

## Clinical Experience with *Ayurvedic* Treatment in Case of Rheumatoid Arthritis—*Ayurvedic* Review and Case Study

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### Abstract

*Rheumatoid arthritis is the most common inflammatory auto-immune disease with unknown etiology. Many clinical features and treatment strategies of rheumatoid arthritis are associated with multiple joint diseases. The clinical phenotype of rheumatoid arthritis is a symmetrical, deformed polyarthritis of small and large joints, which is often associated with features of the disease. The treatment typically involves ongoing management with periods of worsening symptoms followed by periods of improvement. Features of chronic synovitis are often associated with the distribution of peripheral “joints”. An example of infection is inflammation of synovium, which can damage cartilage. If it persists, it can lead to numerous systemic complications such as fatigue, subcutaneous nodules, lung involvement, pericarditis, peripheral neuropathy, vasculitis, and hematological abnormalities. Treatment is the same for all patients such as antibiotics, steroids, etc. A 49-year-old man living in Mehsana, Dela village, Gujarat, India consulted the Panchkarma OPD no.3, Saradar Ayurvedic College and Hospital in Mehsana with main symptoms of Sarvanga Sandhi shoala and Stabdhta (pain and stiffness in all joints of the body, especially 1<sup>st</sup>, 2<sup>nd</sup>, 3<sup>rd</sup>, and 4<sup>th</sup> joints of fingers of the right and left hand are more numerous in all postures, but especially when sitting or bending), Jwaranubhuti (feeling hot), agrashirah Pradeshe Prushtapradeshe, Agra Ubhaya Pada Pradeshe Twakavivarnataevam Twakarukshata (discoloration with severe dryness over frontal head and back region and bilateral front of the legs), and Vilambita Mala Pravriti (defecation usually taking a long time). Patient suffered from diabetes mellitus since 1991, tuberculosis since 1995 (completed nine months of DOT treatment), allergic asthma since 2009, osteoarthritis since 2010, rheumatoid arthritis since 2010, psoriasis since 2012, had a heart attack in 2019 (angioplasty and stent placement). The patient was currently on antiplatelet drugs, anticoagulants, beta-blockers, immunosuppressants and inject insulin in the BD/before meals 18 \*Units. The patient received Jala treatment and mud therapy (natural therapy) for nine months, but still could not get relief. Therefore, he contacted Saradar Ayurvedic College and Hospital Panchakarma OPD-3 in Gujarat, India.*

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### INTRODUCTION

The word “rheumatoid” comes from the Greek words “rheuma” and “reumatosis.” Rheumatism, which means swelling of the joints, can be aggravated by humid weather. The word ‘arthritis’ is derived from the Greek word “arthros” meaning joint. Arthritis is inflammation of the joints [1, 2]. Synovial tissue proliferates, causing fluid production, cartilage damage, marginal bone erosion, and stretching and damage of muscles and ligaments.

### Causes of Rheumatoid Arthritis (RA)

The exact cause of rheumatoid arthritis (RA) is unknown, but there are many theories about its causes [3]. It is an autoimmune disease. Possible causes include:

- *Autoimmune disorder due to the body's immune system against autologous immunoglobulin G (IgG):* It has been shown that allergies to autoantigens may be caused by enzymatic changes or free radicals of proteins such as immunoglobulins (IgG) or type II collagen, leading to the formation of idiotype or negative antibodies in IgG glycosylation.
- *Genetics:* This disease is caused by activation of T lymphocytes.
- *Infection:* Chronic infection of the joint or accumulation of microbial material in the tissue can lead to infection.
- *Smoking:* It is a risk factor for RA in Caucasians; risk increases in non-smokers.
- *Hormonal Factors:* Use of oral contraceptive pills (OCPs) may reduce the incidence of RA. Since the effects of OCPs containing estrogen are at their highest level, estrogen is thought to be protective. It is known that the incidence of RA is higher in women before menopause and that the disease regresses during pregnancy.
- *Economic Activity:* Low economic activity is associated with the most serious diseases. The deformity is based on wear of the bone by the proliferation of synovial tissue. This process causes the ligaments around the joints to loosen and the muscles to subluxate, causing swelling of the joints and many tissues in the body.

