetics play a role in many joint diseases. A family history increases risk. This is true for rheumatoid arthritis. Osteoarthritis risk also has a genetic link. Ankylosing spondylitis shows strong genetic ties.¹⁵

RISK FACTORS OF ARTHRITIS

- **Environmental**

Numerous factors contribute to the development of various health conditions. Understanding these risk factors is key to

prevention and management. Environmental influences play a significant role.

- **Diet**

What we eat, our diet, can impact our health. Processed foods and diets low in essential nutrients can increase risk. Conversely, a balanced diet rich in fruits, vegetables, and whole grains supports good health.

- **Lifestyle Factor**

Lifestyle choices are also critical. Our daily habits shape our well-being. This includes activity levels and stress management. Sedentary behavior can lead to weight gain and other health problems.

- **Sex**

Sex is another recognized risk factor for certain diseases. Biological differences between males and females can influence disease susceptibility. For instance, women have a higher likelihood of developing rheumatoid arthritis. This autoimmune condition causes joint inflammation and pain. Lupus, another autoimmune disorder affecting connective tissues, is also more common in women. Osteoarthritis, a degenerative joint disease, disproportionately affects women as well, particularly after menopause. Men, on the other hand, tend to experience different health challenges. Gout, a form of inflammatory arthritis characterized by sudden, severe attacks of pain, swelling, and redness in joints, is more prevalent in men. Ankylosing spondylitis, a chronic inflammatory disease primarily affecting the spine, also shows a higher incidence in males.

Women are more likely to develop:

- Rheumatoid arthritis
- Lupus
- Osteoarthritis

Men are more likely to develop:

- Gout
- Ankylosing spondylitis
- Smoking

Smoking remains a potent risk factor for a wide array of diseases. The toxins in cigarette smoke damage the body's systems. This includes increasing the risk of cardiovascular disease, lung cancer, and many other conditions. Quitting smoking significantly reduces these risks.

- **Obesity**

Obesity, defined as having excess body fat, presents another major health risk. Carrying too much weight strains the body. It increases the likelihood of developing conditions like type 2 diabetes, heart disease, and certain cancers. Maintaining a healthy weight is crucial for long-term health.^{16,17}

PATHOPHYSIOLOGY OF ARTHRITIS

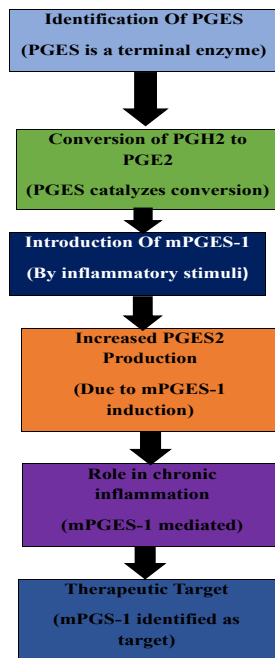


Figure 9: Pathophysiology Of Arthritis ¹⁸

Aim: “Juvenile Idiopathic Arthritis: A Deep Dive into Pediatric Autoimmune Arthritis”.

Objectives:

- To understand the etiology, pathophysiology, and immunological mechanisms underlying Juvenile Idiopathic Arthritis (JIA).
- To classify the various subtypes of JIA based on clinical features and laboratory findings.
- To identify the signs, symptoms, and diagnostic criteria used in the early detection of JIA.
- To explore the systemic complications and comorbidities associated with JIA.
- To review current treatment strategies, including pharmacological and non-pharmacological approaches.
- To emphasize the importance of early diagnosis and multidisciplinary management in improving long-term outcomes for affected children.

LITERATURE REVIEW

1. **Roy, H.S. et al. (2019):** This study compares major types of arthritis using diagnostic ultrasonography, highlighting its effectiveness in differentiating between

inflammatory and degenerative joint disorders through imaging patterns and structural changes.

