Open Journal of
Clinical & Medical
Case Reports

Volume 4 (2018)
Issue 13

ISSN 2379-1039

Case report of leprosy diagnosis in the emergency department: A potential challenge in non-endemic areas

Mhd Samir Alsarakbi; Landric B Dsouza*; Stephen H Thomas

***Landric B Dsouza**

Department of Emergency Medicine, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar
Email: landricdsouza@gmail.com

Abstract

Background: Per the World Health Organization (WHO) definition of prevalence lower than 1 case per 10,000 persons, leprosy has been eliminated as a public health threat. However, it's not clear how many unreported cases exist in the less-developed world; leprosy is likely more common than currently reported. Current estimates are that 220,000+ new cases are diagnosed worldwide each year.

Case Presentation: We encountered a 29-year-old, Indian male who presented to the Emergency Department (ED) with non-tender skin eruptions, left for earm numbness and low-grade fever for two weeks. Physical examination revealed decrease in sensation of the left arm, left hand and dozens of non-tender nodular skin lesions of varying diameter. Patient was treated with a multi-drug regimen and underwent various blood, imaging tests and a skin biopsy.

Conclusion: The fact that leprosy may be more common in some parts of the world than indicated by available data stands in contradistinction to the rarity with which the disease is seen in highly developed countries. ED physicians in countries such as Qatar have never seen leprosy and with globalization of economies and labor forces, it is likely that leprosy may increase in incidence in developed-countries. Literature reveals that leprosy tends to be frequently misdiagnosed or mismanaged when presenting in countries with low incidence. The ED physician is thus well-advised to consider a patient's geographic origin and consider leprosy as a potential cause for skin lesions and findings such as described in this case – the main barrier to diagnosing leprosy in non-endemic areas is not considering it.

Keywords

emergency medicine; leprosy; qatar; emergency department

Abbreviations

WHO: World Health Organization; ED: Emergency department; HGH: Hamad general hospital; MDT: Multi-drug therapy; CDC: Centre for disease control; BCG: Bacillus calmette Guerin; USA: United States of America; PCR: Polymerase chain reaction.

Case Presentation

History

A 29-year-old male presented to the Emergency Department (ED) at Hamad General Hospital (HGH) in Doha, Qatar. He had a chief complaint of non-tenderskin eruptions all over the body, accompanied by left forearm numbness and low-grade fever. He had been ill for two weeks.

The patient had arrived in Qatar a month prior to presentation, to begin work in this country. He had no known exposures to illness or hazardous chemicals, and reported knowing no one with similar complaints. The patient was cooperative with the history, but his native language was an Indian dialect and there was a communication barrier preventing optimal history.

Review of systems was positive only for the previously mentioned entities. He reported no exacerbating or relieving factors for his symptoms. He had no ophthalmologic complaints and had no itching or mucous membrane symptoms. The left arm numbness and loss of sensation was diffuse in the involved upper extremity and not associated with weakness.

The patient had first noted the arm numbness when he accidentally (in a welding incident) sustained minor burns to the forearm that he did not feel, but which he thought should have been painful. The remaining review of systems was negative.

Social history was notable for limited in-country support for the patient, who entered Qatar without family in order to join the local construction workforce. When he perceived he was ill he tried to travel home to India, but was denied boarding at the airport due to elevated temperature. At that point he was sent to the HGH ED for further evaluation.

There was no pertinent previous history, no immune compromise, and no prior surgery. The patient was not taking any chronic medications and had no known allergies.

Physical examination

The physical examination revealed anon-distressed male with vital signs: Heart rate 84 bpm, respirations 20, room-air pulse oximetry 99%, blood pressure 121/77, and oral temperature 38.5 C. The eye, ear, nose, and throat assessment revealed baseline visual acuity and normal mucous membranes; there was no sign of intravascular volume depletion. The neck was supple without adenopathy. Auscultation of the chest revealed normal lung and cardiac sounds. The abdomen was soft and non tender without organomegaly or peritoneal irritation. The patient was appropriate and cooperative with a neurological examination that revealed only subjective decrease in sensation in the left arm and left hand (in the ulnar distribution).

The skin examination revealed dozens of non-tender nodular lesions of varying diameter 1-4 cm. The primary anatomical location of lesions was on the face and trunk, with some extremity lesions as well. The appearance of the lesions is shown in Figures 1-4.