### PATHOLOGY

When irritating substances activate the immune system, many immune mechanisms come into play, causing joint damage and joint deformities in RA. First the cartilage and then the bones are eroded by the proliferating synovial tissue. This process causes the ligaments around the joints to loosen and the muscles to subluxate, causing swelling of the joints and many tissues in the body. Several studies showed that the joints protect against diseases. In individuals with this gene, by activating helper T cells in response to the patient's immune system (possibly an infection), activated CD4 cells produce cytokines that have two main effects—activating macrophages and other cells in the joint area, and freeing the tissue. Enzymes and other factors control inflammation. Activation of B cells leads to the production of antibodies. The resulting autoimmune response damages joints and is thought to play an important role in infection. Antigen microorganism MHC class II (genetic susceptibility) Cytokine CD4 → T cell, B cell activation. Activation of macrophages, production and storage of cytokines, immune system, proliferation of fibroblasts, synoviocytes and chondrocytes. Joint damage releases collagenase, PGE2, and other enzymes to form old bone, cartilage, fibrosis, and ankylosis [4]. Histologically, affected joints exhibit synovitis, enlargement and hypertrophy of the synovial cell lining, sometimes leading to the formation of villi (digital protrusions). The subsynovial connective tissue is densely infiltrated by lymphocytes, plasma cells, and macrophages, which often form the lymph nodes. Neovascularization is evident in plump endothelial cells that express high levels of adhesion molecules. The synovium is highly vascular, inflamed, and repeatedly covered with bony cartilage known as the “pannus.” Once the joint is fully formed, periarticular soft tissue edema often occur, presenting as a spindle-shaped swelling in the proximal interphalangeal region. In chronic stage, the beneath of cartilage becomes worn and damaged over time. The bone below the joint can also be attacked and eroded. Eventually, the pannus in the joint area causes fibrosis, increases in size, becomes cloudy under the influence of bacteria and loses some of its mucin content. Although the synovium is infiltrated by chronic inflammatory cells which are present in the synovial fluid. The presence of neutrophils indicated granular inclusions of phagocytosed immune complexes [5].

### PRODROMAL SYMPTOMS

Prodromal symptoms may precede joint swelling by several weeks. Morning stiffness, fatigue, loss of appetite, general weakness, nonspecific musculoskeletal symptoms such as pain, weight loss, malaise, muscle pain, limb tingling, joint stiffness, and more are common prodromal symptoms of RA [6].

## CLINICAL PRESENTATION AND SYMPTOMS

There are seven types of RA presentation. They are listed below:

1. *Classic*: Pain, stiffness and inflammation of the small joints of the hands and wrists. Symptoms intensity and severity changes from day to day.
2. *Palindromic*: Intermittent episodes of pain, swelling and redness, usually one joint followed by rapid return to normal after a few days.
3. *Systemic*: Weight loss, pleurisy and pericarditis, but minimal joint involvement.
4. *Polymyalgic*: Pain and stiffness in the shoulders and hip with subsequent synovitis.
5. *Monoarthritic*: Involvement of one joint, usually the knee.
6. *Acute Onset*: Sudden nocturnal onset with stiffness and pain.
7. Generalized lymphadenopathy

## CRITERIA FOR DIAGNOSIS

Following criteria were established by the American Rheumatism Association (A.R.A; 1988):

- *Morning Stiffness*: Stiffness in and around the joints lasting for an hour before showing maximum improvement. Arthritis in three or more joint areas, as observed by a doctor, with pain accompanied by soft tissue swelling or joint effusion, not just bony overgrowth. This can occur in any of 14 possible joint areas (right or left). Arthritis in hand joints involves swelling in at least one area in the wrist, metacarpophalangeal (MCP), and proximal interphalangeal (PIP) joint.
- *Symmetric Arthritis*: Bilateral involvement.
- *Rheumatoid Nodules*: Subcutaneous nodules above bony prominences.
- *Rheumatoid Factors*: By any method that were positive in <5% of normal subjects [7].
- *Radiological Changes*: On a PA view radiograph of the hand and wrist, there must be evidence of erosion or definite bone decalcification in or near the affected joint areas. For a diagnosis of RA, patients must meet at least four of the seven criteria, with the first three criteria being present for at least six weeks. In the hand, ulnar deviation, swan neck deformity, Boutonniere deformity, Z-deformity, piano key deformity, club finger deformity, trigger finger deformity are reported. In knees, Jen valgus, Flexible contracture, Backer's cyst are common conditions. In foot, Foot aversion, Foot drop, Hammer toe, Hallux valgus, Cock-up toe are generally observed. Extra-articular manifestations are depicted in Table 1.