2. **Ocampo, V. and Gladman, D. (2019):** The article reviews psoriatic arthritis, focusing on its pathogenesis, clinical features, and treatment options. It emphasizes the connection between psoriasis and joint inflammation, along with current therapeutic approaches.
3. **Radu, A.F. and Bungau, S.G. (2021):** This paper presents an overview of rheumatoid arthritis management, covering pharmacological, biological, and lifestyle-based therapies. It underscores early diagnosis and individualized treatment to prevent joint damage.
4. **Shirtliff, M.E. and Mader, J.T. (2002):** The review discusses acute septic arthritis, detailing its microbiological causes, clinical manifestations, diagnostic methods, and treatment strategies to prevent joint destruction and systemic complications.
5. **Hamilton, E.B.D. et al. (1981):** This longitudinal study traces the progression of arthritis in idiopathic haemochromatosis over ten years, analyzing clinical and radiological changes to understand disease evolution.
6. **Earwood, J.S. et al. (2021):** This article provides an evidence-based review of septic arthritis, emphasizing diagnostic techniques, causative pathogens, and management strategies to improve patient recovery and reduce complications.
7. **Michael, J.W. et al. (2010):** The review outlines the epidemiology, etiology, diagnosis, and treatment of knee osteoarthritis, emphasizing risk factors, disease mechanisms, and advancements in conservative and surgical management.
8. **Romão, V.C. and Fonseca, J.E. (2021):** This paper reviews the etiology and risk factors for rheumatoid arthritis, exploring genetic, hormonal, and environmental influences that contribute to disease onset and progression.
9. **Deane, K.D. et al. (2017):** The authors summarize genetic and environmental risk factors for rheumatoid arthritis, focusing on early immune activation, genetic predisposition, and potential preventive strategies.
10. **Martel-Pelletier, J. (2004):** This brief review discusses the molecular and cellular mechanisms involved in osteoarthritis, emphasizing cartilage degradation, inflammatory mediators, and structural joint changes.

TREATMENT OF ARTHRITIS

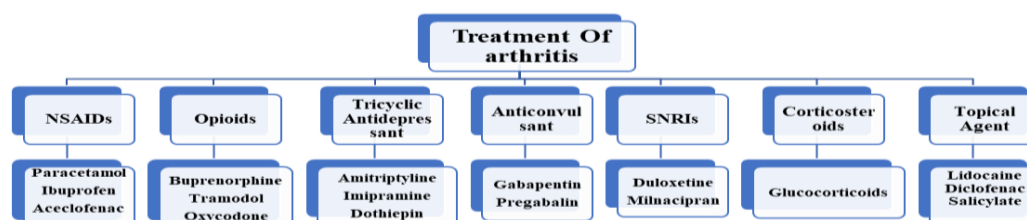


Figure 10: Treatment Of Arthritis ^{19,20}



JUVENILE IDIOPATHIC ARTHRITIS

Juvenile idiopathic arthritis, often shortened to JIA, is not one single illness. This label covers many different types of joint inflammation. These conditions all start in children. The onset must be before the age of 16. The exact cause for these illnesses remains unknown. Doctors classify them as idiopathic, meaning no known cause. In children, JIA stands out as the most common.²¹

HISTORY OF JUVENILE IDIOPATHIC ARTHRITIS

For many years, the condition affecting children was called juvenile rheumatoid arthritis. This name implied a direct link to adult rheumatoid arthritis. However, medical experts recognized differences. The disease in children was not exactly the same. To highlight this distinction, the name was updated. It became known as juvenile idiopathic arthritis, or JIA. This new term better captured its unique characteristics in young patients.

Over the past ten years, our dedicated research team put forth the key signs of juvenile rheumatoid arthritis. We based our findings on the guidelines set by the American College of Rheumatology.²²

In 1972, the American College of Rheumatology proposed a way to classify JIA. They divided it into distinct types. These included systemic, oligoarticular, and polyarticular forms. Oligoarticular arthritis affects a few joints. Polyarticular

arthritis affects many joints. Systemic JIA impacts the whole body.

European doctors felt this classification system had gaps. They believed it didn't fully cover all presentations of the disease. So, they introduced another term. They used "juvenile chronic arthritis." This classification added more specific categories. This included polyarthritis that was rheumatoid factor positive or negative. They also added juvenile spondyloarthropathy and juvenile ankylosing spondylitis. Psoriatic arthritis was another category included.