Initial impression and work-up

Based on the initial presentation and physical examination, the differential diagnosis included a breadth of infectious and non-infectious entities. Dermatologic problems (*e.g.* erythema nodosum) and

other non-infectious conditions (*e.g.* neurofibromatosis) were included in the early listing of alternative diagnoses, but the evaluating physician had previously seen cases of leprosy (Hansen's disease) and felt this presentation was highly suggestive of lepromatous disease.

The ED physician instituted droplet isolation measures (moving the patient to the ED's isolation unit) and began therapy with antipyretics (paracetamol 1 g IV), intravenous fluids (Ringer's lactate 1.5 L over 3 hours) to supplement oral intake, and broad-spectrum antibiotics for coverage of alternative possible diagnoses(2 g ceftriaxone IV). The Qatari Ministry of Public Health was later notified of the patient's suspected diagnosis.

The initial work-up proceeded with general metabolic, sepsis, and hematologic testing. Selected pertinent results are shown in Table 1. The ED lab results were notable for leukocytosis and signs of systemic inflammation with elevated lactate. An initial chest X-ray (Figure 4) was unremarkable.

Based on the initial results, further suspicion of leprosy prompted consultation with the HGH Department of Medicine's Division of Infectious Diseases (ID). The ID consultant saw the patient in the ED isolation unit and agreed with the presumptive diagnosis of erythema nodosum leprosum (lepromatous leprosy), ordering a punch biopsy for histological evaluation.

Hospital course

The patient was admitted to the HGH for performance of the punch biopsy and to institute presumptive therapy for multibacillary leprosy. The leprosy therapy was the WHO multi-drug therapy (MDT) approach (see Discussion section). Treatment commenced on day one with combination of three antimicrobials: rifampicin (600 mg), clofazimine (300 mg), and dapsone (100 mg, administered after negative test results for glucose-6-phosphate dehydrogenase deficiency). After the preceding therapy was given on day one, daily therapy continued with clofazimine (50 mg) and dapsone (100 mg) with the plan to repeat the cycle (day one therapy followed by daily therapy) monthly for a year. The patient's treatment regimen also included prednisolone 60 mg daily.

The patient defervesced by hospital day two. He was never tachycardic, hypotensive, or hypoxemic during the hospitalization.

Two days post-admission, the patient underwent punch biopsy of a left-arm lesion. He was continued on antimicrobials and was afebrile with stable vital signs and minimal complaints. The day after the punch biopsy, the patient had been afebrile for two days and he was discharged from the hospital on MDT with biopsy results pending and ID clinic follow-up arranged.

Trends in selected laboratory tests are shown in Table 2. Anti-nuclear antibody assay was negative, as were serologic tests for hepatitis and human immunodeficiency virus. Lactate levels normalized by hospital day 1 to 1.6 mmol/L and were not rechecked afterward. Aerobic and anaerobic blood cultures were reported as no growth at five days post-admission.

The day after his discharge, the patient left the country and has been lost to follow-up. His biopsy results returned the next day. The punch biopsy results showed dermal epithelioid granulomatous inflammation with periadnexal, perivascular, and perineural distribution. Focal necrosis was noted as well. The special stain for *M. leprae* was negative. However, the hospital laboratory's histological diagnosis was “granulomatous inflammation fully in keeping with tuberculoid leprosy.”

The final clinical diagnosis was leprosy, caused by the bacillus *M. leprae*. Attempts to contact the patient by telephone failed and there is no information on his current status.

Discussion

Leprosy etiology, disease classification, and epidemiology

Leprosy is caused by the intracellular acid-fast bacterium *M. leprae* (and less frequently, by the newly described species *M. lepromatosis*) [1,2]. The disease is classically divided into paucibacillary tuberculoid leprosyor multibacillary lepromatous leprosy, but there are wide variations (*e.g.* leprosy limited to nerve involvement) and overlaps in classification systems [3,4]. While the type of leprosy in terms of organ involvement and bacillary load is important from ID and public health perspectives, for the initial ED work-up and therapy the precise categorization of leprosy subtype is not critical.

Leprosy is a tropical disease. Among the areas with historical or current problems with leprosy are India (and neighboring Nepal), Brazil, Indonesia and nearby areas (*e.g.* Singapore, Malaysia, the Philippines), the Americas (tropical regions in particular) and central Africa [5-12].

