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CASE BASED REVIEW

Leprosy in a rheumatology setting: a challenging mimic to expose

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Abstract Leprosy can manifest arthritis both as a complication and a comorbid disorder and can be a challenging differential diagnosis in rheumatology practice due to several common features. Uncommonly, it may present as acute severe polyarthritis with skin lesions and neurological deficit or a digital vasculitis and gangrene. We demonstrate this profile in a retrospective case series analysis of 33 patients (13 females, median age 55 years) in a community-based clinic setting over the period 1998–2012; an electronic search of case records of 41,000 patients was carried out. Rheumatoid arthritis (RA) coexisted in seven patients (three lepromatous, two tuberculoid, and two polyneuritic). Serological rheumatoid factor and antinuclear antibody were often false positive. Several patients of RA were on long-term supervised methotrexate. Rheumatologists should be aware of this clinical mimic to avoid errors in diagnosis and management.

Keywords Arthritis · Hansen's disease · Infection · Leprosy · Rheumatology

Introduction

Leprosy is an ancient infectious disease that is only known to afflict human beings and continues to be an important prevalent disease in several developing nations. The global registered prevalence of leprosy at the beginning of 2012 stood at 181,941 cases [1].

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Though routinely seen by dermatologists, leprosy is reported to present uncommonly in rheumatology practice as primary arthritis or a coexistent infection or a complication of therapy. Rheumatologists should be aware of this disease since some form of joint involvement is reported to occur in 75 % of cases of leprosy and at times may be the only obvious manifestation [2].

Joint pain and arthritis are usually seen during the reactive inflammatory phase. In addition, Hansen's disease may uncommonly cause a chronic erosive deforming arthritis, involving both large and small joints, which resembles rheumatoid arthritis [3]. It is also known to present with clinical and laboratorial findings that mimic autoimmune connective tissue diseases and vasculitis [4].

In another rare parallel, leprosy has been reported in patients with human immunodeficiency virus (HIV) infection on antiretroviral therapy, as a manifestation of immune reconstitution inflammatory syndrome [5].

We present a retrospective analysis of a case series of 33 patients examined and diagnosed as Hansen's disease. The aim was to describe the clinical profile relevant to rheumatology practice and highlight the error of a misdiagnosis. A brief review of literature is also presented.

Materials and methods

The present study was carried out in the Center for Rheumatic Disease (CRD), Camp, Pune, which is a private community-based referral facility in the state of Maharashtra (www.rheumatologyindia.org). CRD has carried out several community arthritis camps and population surveys [6, 7] in the region and is a recognized research institute with facilities for post-masters doctorate program (PhD). We examine about 85–100 patients daily.

We retrospectively carried out a cross-sectional electronic search of case records in the referral database of CRD

between 1998 and 2012 and retrieved case records of patients who had been diagnosed with leprosy and or leprosy–arthritis during examination in the center (other search words included Hansen's arthritis and lepra reaction). Forty-one thousand case records were screened.

Every patient was examined by a rheumatologist, and findings are recorded in a standard case record form (CRF). The CRF includes a 68/66 joint count chart for pain/tenderness and swelling (as recommended by the American College of Rheumatology), pain visual analog scale (VAS), global assessment, health assessment questionnaire, and detailed systemic evaluation. All patients were also examined by a dermatologist (EC). Some patients were referred to a neurophysician and, if required, evaluated with a nerve conduction study. The diagnosis of Hansen's disease was essentially clinical and supported by a demonstration of acid-fast bacilli (AFB) in skin biopsy.

Diagnostic work-up included complete hemogram, routine urine analysis, metabolic hepatic renal and lipid profile, and serological markers like rheumatoid factor (RF) and C-reactive protein (CRP) by nephelometry. Skin testing/biopsy was often performed by the dermatologist. Slit-skin smear from the ear lobe was obtained and examined for AFB by Ziehl–Neelsen staining method, and the bacteriological and morphological indices were evaluated by the dermatologist. Sural nerve biopsy was carried out in few cases. A summary of the clinical profile of three patients with challenging clinical scenarios is also presented.