A significant step toward global consistency occurred in 1995. Experts from both America and Europe convened in Santiago, Chile. Their goal was to create a unified international system. This collaborative effort led to the establishment of the International League Against Rheumatism, or ILAR. At this meeting, the disease was officially named juvenile idiopathic arthritis. The first ILAR classification system was introduced in 1997. This happened in Durban, South Africa. It provided a standardized diagnostic framework. Later, this system was updated. The revision took place in 2001 in Edmonton, Canada. This updated ILAR classification system is crucial. It allows doctors worldwide to use a common language. This shared approach aids in accurately diagnosing and researching JIA. It helps in understanding the disease better in children across different regions.²³

TYPES OF JIA

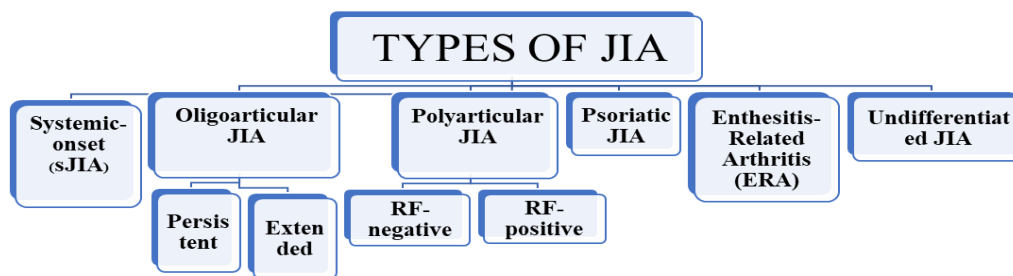


Figure 11: Types of JIA ²⁴

Table 1: Types of Oligoarticular JIA

Types of oligoarticular JIA	Definition	Joint involvement after first 6 months
Persistent Oligoarticular JIA	Affecting not more than 4 joints	≤ 4 joints
Extended Oligoarticular JIA	Affecting more than 4 joints after 6 months	≥ 5 joints after 6 months

1) Systemic-onset juvenile idiopathic arthritis (SoJIA)

Systemic-onset juvenile idiopathic arthritis (SoJIA) is a subtype of JIA that affects the whole body, not just the joints. It causes joint pain, swelling, and stiffness, along with inflammation of internal organs. A hallmark feature is a daily high fever, often accompanied by a transient rash. Children with SoJIA may experience fatigue and a general feeling of being unwell. The condition arises from the immune system mistakenly attacking healthy tissues, making daily activities challenging.²⁵

Risk Factors

- Age
- Gender
- Ethnicity ²⁶



Figure 12: Systemic-onset juvenile idiopathic arthritis

2) Oligoarticular juvenile idiopathic arthritis

Oligoarticular juvenile idiopathic arthritis (JIA) affects four or fewer joints, often emerging before age six. The knees, ankles, and wrists are commonly involved, causing pain, swelling, stiffness, and sometimes limping. It is an autoimmune condition, with the immune system attacking joint tissues, and can occasionally affect the eyes, leading to uveitis. Early diagnosis and treatment are important to manage symptoms and prevent long-term damage. Treatment usually includes anti-inflammatory medications and physical therapy to maintain joint flexibility and strengthen muscles. The outlook is generally good, with many children achieving remission.²⁷

Risk Factors

- Sex
- Environment
- Uveitis ²⁸



Figure 13: Oligoarticular juvenile idiopathic arthritis

3) Polyarticular juvenile idiopathic arthritis

Polyarticular arthritis is a type of arthritis that affects many joints, causing pain, swelling, and stiffness, often worse in the morning. It can result from autoimmune problems, where the body attacks its own tissues, or from infections. Other symptoms may include fatigue and low-grade fever. Treatment depends on the cause and often involves anti-inflammatory or disease-modifying medications, pain relief, and physical therapy to improve joint flexibility and strengthen muscles. Lifestyle changes can also help manage symptoms and support joint health.²⁹

Risk Factors

- Joint damage,
- Decreased quality of life.