In terms of the WHO definition of prevalence lower than 1 case per 10,000 persons, leprosy has been officially eliminated as a public health threat. However, it is not clear how many unreported cases exist in the less-developed world; leprosy is likely far more common than currently reported and current estimates are that 220,000+ new cases are diagnosed worldwide each year [8,13]. Experts have noted that the disease remains endemic (if not rising) in many areas of the world, and due to its causing significant disability leprosy remains a threat particularly in vulnerable populations such as children[14-16]. Information on the WHO program on leprosy is available at

www.who.int/mediacentre/facsheets/fs101/en/ and information on leprosy transmission is outlined at the USA's Centers for Disease Control (CDC) website www.cdc.gov/leprosy/transmission.

The contribution of the immune system susceptibility to leprosy is not fully characterized, but there are data suggesting genetic factors' contributions [14,17]. It also seems likely that immunocompromise such as that seen in transplant recipients, could increase risk of leprosy [18,19].

Clinical manifestations and natural history of disease

Leprosy is most notorious for its skin lesions. However, the disease commonly affects peripheral nerves, upper respiratory mucosa, and the eyes (including causing blindness) [5,20-24].

Facial lesions can have devastating cosmetic effect. Ulcerative changes and even loss of body parts (*e.g.* fingers, nose) due to inflammation, immunecompromise, and secondary infection can occur [24]. The pattern of erythematous nodular lesions seen in our case is not inconsistent with the face, trunk, and extremity distribution of such lesions often reported in lepromatous leprosy [18].

The indolent nature of leprosy infection is such that ongoing nerve involvement causes significant neuropathy over years of disease. Risk of nerve damage is increased due to diagnostic delays [5].

The chronic course of leprosy may be marked by intermittent skin-lesion flares called acute leprosy (or lepra) reactions [25,26]. These lepra reactions are clinically overt and may be the primary reason for patient presentation with previously undiagnosed disease.

M. leprae multiplies very slowly. The disease is characterized by an incubation period averaging 5 years. In some cases, symptoms may occur within a year of contracting the disease but patients may not develop the characteristic skin findings until 20 years after exposure. In one representative pediatric series from Brazil, nearly 3/4^{ths} of cases had no symptoms until at least a year after exposure [5].

Prevention: Historical and current

Although most leprosy cases arise from human transmission, leprosy is not highly contagious and there has long been disagreement as to precise transmission risks and mechanisms. [24, 27-29]. Nasal or mouth droplet transmission seems the most frequent mechanism, but the causative mycobacteria have also been cultured from skin surfaces [30,31]. the major risks to those with long-term exposure (*e.g.* family members caring for patients) [32].

If there are known exposures, chemoprophylaxis (*e.g.* with MDT drugs) can be used since there is no highly effective vaccine[13,33]. A single dose of the Bacillus Calmette-Guerin (BCG) immunization is reported to reduce risk of leprosy contraction by half [13.]

The disconcerting appearance of lepers has combined fears of transmission to translate into a historical "preventive" approach of isolating those with the disease. Over centuries, lepers have been stigmatized, discriminated against, and occasionally banished [17,34,35]. In many countries leper colonies were established in remote or non-populated regions [36]. In the United States of America (USA), for instance, the most notable colony was established in the Hawaiian Islands over a century ago, and operated until the middle of the 20th century [37,38]. A second leper colony was established by the U.S. Public Health Service in the semi-tropical state of Louisiana in the USA's South; this establishment for 75 years after its doors opened in 1922 [39,40].

Diagnosis

Once the disease is considered, punch biopsy and histologic examination should be executed as histopathological examination of the skin lesion is the gold-standard for diagnosis, especially when presentations may be atypical [25,41]. On histopathology, leprosy should be considered any time there are perineural lymphocytes with a pattern of granulomatous infiltrate; the next step is to obtain a Fite acid-fast stain (as was done in our case) although there is evidence that fluorescent histochemical methods may improve diagnostic accuracy [40,41] The *M. leprae* organism is less acid-fast than the bacillus of *M. tuberculosis* due to differences in mycolic acid carbon-chain lengths, but the stain is usually helpful.

Although there are a variety of reported serological and molecular-probe methods for diagnosing leprosy, the precise identification of the organism can be rendered difficult by the inability to grow it *in vitro* [32]. A recent report of a nasal polymerase chain reaction (PCR) swab for detection of *M. leprae* shows promise for making the diagnosis in symptomatic and asymptomatic cases [32]. An advanced form of quantitative PCR (qPCR) has been found to have nearly 85% sensitivity and 100% specificity in diagnosing leprosy, regardless of the form or stage of clinical disease [43].

Treatment

The two main issues with treating leprosy are delays in diagnosis and imperfect therapeutic success even after diagnosis is made. Treatment delays are especially problematic due to the indolent

disease course and these delays are associated with increased tissue damage [5].