*Examination*: Complete blood count (CBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), antinuclear antibody (ANA), Anti CCP antibodies are detected in 60–80% of patients. X-ray, magnetic resonance imaging (MRI) scan, Synovial biopsy, synovial fluid analysis, arthroscopy, renal biopsy, and pulmonary biopsy are done, if needed.

**Table 1.** Extra-articular manifestations [8].

No.	System	Involved features
1	Systemic	Fever, fatigue, weight loss, susceptibility to infection
2	Musculoskeletal	Muscle-wasting, bursitis, tenosynovitis, osteoporosis
3	Hematological	Anaemia, eosinophilia, thrombocytosis
4	Lymphatic	Splenomegaly, Felty's syndrome
5	Nodules	Sinuses, fistulae
6	Ocular	Episcleritis, scleritis, scleromalacia, keratoconjunctivitis sicca
7	Vasculitis	Digital arteries, ulcers, pyoderma gangrenosum, mononeuritis multiplex, visceral arterities
8	Cardiac	Pericarditis, myocarditis, endocarditis, conduction defect, coronary vasculitis, granulomatous aortitis
9	Pulmonary	Nodules, bronchiolitis, pleural effusions, fibrosing alveolitis, Caplan's syndrome
10	Neurological	Cervical cord compression, compression neuropathies, mononeuritis multiplex, peripheral neuropathy

## Differential Diagnosis

Many disease seems like RA but it could be differentiated with the given symptoms:

Such as arthritis based on signs and symptoms and biochemical tests. These diseases are:

1. *Gout*: Laboratory tests show the presence of uric acid in the blood. In this case, taking colchicine may have an effect.
2. *Rheumatic Fever*: First, the onset typically occurs before 15 years of age in 70% of patients. It is marked by frequent joint pain and fever, with fusiform joints of the fingers being rare. Myocarditis, endocarditis, and nodules appear in various histological images [9].
3. *Polymyalgia Rheumatica*: In this case, the ESR is very high and symptoms of peripheral disease are rare (The onset of RA in adults is similar to polymyalgia rheumatica).
4. *Nodular Polyarthritis*: It may mimic RA but there may be minor changes. Severe symptoms and necrotizing vasculitis may occur in the early stages of polyarthritis, but the joint deformity and deformity of RA rarely occur in the later stages.
5. *Osteoarthritis*: Occurs in elderly people with complete lack of systemic features of RA such as fever, weight loss, fatigue etc. The duration of morning stiffness and joint swelling is shorter as compared to RA, and the radiological appearance differs. There are no subcutaneous nodules or rheumatoid factor (RF) present. Typically, Heberden's nodes are associated with the distal interphalangeal (DIP) joints, and the ESR is usually normal. Primarily, the weight-bearing joints are affected.
6. *Systemic Lupus Erythematosus (SLE)*: This condition features joint involvement that is not symmetrical and ankyloses and erosions are uncommon. It is marked by numerous autoantibodies, circulating immune complexes, and significant immunologically induced tissue damage.
7. *Psoriatic Arthropathy*: Characteristic skin and nail lesions may be present, typically involving the DIP joints. The RF is negative.

## TREATMENT OF RA

The goals of RA treatment are: (i) to reduce inflammation, (ii) to reduce pain, (iii) to prevent joint pain, (iv) to maintain function, (v) to maintain cooperation.

Management of patients with RA is associated with many problems experienced by these individuals in their work and social relationships.