Results

Thirty-three patients (20 males, median age 55 years) were identified. All the cases had follow-up records for at least 6 months. None of the patients had a positive family history of leprosy, and all belonged to middle socioeconomic status with an almost equal divide between urban and rural regions.

Inflammatory arthritis was diagnosed in 28 patients (7 rheumatoid arthritis (RA) and 21 undifferentiated), nonspecific arthritis in 5 patients, and vasculitis in 1 patient. Twenty-two, nine, and two patients, respectively, were classified as lepromatous leprosy (LL), tuberculoid leprosy (TT), and polyneuritic leprosy. Table 1 describes the demographic and other selected relevant features.

Leprosy was detected in 19 patients who had presented earlier with primary inflammatory arthritis (often RA-like); in five patients (three women), the flare in arthritis was associated with features of type I reaction. On careful assessment, at least 20 cases had been suspected/diagnosed of leprosy prior to developing chronic arthritis. An almost simultaneous onset of arthritis and detection of leprosy was observed in 13 patients. There was a male preponderance (Table 1) in the study. RA coexisted in seven patients

Table 1 Rheumatoid arthritis (RA) and undifferentiated inflammatory arthritis (IA-U) (percentage frequency)

Features	RA (n = 7)	IA-U (n = 21)
Male	71	57
Female	29	43
Median age years (range)	55 (29–60)	48(20–72)
Median duration arthritis (years) (range)	3(0.5–15)	4 (20–37)
Patients developing arthritis after onset of Hansen's disease	43	62
Patients developing Hansen's disease after onset of arthritis	57	19
Patients developing Hansen's disease and arthritis simultaneously	0	19
Lepromatous leprosy	42	76
Neuritic	29	0
Tuberculoid type	29	24
Methotrexate	58	15
Prednisolone	72	53
Dapsone	58	67
Clofazimine	43	53
Rifampicin	29	43
Type II reactions	15	29
Type I (upgrading)	0	10
Type I (downgrading)	15	10

(three in LL and two each neuritic and TT), and it preceded Hansen's disease in three cases. The patients had symmetrical polyarthritis. Articular deformities akin to RA were seen in 12 patients; in five patients, it was "claw hand" (Fig. 1) rather than actual articular deformity. At least three patients with obvious finger flexion deformities and inability to make a fist did not show any radiological abnormality typical of RA.

Two patients showed features of digital vasculitis with symptomatic oral sicca. None of the patients showed features of enthesitis. On close examination, the skin lesions were often characteristic of Hansen's disease. Twenty-two patients had clinical peripheral neuropathy (Table 2) with

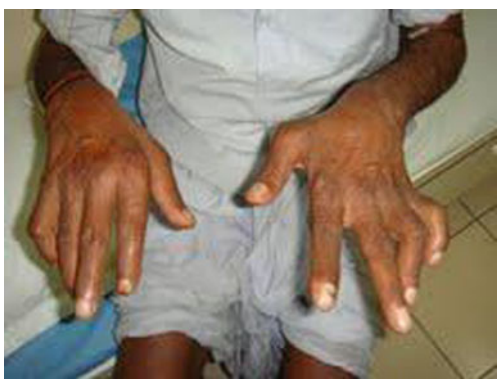

Fig. 1 Patient showing claw hand in both the hands

Table 2 Neurological manifestations ($n = 21$) with lepra reactions ($n = 15$)

Neurology symptoms with reaction	Tuberculoid leprosy ($n = 21$)	Lepromatous leprosy ($n = 10$)	Neuritic ($n = 2$)
Dominant sensory	5	8	0
Dominant sensory motor	1	5	2
Thickened nerve	4	3	0
Claw hand/feet deformity	1	1	2
Trophic ulcers	2	3	0
Diagnostic NCV	1	1	2
Lepra reaction type I (downgrading)	2	3	0
Lepra reaction type II	0	4	0

predominant glove and stock-type pattern of sensory loss; pure sensory loss was found in 13 patients. Five patients (three lepromatous) had feet digital trophic ulcers in addition to neuropathy. Nerve conduction velocity (NCV) was required to confirm the typical axonal type neuropathy in four patients (two tuberculoid, one lepromatous, and one neuritic leprosy).