The disease is at least partially curable by the MDT antimicrobial approach used in our case. Unfortunately, recurrence rates can approach 50%, particularly in immunocompromised patients, even after treatment has been deemed a success [19]. The antimicrobials used for leprosy are dosed differently in the long-term treatment, depending on disease characteristics, but the initial regimen is as used in our case. The MDT approach used in our patient (clofazamine, rifampin, dapsone) is that which was promulgated by the WHO (when it passed a resolution in 1991 to eliminate leprosy as a public health problem)[8,19].

Clofazamine can cause harmless skin discoloration in newborns (when given to pregnant patients)[44]. The drug also rarely causes enteropathy when administered in high doses [45].

The anti-leprosy drugs rifampin and clofazamine also have activity against other acid-fast bacilli, notably *M. tuberculosis* which may be theoretically useful given historical reports of co-infection with the two mycobacteria [26,46,47]. The third drug in MDT, dapsone, has both bactericidal and bacteriostatic activity against *M. leprae* and is a long-standing foundation of anti-leprosy therapy even though problems such as hypersensitivity and hemolysis must be considered [48,49].

Quinolones may have some role in treatment of leprosy, although a DNA gyrase mutation in *M. leprae* is well-known to mediate resistance to this class of antimicrobials [50].

Pregnancy is a risk factor for exacerbation of leprosy. The standard MDT regimen is reported safe and effective for pregnant patients and their newborns [44].

Therapy additional to antimicrobials includes steroids. Steroids appear useful for acute lepra reactions and are very commonly used in leprosy [26,26]. Steroids' therapeutic goals are to control symptoms and to reduce recurrences and long-term deformity [12,26,51]. Steroids are particularly useful, although not invariably effective, in treating erythema nodosum leprosum (as seen in our patient) [52]. Cochrane Review identifies some potential benefit from steroid use in treating nerve damage in leprosy [53].

Immunotherapy with a *Mycobacterium w* (Mw) vaccine (a heat-killed suspension derived from a nonpathogenic, cultivable, atypical mycobacterium named *M.indicuspranii*) may provide additional benefit to MDT in terms of preventing disease worsening or recurrence [54,55].

In most countries, leprosy is a reportable disease. ED physicians or others who suspect or confirm the diagnosis, should correspond with their area public health authorities.

Conclusion

The fact that leprosy may be more common in some parts of the world than indicated by available data stands in contradistinction to the rarity with which the disease is seen in highly developed countries. Many ED physicians in countries such as ours (Qatar) have never seen leprosy, and with globalization of economies and labor forces it is likely that leprosy – like other "third-world diseases" – could be seen with increasing frequency in developed-country Eds.

Unfortunately, the literature from developed-country clinics reveals that leprosy (like other non-endemic diseases) tends to be frequently misdiagnosed or otherwise mismanaged when presenting to

healthcare in countries in which the disease is not commonly seen [20,42,56,57]. The ED physician is thus well-advised to consider a patient's geographic origin, and to think of leprosy as a potential cause for skin lesions and findings such as those described in this case – the main barrier to diagnosing leprosy in non-endemic areas is not considering it [57].

Competing Interests/ Funding/ Declarations/ Acknowledgements: None.

Ethics Approval and Patient Consent: Ethics approval was obtained from the Institutional Review Board (IRB) for the purpose of the case report.

Consent for Publication: The nature of the case report was explained to the patient and informed consent was appropriately taken for photographs of the patient's clinical findings.

Figures

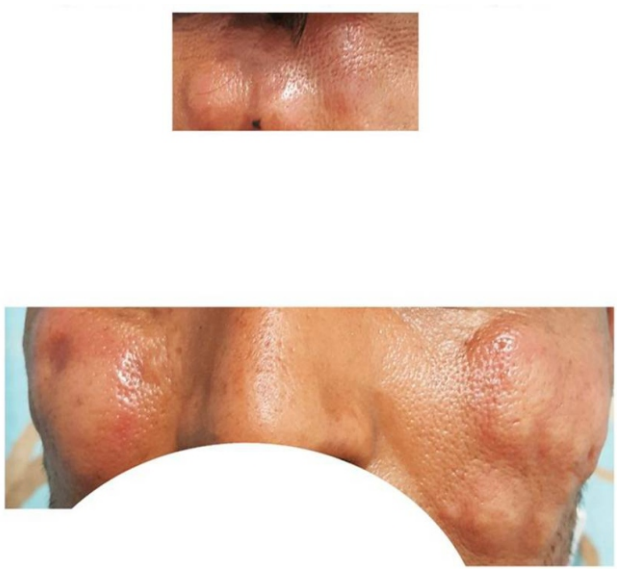

Figure 1: Pale nodular lesions on the forehead (each lession 2 cm diameter) and cheeks