Infection

Table 2: Types of Polyarticular Arthritis

Types of polyarticular JIA	RF test	Age	Gender	Joint involvement
RF negative polyarticular JIA	Negative	Younger children (<10 years)	More common in girls	Small and large joints often knees, ankles, wrists
RF positive polyarticular JIA	Positive	Older children (10-16 years)	Mostly girls	Small joints often hands wrists

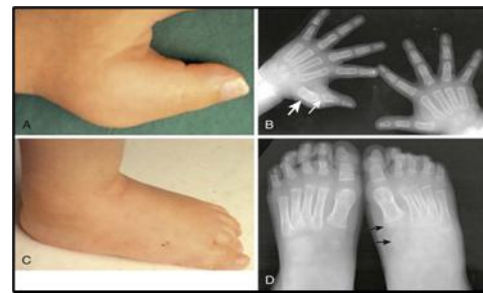


Figure 14: Polyarticular Arthritis

4) Psoriatic JIA

Juvenile psoriatic arthritis (JPsA) is a chronic autoimmune disease in children, where the immune system attacks healthy joints and sometimes the skin, causing psoriasis. Early-onset JPsA mainly affects girls and often involves small joints, with common symptoms like dactylitis (swelling of a finger or toe) and positive antinuclear antibodies (ANA). Late-onset JPsA resembles adult psoriatic arthritis, is more common in boys, and is typically associated with visible psoriasis. The condition leads to joint inflammation, pain, and stiffness, requiring medical management to control symptoms and prevent joint damage.³⁰

Risk Factors

- Genetic
- Environmental
- Obesity

Dactylitis ³¹



Figure 15: Psoriatic JIA

5) Enthesitis-related arthritis (ERA)

Enthesitis-related arthritis (ERA) is a subtype of juvenile idiopathic arthritis linked to the genetic marker HLA-B27. It primarily affects boys, usually after age six, and involves asymmetric joint inflammation, commonly in the heels, knees, and hips. As the disease progresses, children may experience inflammatory back pain in the lower back and pelvis. ERA shares features with adult spondyloarthritis. A significant complication is acute anterior uveitis, causing eye pain, redness, and blurred vision if untreated. Early diagnosis and management are essential to prevent joint and eye damage.^{32,33}

Risk Factors

- (HLA) B27
- Sex
- Age ³⁴



Figure 16: Enthesitis-related arthritis (ERA)

6) Undifferentiated juvenile idiopathic arthritis

Undifferentiated juvenile idiopathic arthritis includes patients who do not fulfill the criteria for any JIA category above described, or who meet the criteria for more than one.

Undifferentiated JIA encompasses cases where:

1. Symptoms don't match any specific JIA subtype.
2. Features span more than one subtype.³⁵

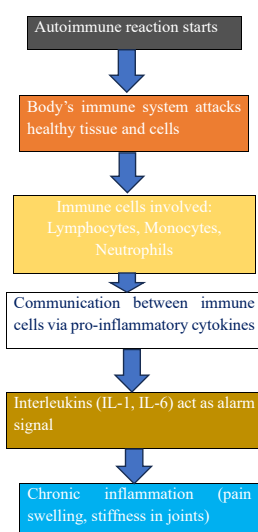


Figure 17: Pathophysiology Of JIA ⁴⁰

ETIOLOGY OF JIA

Juvenile idiopathic arthritis, often shortened to JIA, is a significant childhood illness. It affects children younger than 16 years old. The exact cause of JIA remains unknown. A key feature of this disease is arthritis that lasts for at least six weeks.³⁶

- **Genetic associations in JIA**

Recent research has explored the genetic links to JIA. Scientists reviewed these associations. They found that identical twins shared the JIA diagnosis about 25% of the time. This suggests a strong genetic component. Siblings of children with JIA are also at a much higher risk. Their chances of developing JIA are 15 to 30 times greater than the general population.

- **Environmental triggers of JIA**

Environmental factors likely play a role in JIA. Identical twins share the same genes. Yet, JIA does not develop in all

identical twins. These environmental triggers are thought to be important. For instance, infections might be one trigger. Exposure to certain viruses or bacteria could start the disease. Other environmental factors might include early life stress or diet. It could also improve treatment options for children with JIA.

- **Autoimmune Mechanisms**

JIA is the most common rheumatic disease seen in children. Joints may become damaged. It can also result in long-term disability. The body's immune system mistakenly attacks healthy tissues. This is known as an autoimmune process. These mistaken attacks cause inflammation. This inflammation affects the joints. It can also impact other body parts.³⁷

RISK FACTOR OF JIA

Young age:

Juvenile idiopathic arthritis (JIA) presents a significant risk factor. These conditions all involve joint inflammation. The key characteristic is arthritis with no known cause. Crucially, the onset of this arthritis must happen before a child turns 16. The young age at diagnosis is a critical factor in understanding JIA. Environmental influences are also considered. The impact on a child's life is profound. Daily activities can become difficult. Pain and swelling in the joints are common.