Fifteen patients (13 on anti-Hansen's disease drugs) were seen in reactional states [type I downgrading/upgrading and type II (erythema nodosum leprosum, ENL)] (Table 1). Sixty percent of the reactions in the study were type II, encountered in patients of lepromatous leprosy. At least four patients of tuberculoid leprosy with inflammatory arthritis and on antiarthritis therapy suffered from downgrading reaction and moved towards the lepromatous profile. Patients in the reactional state were usually found to look sick with puffiness on the face, hands, and feet and mild edema; often, skin lesions looked exaggerated, and some even showed small irregular ulcers over the hands and feet. One patient presented with a tender hepatomegaly.

Methotrexate was used to control severe RA. However, two patients on long-term methotrexate developed Hansen's skin lesions (Fig. 3). In 57 % of cases (88 % lepromatous), the skin clipping was shown to be positive for AFB stain; AFB status could not be confirmed in eight patients with history of leprosy in the past. We carried out full thickness skin biopsy/histopathology study in two patients (one each lepromatous and tuberculoid) to confirm the diagnosis. Other rheumatology work-up showed that in patients with inflammatory arthritis and leprosy, 26.6 % were seropositive for RF and 37.5 % were seropositive for ANA. Several patients were tested for HIV, serological tests for syphilis, and other autoantibodies performed in rheumatology practice (including anticyclic citrullinated peptide/anti-CCP and found seronegative).

Case 1 A 33-year-old nonsmoker male, a known case of Hansen's disease (skin biopsy-proven borderline lepromatous) on supervised long-term treatment (prednisolone and clofazimine) since 4 years was referred to the rheumatology clinic with an acute onset of feeling cold in the index, middle, and ring finger of the left hand; since 1 month, he recalled episodic severe pain and burning sensation in the fingers. The patient did not have diabetes or any other relevant disease. Ten days later, he developed painful gangrene of the digital pulps of the index, middle, and ring finger of the left hand (Fig. 2).

On examination, he also had atrophied, nodular, and healed ulcerative scars over the face, neck, and extremities with few scattered diffuse hypopigmented hypoesthetic lesions; ulnar and peroneal nerves were thickened. New skin lesions or any other systemic abnormality was absent. Detailed laboratory investigations showed normal erythrocyte sedimentation rate (ESR) with mild normocytic normochromic anemia, marginally raised C-reactive protein; seronegativity for RF, ANA, anti-CCP, and anticardiolipin antibodies; and seropositivity for isolated anti-ds DNA (ELISA) in low titer. Routine cardiac evaluation (including Doppler/echo) was normal, and peripheral blood flow Doppler studies suggested reduced distal arterial blood flow in the right arm. A right subclavian artery angiography was consistent with focal distal arteritis lesions. Other causes like cervical rib and proximal subclavian/arterial compressive lesions were excluded. A diagnosis of vasculitis was made and considered to be a likely complication of leprosy. A short course of tapering high dose of orally administered prednisolone (begun at 30 mg daily and 6 weeks later

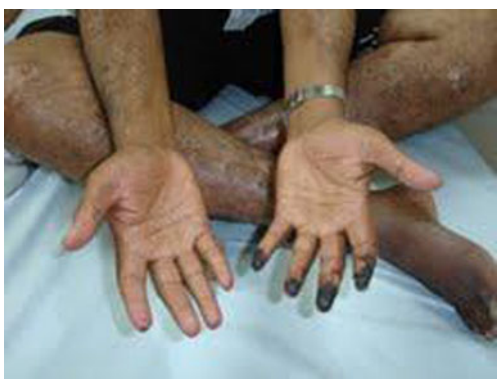

Fig. 2 Case 1 showing digital pulp gangrene in the left hand

on 5 mg daily), orally administered hydroxychloroquine, and antiplatelet agents was added to ongoing anti-leprosy drugs. The patient showed steady recovery, and the affected digital pulps healed with scars and some loss of pulp substance.