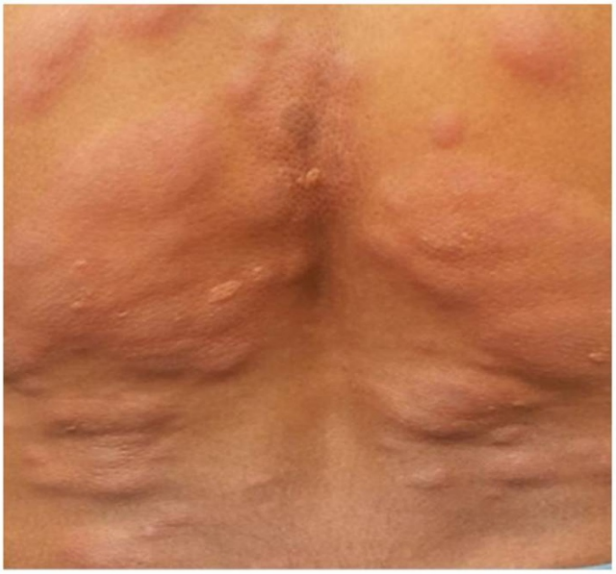

Figure 2: lessions on the patient"s backshowing confluence and plaque-like appearance

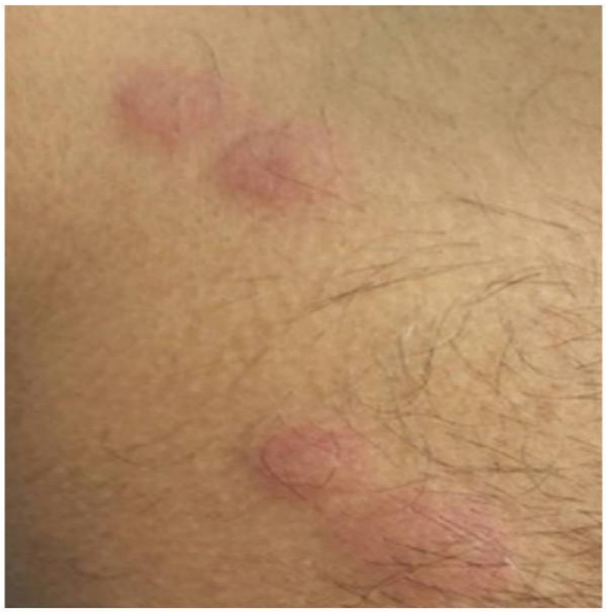

Figure 3: Pectoral lesions (each lesion's diameter 1.5-2 cm) showing erythematous nodule appearance

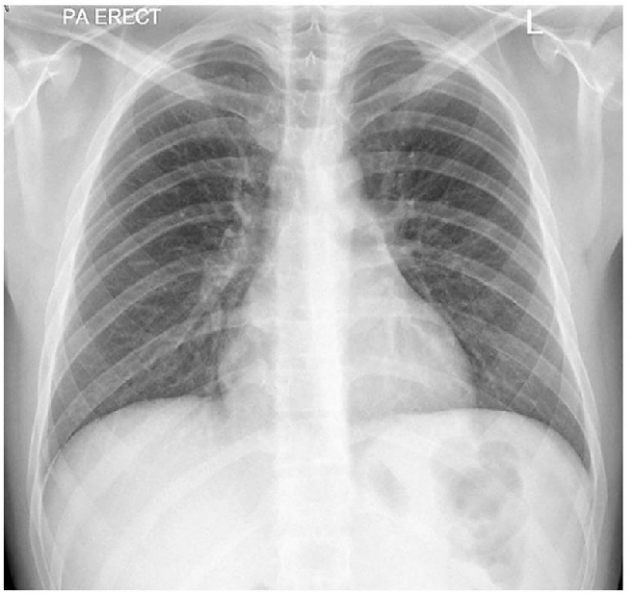

Figure 4: Chest X-ray

References

1. Velarde-Felix JS, Alvarado-Villa G, Vera-Cabrera L. "Lucio's Phenomenon" Associated with Mycobacterium lepromatosis. The American journal of tropical medicine and hygiene. 2016; 94: 483-4.

