

# Cutaneous Leishmaniasis in a Middle Aged Man

MD. ROBED AMIN,<sup>1</sup> SHAFIUL ISLAM,<sup>2</sup> MD HABIBUR RAHMAN,<sup>3</sup> FARUKH AHMED,<sup>4</sup> AHMED RIYADH HOSSAIN,<sup>5</sup> MD AZIZUL KAHHAR<sup>6</sup>

## Abstract:

The incidence of cutaneous leishmaniasis (CL) is greater in the Old World than in the New World. It can be caused by several *Leishmania* spp and is transmitted to human beings and animals by sandflies and is usually more common in rural than urban areas, but it is found in some periurban and urban areas too. In Bangladesh there has been many cases of Post kala azar leishmaniasis (PKDL) in endemic areas but pure form of cutaneous leishmaniasis has not been reported many. Recently a case of cutaneous leishmaniasis was diagnosed in Dhaka Medical College Hospital (DMCH) who presented with increasing number of papulonodular crusted lesions in acral parts of body. The patients although resided in Saudi Arabia, there was no history regarding risk factor to get the disease from there or in Bangladesh indicating search for exploration of disease in Bangladesh is crucial. The patient was diagnosed methodically and treated with inj Sodium Stibogluconate with complete recovery.

**Keyword:** cutaneous, Leishmaniasis

## Introduction:

Leishmaniasis is endemic in 88 countries throughout Africa, Asia, Europe, and North and South America. Cutaneous leishmaniasis is a particupar type of leishmanis which has predilection of lesion in integumentary system of the body. There are an estimated 12 million cases worldwide, with 1.5 to 2 million new cases each year.<sup>1</sup> The incidence of cutaneous leishmaniasis (CL) is greater in the Old World than in the New World. The *Leishmania* protozoan was first described in 1903 by Leishman and Donovan, working separately. Since then, this organism has been found to be a complex grouping of species, at least 20 of which cause infections in humans.<sup>2</sup> Cutaneous leishmaniasis can be caused by several *Leishmania* spp and is transmitted to human beings and animals by sandflies. Because it is rarely fatal, cutaneous leishmaniasis has become one of the so-called neglected diseases.<sup>3</sup> Old World cutaneous disease primarily is caused by *Leishmania tropica* in urban areas and *Leishmania major* in dry desert areas. In the Old World (Eastern Hemisphere), CL is found in parts of the Middle

East, Asia (particularly southwest and central Asia but not very much in southeast asia). CL is usually more common in rural than urban areas, but it is found in some periurban and urban areas (such as in Baghdad, Iraq, and Kabul, Afghanistan).<sup>3</sup> Travelers who might have an increased risk for CL include ecotourists, adventure travelers, bird watchers, Peace Corps volunteers, missionaries, soldiers, construction workers, and people who do research outdoors at night or twilight. However, even short-term travelers in endemic areas have developed CL.<sup>4</sup> In Bangladesh there has been many cases of Post kala azar leishmaniasis (PKDL) in endemic areas (actual data is lacking) but these are detected late and these are not a pure form of cutaneous leishmaniasis. Primary cutaneous leishmaniasis is rarely reported in Bangladesh and recently one report has been observed<sup>5</sup> where diffuse cutaneous leishmaniasis was reported in a HIV positive case but the lesions were also consistent like PKDL.

Here is a case report of a cutaneous leishmaniasis who was diagnosed in Medicine unit of Dhaka Medical College Hospital (DMCH) who had a long working experience in Saudi Arabia but not actually having a risk factor to develop the disease.

## Case Report:

A 35 yrs old electrical waldeiier, presented in Medicine department of Dhaka Medical College Hospital on 24.11.2010 with the complaints of multiple nodular lesion in the body. The lesions were initially erythematous and crusted on both elbows and dorsum of base of left thumb. The lesion started 3 months back, nonitchy, not painful and have no discharge. The lesion while doesn't have healed spontaneously, new

1. Assistant Professor of Medicine, Dhaka Medical College
2. Medical Officer, Department of Medicine, Dhaka Medical College
3. Associate Professor of Medicine, Faridpur Medical College
4. Registrar, Department of Medicine, Dhaka Medical College
5. Assistant Registrar, Department of Medicine, Dhaka Medical College
6. Professor of Medicine, Dhaka Medical College

**Correspondence:** Dr .Md. Robed Amin, Assistant Professor of Medicine, Department of Medicine, Dhaka Medical College, Bangladesh. Apt-C-2, House no-76, Road-5, Block-F, Banani, Dhaka, Bangladesh. e.mail: robedamin@yahoo.com

lesion developed in left sided cubital area and the course remain similar to previous lesions. The skin lesions gradually becomes clearly demarcated with defined border.