*Case 2*A 55-year-old male patient with chronic RA of 9 years in duration was well controlled (near remission) with long-term supervised administration of leflunomide and methotrexate. The patient developed acute urticaria-like rash on the neck, and upon consultation with the dermatologist, oral hydroxychloroquine and a short course of oral prednisolone were added. After a modest response, the patient developed erythematous papulonodular nontender itchy lesions (Fig. 3) all over the body, but maximally over the lower trunk and abdomen along with polyarthralgias and severe painful right ankle swelling and a limping gait. The patient was afebrile and did not have any other systemic abnormality. Leflunomide was suspected to have caused the skin lesions and was stopped. Serum uric acid level was significantly elevated and allopurinol, prescribed. Orally given diclofenac was added for the symptomatic relief of arthritis but was discontinued (? hypersensitivity) because the skin lesions worsened (relapse of generalized urticaria lesions). The skin lesions were opined to be a drug-induced leucocytoclastic reaction/vasculitis; allopurinol was also stopped. A moderately high dose of orally given methyl prednisolone with a slow taper (over weeks) was started. A concurrent work-up was negative for ANA and other collagen vascular diseases. A week later, the patient had made little recovery, and a skin biopsy was carried out which demonstrated AFB and a histopathological picture consistent with a lepromatous reaction.

The patient was started on multidrug treatment (dapsone, rifampicin, clofazimine), and the patient responded clinically (bearable pain and mild limping) over 12 weeks or so. Oral weekly administration of 15 mg methotrexate, along with daily administration of 400 mg hydroxychloroquine and low-dose steroids (7.5–10 mg prednisolone), was continued. Moderate to severe polyarthritis continued, with elevated ESR, and hence, 2 g sulfasalazine and 100 mg minocycline were added. Few days later, the patient developed severe oral aphthous ulcers, requiring stoppage of methotrexate. The arthritis and skin lesions settled over almost 15 months of intense follow up. After a period of waxing and waning, the skin lesions of Hansen's disease also subsided with diffusely scattered reddish brown macules.

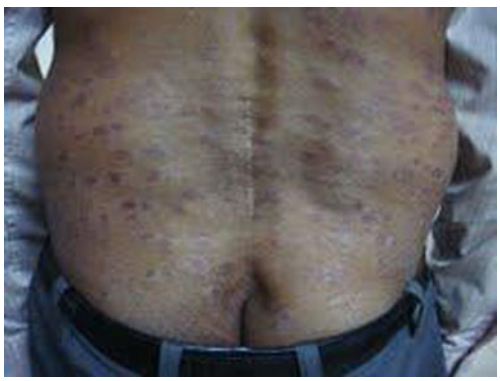

Fig. 3 Case 2 showing erythematous papulonodular itchy skin lesions on the abdomen and lower trunk

Case 3 A 52-year-old woman presented with painful rapidly progressive symmetrical polyarthritis of about 4 months in duration, without any extra-articular features, and was diagnosed with clinical RA. She was grossly limited in her daily activities and complained of prolonged morning stiffness. Other than symptomatic treatment, she also begun on oral daily administration of 10 mg leflunomide and 5 mg prednisolone. She was also on regular supervised medication for diabetes, hypertension, and epilepsy. On questioning, she recalled a history suggestive of suffering from leprosy (?polyneuritic) along with pulmonary tuberculosis several years ago and receiving prolonged therapy for about 18 months.

Two months later, she developed erythematous scaly macular rash on the back and lower limbs along with small ulcers on both soles. The arthritis was almost settled, and minimal tenderness elicited in the wrists and small joints of the hands. Her ankles and feet looked somewhat puffy. She complained of numbness and abnormal feeling in the feet and lower part of the legs. Neurological examination showed wasting of thigh muscles and hyperesthesia in the upper and lower limbs. She did not have any other systemic abnormality. Laboratory investigations showed a near normal hemogram with elevated CRP and seronegative for RF, ANA, and anti-CCP. Metabolic and hepatic parameters were normal, but renal functions were mildly deranged with elevated serum creatinine. Her serum uric acid was grossly elevated at 12.0 mg/dl (lab normal <7 mg/dl). X-ray of the hands showed juxta-articular osteoporosis and erosions typical of RA. Ultrasonography of the abdomen and pelvis showed mild hepatomegaly only. She was further investigated by a dermatologist who opined that the current complication was an activation of old leprosy in the form of probable lepra reaction, but a close differential of an adverse event due to leflunomide was also considered; skin clip was negative for AFB. The antiarthritis medication was changed to only symptomatic drugs, and anti-leprosy drugs were added and continued with low-dose orally given prednisolone. Six months later, she improved considerably with healed skin lesion, minimal numbness in the extremities, and absence of arthritis.