2. Han XY. Detection of the Leprosy Agent Mycobacterium lepromatosis in South America and Europe. The American journal of tropical medicine and hygiene. 2017; 96: 260.

3. Rao PN, Suneetha S. Pure neuritic leprosy: Current status and relevance. Indian J Dermatol Venereol Leprol. 2016; 82: 252-61.

4. Rodrigues Junior IA, Gresta LT, Noviello Mde L, Cartelle CT, Lyon S, Arantes RM. Leprosy classification methods: a comparative study in a referral center in Brazil. International journal of infectious diseases : IJID : official publication of the International Society for Infectious Diseases. 2016; 45: 118-22.

5. Bandeira SS, Pires CA, Quaresma JA. Nerve Damage in Young Patients with Leprosy Diagnosed in an Endemic Area of the Brazilian Amazon: A Cross-Sectional Study. J Pediatr 2017.

6. Banger HS, Sethi A, Malhotra S, Malhotra SK, Kaur T. Clinico-epidemiological profile of patients attending Suraksha Clinic of tertiary care hospital of North India. Indian J Sex Transm Dis. 2017; 38: 54-9.

7. Thangaraju P, Venkatesan S, Showkath Ali MK. Leprosy case detection campaign (LCDC) for active surveillance. Trop Doct. 2017: 49475517702059.

8. Valentin DC, Candelario N, Carrasquillo OY, Figueroa L, Sanchez JL. Leprosy in Puerto Rico: insight into the new millennia. Int J Dermatol. 2017; 56: 440-3.

9. Kua EH, Kua JP. The social transformation of Singapore medicine through 55 years of the SMJ. Singapore Med J. 2016; 57: 587-90.

10. Jamil A, Muthupalaniappen L, Md Nor N, Siraj HH, Salam A. Identifying the Core Content of a Dermatology Module for Malaysian Medical Undergraduate Curriculum Using a Modified Delphi Method. Malays J Med Sci 2016; 23: 78-85.

11. Joloba M, Mwangi C, Alexander H, et al. Strengthening the Tuberculosis Specimen Referral Network in Uganda: The Role of Public-Private Partnerships. The Journal of infectious diseases. 2016; 213.

12. Lambert SM, Alembo DT, Nigusse SD, Yamuah LK, Walker SL, Lockwood DN. A Randomized Controlled Double Blind Trial of Ciclosporin versus Prednisolone in the Management of Leprosy Patients with New Type 1 Reaction, in Ethiopia. PLoS neglected tropical diseases. 2016; 10: e0004502.

13. Duthie MS, Balagon MF. Combination chemoprophylaxis and immunoprophylaxis in reducing the incidence of leprosy. Risk Manag Healthc Policy. 2016; 9: 43-53.

14. Aguilar-Medina M, Escamilla-Tilch M, Frias-Castro LO, et al. HLA Alleles are Genetic Markers for Susceptibility and Resistance towards Leprosy in a Mexican Mestizo Population. Annals of human genetics 2017; 81: 35-40.

15. Barreto JG, Frade MAC, Bernardes Filho F, da Silva MB, Spencer JS, Salgado CG. Leprosy in Children. Curr Infect Dis Rep. 2017; 19: 23.

16. Kumar A, Girdhar A, Chakma JK. Increase in cases of leprosy in the Firozabad district of India: a two-time cross-sectional survey. The British journal of dermatology 2017; 176: 520-2.

17. Andrade FA, Beltrame MH, Bini VB, Goncalves LB, Boldt AB, Messias-Reason IJ. Association of a new FCN3 haplotype with high ficolin-3 levels in leprosy. PLoS neglected tropical diseases. 2017; 11: e0005409.

18. Aytekin S, Yasar S, Goktay F, et al. Lepromatous leprosy in a renal transplant recipient. American journal of transplantation : official journal of the American Society of Transplantation and the American Society of Transplant Surgeons 2017.

19. Vieira AP, Trindade MAB, de Paula FJ, et al. Severe type 1 upgrading leprosy reaction in a renal transplant recipient: a paradoxical manifestation associated with deficiency of antigen-specific regulatory T-cells? BMC Infect Dis. 2017; 17: 305.

20. Villada G, Zarei M, Romagosa R, Forgione P, Fabbrocini G, Romanelli P. Autochthonous borderline tuberculoid leprosy in a man from Florida. Lepr Rev. 2016; 87: 101-3.

21. Del'Arco R, De Oliveira AB, Nardi SM, Paschoal VD. The association between neuropathic pain and disability grades in leprosy. Lepr Rev. 2016; 87: 53-9.