Sex:

A key risk factor for Juvenile Idiopathic Arthritis (JIA) is the patient's sex. This is particularly true for a subtype known as polyarticular arthritis. Polyarticular arthritis, meaning it affects multiple joints, shows a clear gender disparity. Research consistently shows this form of JIA occurs more frequently in girls than in boys. While JIA can affect any child, the likelihood of developing polyarticular arthritis is higher for female children.

Antinuclear antibody positivity:

A significant risk factor for Juvenile Idiopathic Arthritis (JIA) has been identified. This factor is antinuclear antibody positivity. This means a person's blood tests show these specific antibodies. These antibodies are proteins made by the immune system. They mistakenly target the body's own cells. These antibodies are common in children who develop JIA. Researchers study these antibodies to understand JIA better. Testing for antinuclear antibodies is a common practice. They indicate a potential autoimmune issue. Autoimmune diseases happen when the body attacks itself.

Uveitis:

Juvenile Idiopathic Arthritis (JIA) presents a significant risk factor. Specifically, a heightened risk of uveitis was identified. This eye inflammation appeared linked to a particular demographic. This connection was observed only in girls. Furthermore, these girls were younger than seven years old.^{38,39}



TREATMENT OF JIA

Historically, non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids were the primary treatments. Methotrexate continues to be the go-to first-line medication for most children with juvenile idiopathic arthritis (JIA). Other options like sulfasalazine and leflunomide may be used as second-line treatments. Tumor necrosis factor inhibitors (TNFi) have revolutionized care for children with polyarticular JIA, which affects many joints.

They also show significant promise for enthesitis-related arthritis and psoriatic JIA. Abatacept and tocilizumab are also proving beneficial for polyarticular JIA. The precise role of rituximab in JIA treatment is still being explored. For systemic JIA, which impacts the entire body, TNFi are less effective. However, blocking interleukin-1 or interleukin-6 offers remarkable control. Interleukin-1 blockade is particularly important. Methotrexate, alongside TNFi and abatacept, are effective treatments for managing JIA-related uveitis.

PATHOPHYSIOLOGY OF JIA

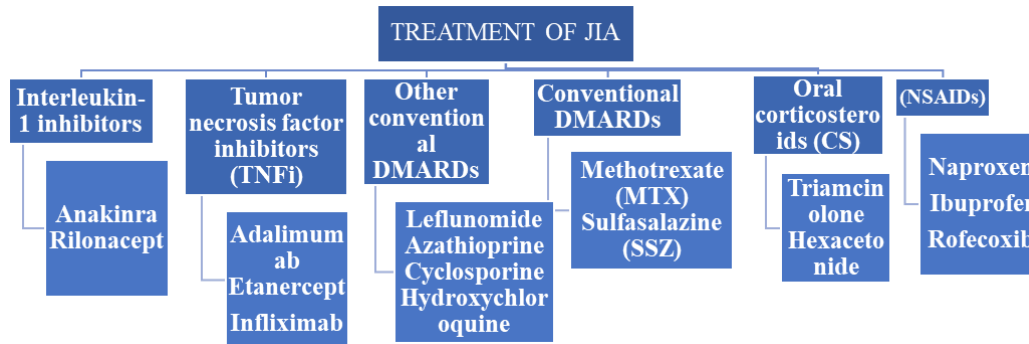


Figure 18: Treatment of JIA

• **Nonsteroidal anti-inflammatory drugs (NSAIDs)**

Nonsteroidal anti-inflammatory drugs, commonly known as NSAIDs, are a class of medications often used to reduce pain and inflammation. Examples include ibuprofen and naproxen. These drugs can cause stomach upset, ulcers, and even kidney problems.