Discussion

Hansen's disease [8] continues to manifest protean clinical features that include rheumatic musculoskeletal disorders as demonstrated in this report. A 1–5 % of leprosy patients are

reported to develop arthritis of the small joints of the hands and feet akin to that seen in rheumatoid arthritis [9, 10]. Considering that Hansen's disease is not an uncommon disorder in India and only 33 patients of leprosy with arthritis were encountered over a 20-year period in a busy rheumatology clinic (current report) suggests a much lower prevalence of this complication. However, cases of leprosy are predominantly managed in dermatology outpatient of government-run general hospitals and primary health centers (rural) in our setting and are generally not referred to the rheumatologist. In this report, we also show that patients of chronic RA on long-term immunosuppression can develop leprosy, requiring a high index of clinical suspicion for timely diagnosis.

We encountered leprosy reactions in several of our patients (Table 1). In the current report, 15 patients were seen in reactional states and were divided between all the three types of lepra reaction causing diagnostic confusion. Sixty percent of the reactions in the study were type II, encountered in patients of lepromatous leprosy. At least four patients of tuberculoid leprosy with inflammatory arthritis suffered from downgrading reaction and moved towards the lepromatous profile. The expression of the reactions was largely edematous and seen on the face and lower limbs (puffy feet and ankles).

Patients with leprosy can present with two types of immune-mediated lepra reactions which are characteristic and need early detection and management [11] to avoid considerable morbidity (and rarely mortality). Type I reaction is a class IV hypersensitivity reaction with heightened cell-mediated immunity and seen in borderline forms (not polar) of leprosy. When occurring prior to therapy, the patient worsens (downgrading) and develops features of lepromatous leprosy: fever, edema, inflammatory skin lesions, and severe painful neuritis (manifesting as tender peripheral nerves, foot drop, claw hand, and facial palsy). Sometimes, type 1 reaction (reversal) is seen after the therapy is begun, and the clinical expression may progress toward the tuberculoid end. Type II is a class III hypersensitivity reaction occurring during the course of the treatment of patients with lepromatous forms (borderline/BL and polar lepromatous /LL). Also called erythema nodosum leprosum (ENL), the patient may develop severe immune complex-driven multisystemic features. Histologically, it appears to be a polymorph nuclear vasculitis or panniculitis. Although ENL is characterized by a TH2 cytokine profile with a high interleukin 6 and 8 assay [12], it may exhibit unusually high levels of tumor necrosis factor and IL-2. It is an immune complex deposition disorder. ENL is characterized by a sudden appearance of crops of evanescent erythematous tender nodules, which may become pustular and ulcerate. Patients are often febrile with features of neuritis, lymphadenitis, orchitis, and arthritis (often seen in knees and other large joints) and glomerulonephritis. Besides arthritis, ENL has been associated with necrotizing cutaneous vasculitis, septic arthritis, neuropathic arthritis, and swollen hand syndrome. Further, anemia may be caused due to red blood cell destruction or bone marrow suppression.

The multisystemic nature of leprosy, especially in a setting of a lepra reaction, may mimic septicemia. Finally, just like syphilis, Hansen's disease is a great mimic and needs an astute clinical sense with a high index of clinical suspicion to capture it early enough for an effective and complete cure [13]. The disease manifestation depends on the status of the host T cell immunity. The T cell immunity per se is protective. If the person contracts infection with a strong T cell immune response, the bacillary growth is restricted, but the resultant nerve damage may be profound, as seen in tuberculoid leprosy. In contrast, a widespread dissemination of mycobacterium takes place if the T cell immune response is low, resulting in the lepromatous spectrum of disease. Borderline disease subsets have intermediate T cell immune response.