22. Ajalla ME, Oliveira De Andrade SM, Tamaki EM, Waissmann W, Deittrich SH, Aragao Do Nascimento V. Leprosy in Brazilian counties bordering Paraguay: Mato Grosso do Sul State, 2001-2011. Lepr Rev. 2016; 87: 32-41.

23. Santos SD, Penna GO, Costa Mda C, Natividade MS, Teixeira MG. Leprosy in children and adolescents under 15 years old in an urban centre in Brazil. Mem Inst Oswaldo Cruz. 2016; 111: 359-64.

24. Franco-Paredes C, Rodriguez-Morales AJ. Unsolved matters in leprosy: a descriptive review and call for further research. Annals of clinical microbiology and antimicrobials. 2016; 15: 33.

25. Chiaratti FC, Daxbacher EL, Neumann AB, Jeunon T. Type 2 leprosy reaction with Sweet's syndrome-like presentation. An Bras Dermatol. 2016; 91: 345-9.

26. Ganesan V, Mandal J. Primary oral tuberculosis in a patient with lepromatous leprosy: Diagnostic dilemma. Int J Mycobacteriol. 2016; 5: 102-5.

27. Frade MA, Foss NT. Editorial Commentary: Evidences of Aerial Route of Mycobacterium leprae Infection and Doubts About Transmission and Natural Protection in Leprosy. Clin Infect Dis. 2016; 63: 1421-2.

28. Turankar RP, Lavania M, Singh M, Sengupta U, Siva Sai K, Jadhav RS. Presence of viable Mycobacterium leprae in environmental specimens around houses of leprosy patients. Indian J Med Microbiol. 2016; 34: 315-21.

29. Benchimol JL, Romero Sa M. Adolpho Lutz and controversies over the transmission of leprosy by mosquitoes. Hist Cienc Saude Manguinhos. 2003; 10: 49-93.

30. Araujo S, Freitas LO, Goulart LR, Goulart IM. Molecular Evidence for the Aerial Route of Infection of Mycobacterium leprae and the Role of Asymptomatic Carriers in the Persistence of Leprosy. Clin Infect Dis. 2016; 63: 1412-20.

31. Avanzi C, Busso P, Benjak A, et al. Transmission of Drug-Resistant Leprosy in Guinea-Conakry Detected Using Molecular Epidemiological Approaches. Clin Infect Dis. 2016; 63: 1482-4.

32. Arunagiri K, Sangeetha G, Sugashini PK, Balaraman S, Showkath Ali MK. Nasal PCR assay for the detection of Mycobacterium leprae pra gene to study subclinical infection in a community. Microbial pathogenesis. 2017; 104: 336-9.

33. Malheiro L, Pinto SC, Sarmento A, Santos L. Comparison and Contrast of the Elimination Campaigns for Poliomyelitis and Leprosy: Which is More Feasible? Acta Med Port. 2016; 29: 279-83.

34. Zamparoni V. Leprosy: disease, isolation, and segregation in colonial Mozambique. Hist Cienc Saude Manguinhos. 2017; 24: 13-39.

35. John HV. Translating leprosy: the expert and the public in Stanley Stein's anti-stigmatization campaigns, 1931-60. J Hist Med Allied Sci. 2013; 68: 659-87.

36. White C. Carville and Curupaiti: experiences of confinement and community. Hist Cienc Saude Manguinhos. 2003; 10: 123-41.

37. Michael JM. The Public Health Service leprosy investigation station on Molokai, Hawaii, 1909-13--an opportunity lost. Public health reports. 1980; 95: 203-9.

38. Fennel EA. Kalauppa, Molokai, then and now. Star. 1948; 7: 4.

39. Moschella SL. Carville: 100 years of excellence. J Am Acad Dermatol. 1997; 36: 126-30.

40. Sato H, Frantz JE. [History of Hansen's disease control policy in the United States]. Nihon Hansenbyo Gakkai Zasshi. 2005; 74: 23-41.

41. Adiga DS, Hippargi SB, Rao G, Saha D, Yelikar BR, Karigoudar M. Evaluation of Fluorescent Staining for Diagnosis of Leprosy and its Impact on Grading of the Disease: Comparison with Conventional Staining. J Clin Diagn Res. 2016; 10: EC23-EC6.

42. Zhu TH, Kamangar F, Silverstein M, Fung MA. Borderline Tuberculoid Leprosy Masquerading as Granuloma Annulare: A Clinical and Histological Pitfall. Am J Dermatopathol. 2017; 39: 296-9.