**Fig.-1:** The Papulo nodular lesions in left elbow

There was a history of low grade fever for 5 months which were never documented in his past history. The patient didn't have any joint pain or swelling in and around the elbow. There was no history of jaundice, sore throat, cough, chest pain, respiratory distress, abdominal pain, vomiting or diarrhoea. There was no urinary complaints, genital ulcer or discharging pus through urethra and no history of extramarital sexual exposure. Professionally he was an electrical worker for 10 yrs and denied any homosexuality in life. He was non-alcoholic, non-smoker and there was no history of contact with animal bite prior to the skin lesion. There was no such lesions in the family. With all these skin lesions he consulted local doctors in Saudi Arabia. There was no diagnosis of his skin lesions by the consultations. Then in Bangladesh, he was seen by a medicine specialist and advised for admission in medicine department of DMCH for evaluation.

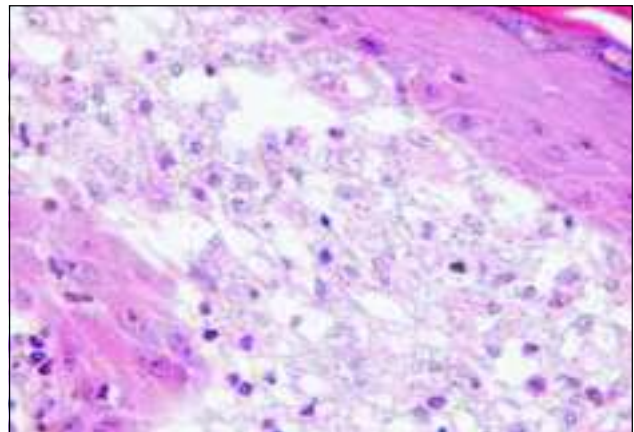
On examination of this young well built gentleman revealed stable clinical status. His vitals were pulse-88/min, BP-100/80 mm Hg, temp-98.2 degree, and respiratory rate 20/min. He was not anemic and non icteric. Systemic examination was unremarkable. On local examination of integumentary system revealed papulo nodular lesion with erythematous base and crusted edge on both elbow and dorsum of thumb left thumb and left cubital area. The lesions were non itchy, nontender, non discharging and no cicatrization or scaliness present. Nikolski sign was negative. The triple response of hypersensitivity was also negative. There was no

surrounding lymphadenopathy or organomegaly. The thumb nail and all other fingers and toes were normal and there was no pitting in any of nails. The musculoskeletal system examination was normal.