Patients with leprosy can present with dominant rheumatic manifestations as shown in the current report. More uncommonly, this presentation may be an acute inflammatory polyarthritis mimicking RA: two males with borderline lepromatous leprosy presented with type I reaction in our rheumatology outpatient [13]. We did not find tenosynovitis in isolation though leprosy has been uncommonly diagnosed in a patient with tenosynovitis (in the absence of arthritis) with thickened nerves [14]. Leprosy can also mimic several forms of collagen vascular disease, including systemic lupus erythematous and scleroderma, due to its predominant skin affection, but the clinical profile of digital gangrene due to arteritis in case 1 in the current report is rather unusual.

Cossermelli-Messina et al. [15] described 39 cases of leprosy with chronic RA-like arthritis (mean duration 11 years); leprosy-related reactions were not reported. Leprosy was present for >10 years in most of their patients and was currently inactive in 19 of them. Although these patients had considerable relief with anti-leprosy therapy, the arthritis never resolved completely and was likely to have been complicated by neuropathic features. Surprisingly, though we did see some patients with chronic leprosy, none of the patients had neuropathic destructive arthritis, and this is likely to be due to early diagnosis and appropriate anti-leprosy treatment. In the current report, 22, 9, and 2 patients, respectively, were classified as LL, TT, and neuritic according to the Ridley–Jopling classification scale [16]; intermediate form was not seen. In several patients, polyarthritis was similar to RA, but we did not find erosive arthritis on radiological examination. The flexion contractures were generally subtle except in two patients with distinct claw hand who were mislabeled as having rheumatoid hand with Boutonniere's deformity; a closer look revealed typical neurologic abnormality. We did not find a foot drop which is often encountered in leprosy. Seventeen patients in our series had sensory system involvement, and three had feet

digital trophic ulcers; NCV study was carried out in five patients and showed sensory motor peripheral neuropathy of axonal type. Painless thickened ulnar nerve (near the elbow) and peroneal nerve (below the knee) were often found in our patients. Peripheral nerve involvement is usually more and appears earlier in TT than in LL [17]. Though RA can be complicated by peripheral neuropathy, and uncommonly, a claw hand seen as a manifestation of ulnar nerve affection, leprosy must be meticulously excluded in our setting. Rheumatoid vasculitis may involve virtually any organ of the body including the skin and nervous system [18].

Tuberculosis is an important complication of prolonged steroid and immunosuppressive therapy in our community [19], but leprosy should also be kept in mind as was highlighted by case no. 2 of this report. Patients with inflammatory rheumatic disorders often receive chronic immunosuppressive therapy and biologic agents (anti-TNF) [20], and this makes them prone to several opportunistic and community infections. In another rare parallel, leprosy was reported in patients with HIV infection on antiretroviral therapy, as a manifestation of immune reconstitution inflammatory syndrome [5, 21].

Several autoantibodies like RF and ANA which are commonly tested in rheumatological disorders may be false positive in leprosy [22]. In the current report, we report similar false seropositivity. Interestingly, none of the patients tested in the current series were seropositive for anti-CCP, which has a higher diagnostic value in RA [23].

The author (AC) completed population surveys of over 12,000 population in urban Pune and rural Bhigwan (Pune district) under the auspices of the WHO International League of Associations for Rheumatology Community-Oriented Program for Control of Rheumatic Diseases and reported a wide spectrum of rheumatic pain and disorders [24]. About 10 % of the community patients suffered from inflammatory arthritis, but none was found to suffer from leprosy or tuberculosis-associated arthritis. One woman patient of RA slowly developed claw hand and other features of tuberculoid leprosy during the 10-year follow-up in the Bhigwan community (unpublished).

Though uncommon, leprosy can present in rheumatology outpatient as was shown by the current study. Rheumatologists ought to be aware of this important differential diagnosis. It is also prudent to recommend a close interaction with a dermatologist in such situations where the excellent response to anti-lepromatous drugs can save patients from needless antirheumatic therapy.

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Disclosures None

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