1. *Education*: Provides information about the disease and treatment.
2. *Movement*: Most of the pain and stiffness in RA comes from specific tissues such as muscles and tendons. Patients should exercise to develop and maintain the muscles around the joint.
3. *Physiotherapy/Occupational Therapy*: Physiotherapy methods such as electrotherapy (ultrasound or intervention), prevention of joints of bones to prevent deterioration during acute pain and advice on adaptation or other methods of work have an important role [10].
4. *Diet*: Diet recommendations include weight loss and taking fish oil or evening primrose oil. Fish oil replacement led some patients with RA to reduce or discontinue nonsteroidal anti-inflammatory medications.
5. *Treatment*: Once RA is diagnosed, treatment with disease-modifying anti-inflammatory drugs (DMARDs) should be started immediately.
6. *Nonsteroidal anti-inflammatory drugs (NSAIDs)*: NSAIDs do not impact long-term disability but can alleviate symptoms such as pain and stiffness. Response to NSAIDs varies from patient to patient, and it is important to find the right NSAID for each patient. It also has a negative effect on kidney function and the gastrointestinal tract, so it should be used with caution [11].
7. *Glucocorticoid Therapy*: Another treatment option includes using low doses of oral glucocorticoids. Although low-dose corticosteroids are widely used to reduce the signs and symptoms of inflammation, recent evidence suggests that they may delay the onset and progression of bone erosion. However, long-term treatment is not recommended due to many side effects.

8. *Disease-modifying Anti-inflammatory Drugs (DMARDs)*: Third party drugs include a variety of drugs classified as anti-inflammatory drugs or slow-acting anti-inflammatory drugs. Order start is often late (4 weeks to 3 months) and order processing varies. They can change diagnostic markers such as ESR and CRP. Recently, a combination of DMARDs has shown promise in managing the signs and symptoms of RA.
9. *Anticytokines*: The fourth group of drugs that is, cytokine neutralizers, significantly affect the signs and symptoms of RA and also cause joint damage [11].
10. *Immunosuppressive Therapy*: The fifth drug is an immunosuppressive and cytotoxic drug that has been shown to improve the disease process in some patients.

## Orthopedic Treatment

### Non-Operative Methods

1. *Physiotherapy*: It includes heat therapy such as wax baths and hot water applications for relief of symptoms, joint mobilization exercises to preserve joint function, and muscle-building exercises to increase strength.
2. *Occupational Therapy*: To assist the patient in adapting their occupational needs for optimal comfort.
3. *Rehabilitation*: Its purpose is to enhance patient function through the use of devices such as braces and walking aids [12].

## CASE PRESENTATION

A male patient aged 49 years living in Dela village at Mehsana, Gujarat, India approached to the Panchkarma OPD-3 at Saradar Ayurvedic College and Hospital, Mehsana, Gujarat with chief complaints of *Sarvanga Sandhi shoola* and *Sthabdhta* (whole body joints pain with stiffness specially 2<sup>nd</sup>, 3<sup>rd</sup>, 4<sup>th</sup> finger in right and left hand severity more with all position but specially sitting, bending and after sitting position), *Jwaranubhuti* (feverish feeling), *agrashirah Pradeshe Prushtapradeshe*, *Agra Ubhaya Pada Pradeshe*, *Twakavivarnataevam Twakarukshata* (discoloration with severe dryness over frontal head and back region and bilateral anterior aspect of legs), *Vilambita Mala Pravriti* (defecation taking more time) The past history of the patient was recorded. The patient was suffering from diabetes mellitus since 1991, tuberculosis since 1995 [medicine course (DOT medicine) was completed for nine months], allergic asthma since 2009, osteoarthritis since 2010, RA since 2010, psoriasis since 2012, heart attack in 2019 (that time angioplasty was done and one stent was placed). Now patient took anti-platelet, anti-coagulant, beta-block, immuno-suppressant, Injection insulin 18\* Units BD/before meal. Patient was given *Jala* therapy, Mud therapy (Naturopathy treatment) for nine months but could not get relief. So he came to Panchkarma OPD-3 at Saradar Ayurvedic College and Hospital, Mehsana, Gujarat, India. In evaluating the patient's progress, blood reports on 20/2/2023 (Table 2) indicated significant changes in haemoglobin (Hb), white blood cells (WBCs), CRP, random blood sugar (RBS), and ESR levels. Subsequent tests on 27/2/2023 (Table 3) further illustrated the evolving trends in these parameters.

**Table 2.** Blood reports (On 20/2/2023).

Hb	12.5%	Total RBC	4.5 cc/mm
Total WBC count	7300	S. uric acid	2.9
Platelets	2.90 lacs	ESR	79
CRP	11.4	S. Creatinine	0.80 mg%
RBS	308 mg/dl		

Hb: Hemoglobin, WBC: white blood cell; CRP: C-reactive protein, RBS: random blood sugar, ESR: erythrocyte sedimentation rate.