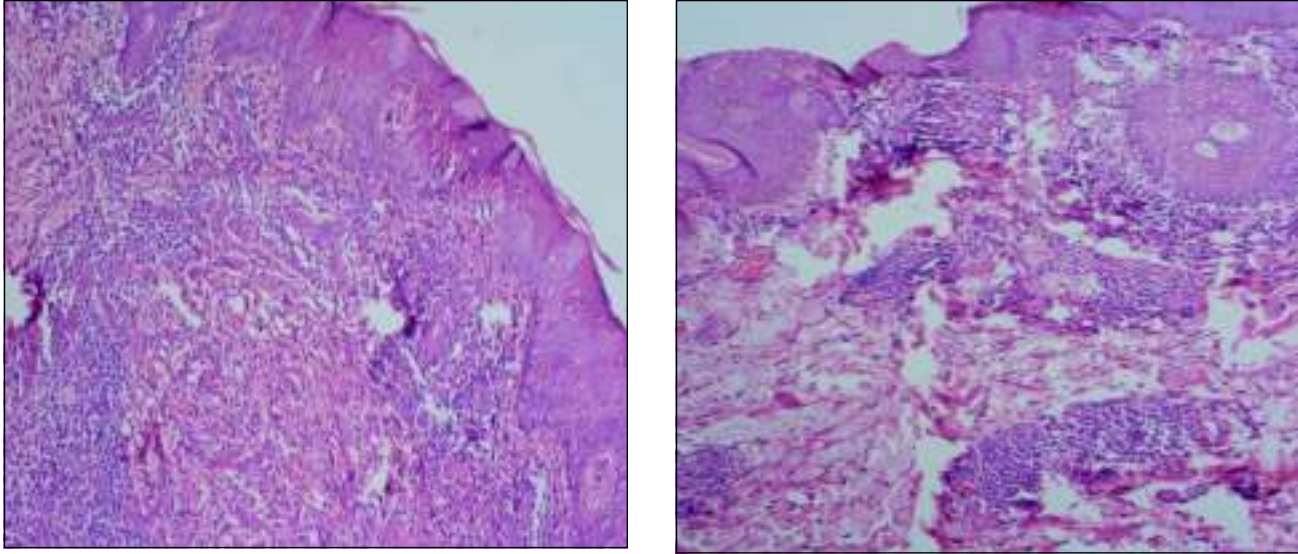


**Fig.-2:** Papulo nodular lesion in left dorsum with biopsy mark

Patients Hb was 15.2%, ESR was 10 mm in 1<sup>st</sup> hour, Total count of WBC was 4800, Neutrophil-57%, Lymph-38%. Routine urine microscopy was normal. Liver function test revealed SGPT -93IU/L, SGOT-78IU/L, s albumin was 46mg/dl, sr Globulin was 32mg/dl and albumin globulin ratio was 1.44:1, sr creatinine was 1.2 mg/dl. His HBsAg and Ig G anti HCV status was normal. His chest x ray revealed no abnormalities. Ultrasonography of whole abdomen showed incidental left sided nephrolithiasis. ICT (rK 39) for Kala azar was negative. Skin biopsy with specimen consists of 2x8x4 cm piece of skin from left elbow showed dermis revealing dense focal infiltrates of lymphocytes, histiocytes and small number of plasma cells. A small number of apoptotic bodies are present. In H and E stains and Giemsa stain revealed small number of *Leishmania donovani* (LD) body.



**Fig 3:** Skin histopathology showed LD body



**Fig.-4:** Skin histopathology showed absence or anergy of LD body after getting treatment.

After all the investigation he was diagnosed as a case of Cutaneous leishmaniasis and nephrolithiasis (left). The patient was treated with Inj Sodium Stibogluconate (SSG) 20mg/kg body weight (14.4 ml) in two deltoids for 30 days. He was followed up regularly for skin lesions. After completion of his course, the skin lesions of nodularity disappeared but the lesions remain flattened and hyperpigmented. The second skin biopsy was done after completion of SSG course and it revealed no abnormality. He was seen regularly in follow up and his last follow up was on 27.02.2011. There was no relapse of the skin lesion and the older papular lesions become hyperpigmented only.

#### **Discussion:**

Cutaneous leishmaniasis (also known as “Aleppo boil,” “Baghdad boil,” “Bay sore,” “Biskra button,” “Chiclero ulcer,” “Delhi boil,” “Kandahar sore,” “Lahore sore,” “Leishmaniasis tropica,” “Oriental sore,” “Pian bois,” and “Uta”) is the most common form of leishmaniasis.<sup>1</sup>

The geographical distribution of CL is mainly determined by the sandfly vectors (*Phlebotomus* sp and *Lutzomyia* sp). They live in dark, damp places, weak flyers, with a range of only 50 metres from their breeding site, most active at evening and night and rainfall and global warming has direct effect for its number. The risk is highest from dusk to dawn because sand flies typically feed (bite) at night and during twilight hours. Although sand flies are less active during the hottest time of the day, they may bite if they are disturbed (for example, if hikers brush up against tree trunks or other sites where sand flies are resting). Vector activity can easily be overlooked: sand flies do not make noise, they are small (approximately one-third the size of mosquitoes), and their bites might not be noticed<sup>6,7</sup>

That is the reason why the disease is more commonly seen in tropics and especially in desert areas. In this case report the patient who suffered from the lesion did not have any residence or working habits in rural or desert areas. He lived in Riyadh (capital city) of Saudi Arabia and during his long stay, he did not have any travel to the areas where the disease is common. It is surprising how he got the interaction with the sandfly. In Bangladesh he used to live in non endemic zone of kala azar and there was no brief travel in lifetime to the endemic zone too. So the introduction of the disease in Bangladesh is also a remote possibility.

The incubation period of CL is two to eight weeks, although longer periods have been noted. The disease begins as an erythematous papule at the site of the sandfly bite on exposed parts of the body. The papule increases in size and becomes a nodule. It eventually ulcerates and crusts over. The border is usually raised and distinct. There may be multiple lesions, especially when the patient has encountered a nest of sandflies. The patient in this case reports have lesion similar that is characteristics of the cutaneous leishmaniasis although there was no ulcer and there was no scaliness or regional lymphadenopathy. Most lesions heal over months or years, leaving an atrophic scar. In general half those lesions caused by *L. major* or *L. mexicana* will have healed in 3 months, those caused by *L. tropica* take longer – about 10 months – and those due to *L. braziliensis* persist much longer.<sup>8,9</sup> Natural resolution leads to partial resistance to reinfection.<sup>9,10</sup> The patients in our case has illness for only three months but as the new lesions are coming up with existence of older one, he felt annoyed with the skin lesions and took many consultation for cure.