43. Azevedo MC, Ramuno NM, Fachin LR, et al. qPCR detection of Mycobacterium leprae in biopsies and slit skin smear of different leprosy clinical forms. Braz J Infect Dis. 2017; 21: 71-8.

44. Ozturk Z, Tatliparmak A. Leprosy treatment during pregnancy and breastfeeding: A case report and brief review of literature. Dermatologic therapy. 2017; 30.

45. Szeto W, Garcia-Buitrago MT, Abbo L, Rosenblatt JD, Moshiree B, Morris MI. Clofazimine Enteropathy: A Rare and Underrecognized Complication of Mycobacterial Therapy. Open Forum Infect Dis. 2016; 3: ofw004.

46. Allen HB, Moschella SL. The Role of Rifampin in Leprosy : Leprosy Through a New Lens. JAMA dermatology. 2017; 153: 261-2.

47. Ammerman NC, Swanson RV, Tapley A, et al. Clofazimine has delayed antimicrobial activity against Mycobacterium tuberculosis both in vitro and in vivo. The Journal of antimicrobial chemotherapy 2017; 72: 455-61.

48. Wang N, Parimi L, Liu H, Zhang F. A Review on Dapsone Hypersensitivity Syndrome Among Chinese Patients with an Emphasis on Preventing Adverse Drug Reactions with Genetic Testing. The American journal of tropical medicine and hygiene 2017.

49. Cha YS, Kim H, Kim J, et al. Incidence and patterns of hemolytic anemia in acute dapsone overdose. Am J Emerg Med. 2016; 34: 366-9.

50. Yamaguchi T, Yokoyama K, Nakajima C, Suzuki Y. Quinolone resistance-associated amino acid substitutions affect enzymatic activity of Mycobacterium leprae DNA gyrase. Biosci Biotechnol Biochem. 2017: 1-5.

51. Hossain D. Management of chronic neuritis with a combination regimen of lower doses prednisolone and methotrexate: a brief report. Lepr Rev. 2016; 87: 118-21.

52. Lambert SM, Nigusse SD, Alembo DT, et al. Comparison of Efficacy and Safety of Ciclosporin to Prednisolone in the Treatment of Erythema Nodosum Leprosum: Two Randomised, Double Blind, Controlled Pilot Studies in Ethiopia. PLoS neglected tropical diseases. 2016; 10: e0004149.

53. Van Veen NH, Nicholls PG, Smith WC, Richardus JH. Corticosteroids for treating nerve damage in leprosy. Cochrane Database Syst Rev. 2016: Cd005491.

54. Khullar G, Narang T, Nahar Saikia U, Dogra S. Generalized granulomatous dermatitis following Mycobacterium w (Mw) immunotherapy in lepromatous leprosy. Dermatologic therapy. 2017; 30.

55. Kamal R, Pathak V, Kumari A, Natrajan M, Katoch K, Kar HK. Addition of Mycobacterium indicus pranii vaccine as an immunotherapeutic to standard chemotherapy in borderline leprosy: a double-blind study to assess clinical improvement (preliminary report). The British journal of dermatology 2016.

56. Zammarchi L, Vellere I, Stella L, Bartalesi F, Strohmeyer M, Bartoloni A. Spectrum and burden of neglected

tropical diseases observed in an infectious and tropical diseases unit in Florence, Italy (2000-2015). Internal and emergency medicine 2017.

57. Keystone JS. Imported leprosy: If you think about it, you can diagnose it. Travel Med Infect Dis. 2016; 14: 297-8.

Manuscript Information: Received: March 29, 2018; Accepted: July 06, 2018; Published: July 16, 2018

Authors Information: Mhd Samir Alsarakbi[1]; Landric B. Dsouza[1*]; Stephen H Thomas[1,2]

[1]Department of Emergency Medicine, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar
[2]Weill-Cornell Medical College in Qatar, Doha, Qatar

Citation: Alsarakbi S, Dsouza LB, Thomas SH. Case report of leprosy diagnosis in the emergency department: A potential challenge in non-endemic areas.. Open J Clin Med Case Rep. 2018; 1434.

Journal: Open Journal of Clinical and Medical Case Reports is an international, open access, peer reviewed Journal focusing exclusively on case reports covering all areas of clinical & medical sciences.

Visit the journal website at **www.jclinmedcasereports.com**
For reprints and other information, contact editorial office at **info@jclinmedcasereports.com**