• **Conventional DMARDs:**

✓ **Methotrexate (MTX)**

Conventional Disease-Modifying Antirheumatic Drugs (DMARDs) form a cornerstone in managing childhood inflammatory conditions. Among these, methotrexate (MTX) stands out as a highly effective treatment. Furthermore, MTX is generally well-tolerated by young patients, making it a preferred choice for many.

✓ **Sulfasalazine (SSZ)**

Sulfasalazine, often abbreviated as SSZ, has a long history in treating juvenile idiopathic arthritis (JIA). Indirect comparisons of studies indicate that MTX often proves to be better tolerated by patients. Many individuals also find MTX to be more effective. This is particularly true for patients who do not have enthesitis-related arthritis (ERA).

✓ **Other conventional DMARDs**

Leflunomide shows promise as a treatment option. It may work as well as methotrexate. It is especially useful for young patients. These are the patients who cannot tolerate methotrexate. Leflunomide provides an alternative path. Azathioprine is another immunosuppressant. This reduces inflammation. It is often used for other autoimmune diseases. Cyclosporine is a powerful immunosuppressant. It

can have significant side effects. Hydroxychloroquine is a milder DMARD.

• **Tumor necrosis factor inhibitors (TNFi)**

Five tumor necrosis factor inhibitors, or TNFi, are on the market. Doctors widely use three of these in children with JIA. These three are adalimumab, etanercept, and infliximab. This group of drugs works by blocking a specific protein called TNF. TNF can cause swelling and pain in joints. By blocking TNF, TNFi can reduce these symptoms. Adalimumab and infliximab are types of antibodies. Adalimumab is a fully human antibody. This means it is very similar to antibodies made naturally in the human body. Infliximab is a chimeric molecule.

• **Interleukin-1 inhibitors**

Interleukin-1 (IL-1) is a key signaling molecule. IL-1 fuels the body's response to injury or infection. Conditions like rheumatoid arthritis and gout involve IL-1. Understanding IL-1's role is crucial for treatment. Several medications target IL-1. These are called IL-1 inhibitors. They work by blocking IL-1's action. All are given by injection under the skin. Anakinra is one such drug. It acts like the body's natural IL-1 blocker. It's an analogue of interleukin-1 receptor antagonist. Rilonacept is another option. This protein combines parts of an antibody. It also includes parts of the IL-1 receptor. This design helps capture and neutralize IL-1. The third inhibitor is canakinumab. This drug is a monoclonal antibody. It specifically targets a form of IL-1 called IL-1 beta. By binding to IL-1 beta, canakinumab prevents it from causing inflammation.^{41,42}



Precautions to Avoid JIA

1. Healthy Lifestyle for Children

Encourage a balanced diet rich in vitamins, minerals, and omega-3 fatty acids to support immunity and joint health. Maintain a healthy body weight to reduce stress on joints.

2. Infection Prevention

Since infections can trigger autoimmune reactions, proper vaccination (as advised by a doctor) and maintaining good hygiene are important.

3. Early Detection and Regular Check-ups

If a child has persistent joint pain, swelling, or stiffness, early medical evaluation is crucial.

4. Avoiding Joint Stress

Encourage moderate physical activity such as swimming, cycling, and stretching to keep joints flexible without overloading them.

5. Medication Compliance

For children already diagnosed, taking medicines exactly as prescribed helps prevent joint damage.

6. Sunlight & Vitamin D

Adequate sun exposure and Vitamin D supplementation (if needed) may support immune regulation and bone health.⁴³

Future treatment options

Juvenile idiopathic arthritis (JIA) treatment has seen remarkable progress. This improvement is over the last twenty years. Big international groups collaborated. They worked together on studies. New, powerful medicines became available. These drugs offered better control. They helped many children feel better. We need even more progress.

