

Chikungunya Arthritis vs Rheumatoid Arthritis: Delineating the Façade Linking Discrete Pathologies

Rachit Gandhi, Vismit Gami and Dev Desai*

Smt. NHLMMC, Ahmedabad, India

*Corresponding Author

Dev Desai, Smt. NHLMMC, Ahmedabad, India.

Submitted: 2023, June 15; Accepted: 2023, July 24; Published: 2023, Aug 31

Citation: Gandhi, R., Gami, V., Desai, D. (2023). Chikungunya Arthritis Vs Rheumatoid Arthritis: Delineating the Façade Linking Discrete Pathologies. *Int J Clin Med Edu Res*, 2(8), 242-244.

Abstract

Arthritis is a common manifestation of a wide range of pathologies. Striking parallelism in the presentation of joint symptoms can often result in misinterpretation of clinical conditions. This article tends to outline the difference between Chikungunya Arthritis and Rheumatoid Arthritis which is essential and pertinent to the idea of chronic chikungunya arthritis acting as a post-infectious, inflammatory process which can lead to faulty diagnosis. Analysing the discrepancies can offer insights into accurate diagnosis and management of both conditions.

Main Body

Chikungunya and Rheumatoid Arthritis are two distinct pathological conditions but both can clinically present with painful swollen joints[1]. Chikungunya is a re-emerging viral infection that is caused by the Chikungunya virus, an arbovirus, transmitted to humans via Aedes species mosquitoes, primarily Aedes aegypti[2]. This commonly leads to arthritis as one of its many clinical manifestations. Rheumatoid Arthritis, on the other hand, is an auto-immune, systemic inflammatory disorder which mainly affects joints but can affect many other tissues and organs[3].

Symptoms of Chikungunya, in its acute phase, typically begin with a sudden high-grade fever above 39C/102F which is followed by severe muscle and joint pain, weakness, headache, skin rashes and lymphadenopathy. The acute phase normally lasts for less than 3 months[2]. However, in some cases, the disease progresses to a chronic phase (>3 months) marked by painful, destructive inflammatory arthritis that often mimics Rheumatoid arthritis. The exact pathogenesis of Chikungunya arthritis is unclear and is supposed to be a combination of viral, immune and inflammatory factors. Whereas Rheumatoid Arthritis is a chronic, auto-immune process that is triggered by the interaction between genetic and environmental factors that leads to modification of our antigens, usually involving citrullination. Consequently, immune cells with susceptibility genes (HLA-DR1 and HLA-DR4) don't recognize the proteins as self-antigens inducing an inflammatory reaction in joint space leading to the formation of pannus (proliferative granulation tissue) which erodes articular cartilage and bone [3].

Both forms of arthritis most commonly affect middle-aged females and involve radiographic joint effusions, bone erosions,

marrow oedema, synovitis, tendinitis and tenosynovitis. Chikungunya arthritis is polyarticular, migratory and oedematous. It causes sudden onset of joint pain which is often crippling and debilitating[4]. Polyarthritis is usually symmetrical and most often involves metacarpophalangeal, proximal interphalangeal and wrist joints. It can also affect knee and ankle joints. This is usually self-limiting and short-term, lasting for a few weeks or months as in chronic chikungunya arthritis. In contrast, rheumatoid arthritis symptoms tend to develop more gradually. Early RA tends to affect smaller joints first, usually certain joints in the hands and feet. This can migrate to the wrist, knee, ankle, elbow, hip and shoulder joints as the disease advances. This is a chronic condition which is not self-limiting. In some cases, RA may go into remission but it is not curable and symptoms will return.

Chikungunya arthritis is diagnosed based on the patient's history, symptoms and physical examination. A patient's travel history and recent illness can be important determinants for the diagnosis of Chikungunya arthritis. The condition is endemic in Africa, Southeast Asia, the Indian subcontinent, the Pacific region and most probably in tropical/subtropical regions of the Americas. Patients who have concomitant co-morbidities like osteoarthritis, heart failure, respiratory disease, renal disease and diabetes are likely to have severe manifestations of acute infection. Examination of the patient's joints for pain, swelling and redness is essential. Several tests can be used to confirm the diagnosis of chikungunya arthritis [5]. These include: -

1) Serum antibody detection: IgM appears within 4 days of infection and lasts for 3 months while IgG appears late (after 2 weeks) and lasts for years. So, the detection of IgM or a fourfold rise in IgG titre is significant[6].

2) MAC ELISA involves IgM antibody capture and has high sensitivity and specificity with very low cross-reactivity with other alphaviruses and dengue.

3) Molecular method: RTPCR can detect specific viral genes in blood. This can be useful up to the first 10 days of infection before the acute phase progresses to the post-viraemic phase.

4) Biological markers like IL-1B and IL-6 are increased

5) Viral isolation in mosquito cell lines (takes 1-2 weeks) is useful for early diagnosis.

6) Imaging methods such as ultrasound and MRI can detect joint involvement, especially in patients who develop chronic arthritis.

7) Haematological finding: leukopenia with lymphocyte predominance, thrombocytopenia(rare), elevated ESR and CRP.

In the Case of Rheumatoid Arthritis, 4 of the Following Are Required for Diagnosis: -

1) Morning stiffness >1hr for 6 weeks [7].

2) Swelling of wrists, Metacarpophalangeal and proximal interphalangeal also joints for 6 weeks. RA usually spares the distal interphalangeal joint [8].