The lesions of cutaneous leishmaniasis is usually anergic and sometimes it is difficult for microbiologist to yield the positivity in biopsy specimen. In our case two biopsy was done before starting treatment. One with the positive result while the other one was negative. The posttreatment clinical improvement was also followed by rebiopsy of lesions around elbow and it was found negative. The immunological response of CT is also variable from that of PKDL. The nodular lesions of PKDL is usually composed of dense parasite at lesions site and immunological test is usually (rK39) positive while in CT it is more commonly negative. The case in our hospital was also similar to the above statement where we found that rK39 test was negative while intensive search was needed by microbiologist to delineate the LD body in skin histopathology.

The disease of CL is not common in Bangladesh. The expertise to diagnosis and treatment is also lacking. The treatment protocol followed in this patient was based on management of CL in Mediterranean region where travellers are usually treated with inj SSG when lesions are more than 5 in number and increasing with time<sup>11</sup>. The duration is usually 20 days. The intralesional injection is also common practised in the desert areas when the lesions number is less. In our index case we started the treatment with SSG IM and observe his lesions daily for two weeks. There was small improvement initially and so we decided to continue the treatment for another two weeks and eventually gave him 30 days of SSG. ECG monitoring was done in every week. There was no complication related to SSG happened to him. After completion of therapy we examined him clinically and found the lesions healed up with only hyperpigmentation. The rebiopsy was done and it was found absence or anergy of LD body as expected.

#### Conclusion:

Although cutaneous leishmaniasis was not reported frequently in Bangladesh, recent few cases with purely skin lesions of leishmaniasis which are not lesions like PKDL are observed. The pattern, distribution of lesions and response to treatment suggested that cutaneous leishmaniasis is actually presented in Bangladesh recently. Although the reported cases are all derived from desert areas, the absence of risk factor and absence of travel to endemic zones of cutaneous leishmaniasis prone zone invites questions of origin from Bangladesh. The vector search is crucial at index case

residence to explore the disease in Bangladesh and thereby we need expertise to develop in cutaneous leishmaniasis.

#### Acknowledgement:

Professor kamaluddin who had done the histopathological slides and also provide the photography.

**Conflicts of Interest:** None

#### References:

1. Dedet JP, Pratlong F. Leishmaniasis. In: Manson P, Cook GC, Zumla A, eds. *Manson's Tropical diseases*. 21st ed. London: Saunders, 2003:1339–64
2. Herwaldt BL. Leishmaniasis. *Lancet*. 1999;354:1191–9
3. Reithinger R, Dujardin JC, Louzir H, Pirmez C, Alexander B, Brooker S. *Lancet Infect Dis*. 2007; 7(9):581-96
4. Ahluwalia S, Lawn SD, Kanagaingam J, Grant H, Lockwood DN. Mucocutaneous leishmaniasis: an imported infection among travellers to central and South America. *BMJ*. 2004 9; 329(7470):842–4
5. A Case of Diffuse Cutaneous Leishmaniasis in a HIV Positive Patient. I Patwary, M Rahman, M Ahmed, S Ahmed, Md, MSR Chowdhury. *Journal of Bangladesh College of Physicians and Surgeons* 2011; 29: 106-108
6. James, William D.; Berger, Timothy G.; et al. (2006). *Andrews' Diseases of the Skin: clinical Dermatology*. Saunders Elsevier. ISBN 978-0-7216-2921-6
7. Gonzalez R, De Sousa L, Devera R JA, Ledezema E. Seasonal and nocturnal domiciliary human landing/biting behaviour of *Lutzomyia (Lutzomyia) evansi* and *Lutzomyia (Psychodopygus) panamensis* (Diptera: Psychodidae) in a periurban area of a city on the Caribbean coast of eastern Venezuela (Barcelona; Anzoategui State). *Trans R Soc Trop Med Hyg* 1999; 93: 361-4
8. Bryceson A. Therapy in man in: Peters W. Killick-Kendrick eds. *The leishmaniasis in biology and medicine*. London: Academic press; 1987. pp.847-907
9. Herwaldt BL, Arana BA, Navin TR. The natural history of cutaneous leishmaniasis in Guatemala. *J Infect Dis* 1992; 165:518-27
10. Locksley RM, Louis JA. Immunology of leishmaniasis. *Curr Opin Immunol* 1992; 4: 413-8
11. Blum J, Desjeux P, Schwartz E, Beck B, Hatz C. Treatment of cutaneous leishmaniasis among travellers. *J Antimicrob Chemotherapy*. 2004; 53(2):158-66