1. Finding lab and imaging markers : Use of genetic and biomarker testing (e.g., HLA marker, cytokine profiles) to treatment of each child's disease subtype. These biomarkers could guide therapy. They would help doctors adjust medicines. Faster trials mean new drugs reach patients sooner. They need to plan future trials jointly.⁴⁴

2. The use of more effective drugs and their combination (Methotrexate+NSAIDs+Corticosteroid) has changed the course of the disease in many patients.⁴⁵

CASE STUDY

Gout in a 15-year-old boy with juvenile idiopathic arthritis: a case study

Initial Presentation (Age15):

Right ankle pain after sports injury → Medical ankle Pain+ Swelling+ limited dorsiflexion → X-ray: Suspicious for OCD → Physical therapy → No improvement

New Symptoms (1 year later):

Gradual left shoulder pain (no trauma) → Reduced ROM → tenderness → MRI: Synovitis → Suspected Juvenile Idiopathic Arthritis (JIA)

Progression Of Symptoms:

Multi-joint involvement (elbow, knees, wrists, fingers, ankles, PIP joints) → Morning stiffness+ symmetric joint pain

Laboratory Findings:

RF positive (24.7 IU/ml) → Anti-CCP Positive → ESR 61 mm/hr + CRP 6.7 mg/dl → ANA, anti-dsDNA, HLA-B27 negative

Diagnosis: RF-positive polyarticular JIA

Treatment: Naproxen → Methotrexate → Biologics: Etanercept → Adalimumab → Rituximab

Persistent Ankle Pain:

Arthroscopy: "crystalline white debris" (not analyzed) → imaging: severe joint damage → resembled gouty tophi

New Findings (2.5 years):

Subcutaneous nodules (forearms, elbows, knees) → Suspected rheumatoid nodules → Pain worsens after red meat → Uric acid 13.3 mg/dL → Nodules confirmed as tophi → Gout

Adjusted Treatment: Colchicine+ Allopurinol → Symptom improvement

Continued JIA therapy: Prednisone+ Etanercept+ Methotrexate+ Naproxen+ Sulfasalazine

Outcome at Adult Transition:

Persistent joint pain: knees, elbow, wrists, MCP, PIP joints → Limited mobility → Difficulty Waking

Final Diagnosis: JIA+ Gout⁴⁶

Self-investigation to explore the impact of juvenile arthritis on adolescent life: A case-study

Objective:

Study aim → Explore personal experience and feelings of an adolescent living with JIA → Understand their inner world

Methods:

Use Self- Confrontation Methods (SCM) → Technique for self- reflection → Examines thoughts and feelings closely

Case Example: Laura

18-year-old female → Poly-articular JIA, in remission since age 10 → sees rheumatologist once a year → Student+ shop worker → Few physical limits → Occasional challenges in PE classes → Positive outlook and family support → Standard questionnaires insufficient



Result (SCM Insights):

Surface: Appeared normal, arthritis not limiting→ SCM revealed deeper emotional aspects→ Emotional strain of hiding illness→ Pressure to conceal JIA→ Pushed physical limits→ Hidden struggle impacted well-being→ Self-reflection led to better integration of JIA into life→ Reduced sense of burden→ Greater sense of wholeness.

Conclusion

SCM valuable→ Explored functioning on multiple levels→ Physical experiences + Emotional responses+ Motivations → Improved self-understanding→ Greater acceptance of chronic condition

Practice Implications:

Future research→ Apply SCM to larger of adolescents→ Identify shared patterns and risk factors→ Clinical practice→ SCM promotes self-knowledge, inner drive→ Helps adolescents face emotional challenges→ Empowers coping with chronic illness.⁴⁷

CONCLUSION

Juvenile Idiopathic Arthritis (JIA) is a significant chronic rheumatic condition in children, marked by persistent joint inflammation that can result in pain, stiffness, and long-term joint damage. Although the precise cause remains unclear, abnormal immune responses influenced by genetic and environmental factors are central to disease development. Early recognition and accurate diagnosis are crucial to initiate timely treatment and prevent complications while preserving joint function. Modern therapeutic strategies, including disease-modifying antirheumatic drugs (DMARDs) and targeted biologic agents, have transformed disease management and significantly improved quality of life. Alongside medical therapy, physiotherapy, lifestyle adjustments, and multidisciplinary care play a vital role in maintaining overall health and functional mobility. Ongoing research into molecular mechanisms, biomarkers, and personalized therapies continues to offer promise for more effective, tailored interventions. From this review we conclude that to reduce the case of JIA early intervention, proactive management- healthy food, adopting a healthy lifestyle, preventing infections, early diagnosis, and proper management can help reduce its complications and progression of JIA can be minimized, helping children lead healthier and more active lives with reduced long-term disability."

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For any questions related to this article, please reach us at: globalresearchonline@rediffmail.com

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