3) Swelling of 3 or more joints for 6 weeks

4) Symmetric joint swelling for 6 weeks

5) IgM Rheumatoid Factor (non-specific test) or Anti-CCP[9] (specific diagnostic test) positive

6) ESR or CRP raised on examination, Rheumatoid arthritis is characterised by: -

- Tender, swollen joints with a limited range of motion
- Trigger finger due to tenosynovitis of palms which causes contraction of muscles
- Rheumatoid nodules especially on elbows
- Cervical joint involvement which can lead to spinal cord compression
- Boutonnière deformity- Proximal inter-phalangeal (PIP) flexed and Distal inter-phalangeal (DIP) hyperextended.
- Swan neck deformity- Distal inter-phalangeal (DIP) flexed and Proximal inter-phalangeal (PIP) hyperextended.

There is no specific cure for chikungunya arthritis but there are treatments that can help relieve the symptoms. The treatment depends on the stage and extent of the disease. In the early acute stage of arthritis, supportive care including rest, hydration and pain relievers like weak opioids, acetaminophen or ibuprofen is preferred [10]. Corticosteroids are not indicated during the acute phase of the disease, as they may cause immunosuppression and worsen infection. In the chronic stage, when arthritis lasts for more than 3 months despite full doses of NSAIDs, the patient can be started on low-dose steroids or DMARDs (Disease-modifying antirheumatic drugs) like Methotrexate, Sulfasalazine or Hydroxychloroquine. Drugs should be altered if the appropriate dose does not elicit improvement in symptoms. In refractory conditions, biological agents can be used to treat severe cases of chikungunya arthritis.

For the management of Rheumatoid Arthritis, the first-line drug is Methotrexate. If the patient is not improving and symptoms persist for more than 6 months, another DMARDs (Disease-

modifying antirheumatic drugs) or non-biological agents like hydroxychloroquine or sulfasalazine is added called parallel therapy[11,12]. Another option is the step-up therapy in which a biological agent like TNF inhibitor is added which often leads to inhibition of disease progression. In refractory cases, an alternate TNF inhibitor is added while Methotrexate is continued. Effective TNF inhibitors are Infliximab, Adalimumab and Etanercept [13].

In conclusion, the key to differentiating both conditions on presentation is via thorough analysis of history, symptoms and physical examination, with the mode of progression of symptoms being a vital indicator. Lab investigations and serum markers can confirm the diagnosis. There is also significant overlap as to how arthritis is controlled in either case. However, with an accurate diagnosis, the right modality of treatment can be implemented with an appropriate choice of drug and dosage regimen corresponding to the stage and severity of the disease.

References

1. J. K. Amaral, J. B. Bilborrow, and R. T. Schoen, "Chronic Chikungunya Arthritis and Rheumatoid Arthritis: What They Have in Common," *Am J Med*, vol. 133, no. 3, pp. e91–e97, Mar. 2020, doi: 10.1016/j.amjmed.2019.10.005.
2. J. A. Ojeda Rodriguez, A. Haftel, and I. J. R. Walker, *Chikungunya Fever*. 2023.
3. K. Chauhan, J. S. Jandu, L. H. Brent, and M. A. Al-Dhahir, *Rheumatoid Arthritis*. 2023.
4. H. Pathak, M. C. Mohan, and V. Ravindran, "Chikungunya arthritis," *Clinical Medicine*, vol. 19, no. 5, pp. 381–385, Sep. 2019, doi: 10.7861/clinmed.2019-0035.
5. M. Dash, I. Mohanty, and S. Padhi, "Laboratory diagnosis of chikungunya virus: do we really need it?," *Indian J Med Sci*, vol. 65, no. 3, pp. 83–91, Mar. 2011.
6. D. M. do N. Costa et al., "Long-Term Persistence of Serum-Specific Anti-Chikungunya IgM Antibody - A Case Series of Brazilian Patients," *Rev Soc Bras Med Trop*, vol. 54, 2021, doi: 10.1590/0037-8682-0855-2020.
7. T. Sokka, "Morning stiffness and other patient-reported outcomes of rheumatoid arthritis in clinical practice," *Scand J Rheumatol*, vol. 40, no. sup125, pp. 23–27, Jan. 2011, doi: 10.3109/03009742.2011.566437.
8. B. Heidari, "Rheumatoid Arthritis: Early diagnosis and treatment outcomes.," *Caspian J Intern Med*, vol. 2, no. 1, pp. 161–70, 2011.
9. C. Chou, H. Liao, C. Chen, W. Chen, H. Wang, and K. Su, "The Clinical Application of Anti-CCP in Rheumatoid Arthritis and Other Rheumatic Diseases.," *Biomark Insights*, vol. 2, pp. 165–71, May 2007.
10. R. V. da Cunha and K. S. Trinta, "Chikungunya virus: clinical aspects and treatment - A Review," *Mem Inst Oswaldo Cruz*, vol. 112, no. 8, pp. 523–531, Aug. 2017, doi: 10.1590/0074-02760170044.
11. O. Benjamin, A. Goyal, and S. L. Lappin, *Disease-Modifying Antirheumatic Drugs (DMARD)*. 2023.
12. S. A. Saunders et al., "Triple therapy in early active rheumatoid arthritis: A randomized, single-blind, controlled

-
- trial comparing step-up and parallel treatment strategies,” *Arthritis Rheum*, vol. 58, no. 5, pp. 1310–1317, May 2008, doi: 10.1002/art.23449.
13. X. MA and S. XU, “TNF inhibitor therapy for rheumatoid arthritis,” *Biomed Rep*, vol. 1, no. 2, pp. 177–184, 2013, doi: 10.3892/br.2012.42.

Copyright: © 2023 Dev Desai, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.