**Table 3.** Blood reports (On 27/2/2023).

FBS	137.8 mg/dl	HbA1c	13.3%
PPBS	291.2 mg/dl	Average blood glucose	335.01 mg/dl
Postprandial urine sugar	2+		

FBS: fasting blood sugar, PPBS: postprandial blood sugar, HbA1c: glycosylated haemoglobin.

**Samprapati**

- *Dosha-kapha pradhana Tridoshajavyadhi*
- *Dushya-rasa, rakta, mansa, Meda, Asthi*
- *Agni-Jatharagni, Dhatvagni*
- *Srotas-Annavaha, Rasavaha, Raktavaha and Pranavaha*
- *Dushti-Sanga, Atipravriti*
- *Vyadhiudbhavasthana-PakwashayagataVyadhi*
- *Adhithana-Pakwashaya, Phupphusa, Twacha, Asthi*
- *Avastha-Chirakari*
- *Prabhava-Kruchhasadhya, Asadhya.*

*Panchatikta Ghruta* and *Trikatu Churna* were prescribed to the patient by the Panchkarma OPD-3 at Saradar Ayurvedic College and Hospital, Mehsana, Gujarat, India (Table 4).

**PROBABLE MODE OF ACTION**

*Panchatikta Ghruta*: It has *Nimba, Vasa, Guduchi, Patola, Kantakari, Triphala, Ghruta*. It has *Sukshma strotogami Guna* due to its *Tikta Rasa*. *Ghruta* itself has *Yogavahi Gunas*; others include *Rasayana, Balya, Vata-Pitta shamaaka, Ropana, Dipana-Pachana* properties. It stimulates *Agni* and maintain *dhatu pariposhanakarma*, so it directly works on seven *dhatu*s, *Tridoshas*. All contents having antibacterial, antiviral, anti-inflammatory, and nourishing properties so it directly boosts immunity and properly increases metabolism and excretes waste very easily from the body because of its *Snigdha Guna* [13].

**Table 4.** Medicine.

Medicine	Dose	Timing
<i>Panchatikta Ghruta</i>	10 ml/BD	7 am-----7 pm
<i>Trikatu Churna</i>	2 g in BD mixed with <i>Grita</i>	7 am-----7 pm

**Table 5.** Observation after treatment.

	20/2/23	27/2/2023	4/3/2023	3/4/2023	7/5/2023	14/6/2023	7/7/2023	10/8/2023	7/9/2023
<i>Sarvanga Sandhi shoola</i> and <i>Sthabdhta</i> (whole body joints pain with stiffness)	++++	++++	++	+	-	-	-	-	-
<i>Jwaranubhuti</i> (feverish feeling)	+++	+			-	-	-	-	-
<i>Agrashirah Pradeshe Prushtapradeshe, Agra Ubhaya Pada Pradeshe Twakavivarnataevam Twakarukshata</i> (discoloration with severe dryness over frontal head and back region and bilateral anterior aspect of legs),	++++	++++	++	+	-	-	-	-	-
<i>Vilambita Mala Pravriti</i> (defecation taking more time)	++++	+++	++	+	-	-	-	-	-

*Trikatu Churna*: It contains *Sunthi*, *Maricha*, and *Pippali*. It works on *Jatharagni* as well as all *Dhatvagni*, so *Dhatunirmitikrama* will be in proper way and maintain *dhatu pariposhana karma*. It works on *Amavasta* in the body. All contents have *Sukshma strotogami*, *Deepana-Pachana* properties, so it works on *Amavasta*, *Sothavastha*, *Pratishaya*, *Kaphaja Vyadhi*, *Agnimandhya* in the body. The presented patient had multiple diseases since a long time, so *Dhatvagnimandhya* occurs in the body, *Kapha* as a *mala roopa* continuously produced vice versa *Ama* increased in the body which play a causative factor for other diseases. Also the patient had multiple medication since long time, so *Amavisha* is also generated that causes other diseases [14].

## OBSERVATION

The symptoms gradually decreased as shown in Table 5.

## CONCLUSION

The present study concluded that there is not a single disease in *Ayurveda* which can be completely correlated as multiple disease syndrome. The pathological condition of the present case study was cured because of proper *Ayurvedic* concepts and potent medicines. Multiple disease syndromes can also be benefited by oral medicines.

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