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A Case Report of Leprosy in Eastern Sudanese

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Abstract

Leprosy (Hansen's disease) is an ancient contagious chronic disorder, affects mainly skin, peripheral nerves, mucosa of upper respiratory tract and the eyes. It is a granulomatous infectious disease that is triggered by *Mycobacterium leprae*. If untreated, leprosy is a leading cause of long-term physical disability. Statistically speaking, Sudan has not achieved the World Health Organization target for the elimination of leprosy (<1 case per 10.000 people). Mycobacterium leprae is the only bacterium that has not been cultured in a laboratory. It is diagnosed by skin smear or scraping, biopsy, serology, animal inoculation, and polymerase chain reaction (PCR). We present a case of diffuse lepromatous leprosy (multibacillary leprosy) in an eastern Sudanese, 43-years old male complaining of a persistent pruritic rash throughout his body. The diagnosis was established after Zeihl-Neelsen staining (ZN) of a skin smear prepared from skin lesions on the upper limb revealed the detection of an acid-fast bacilli (AFB). The patient was not diagnosed until he met the consultant dermatologist. Overall, this report indicates that lepromatous leprosy is frequent in eastern Sudan populations and highlights the demand for continued efforts that encourage awareness programs and conserve, control activities in the eastern Sudan.

Keywords

Leprosy, AFB, Skin Smear, Multibacillary, Sudan

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1. Introduction

Hansen disease or leprosy is a chronic contagious disease that is caused by an acid-fast rod's bacillus called *Mycobacterium leprae* [1]. About 211,009 new cases in 159 countries were registered globally [2]. World health organization (WHO) reports appraised that some countries still have a few districts which have not achieved elimination including Sudan. Sudan reported between 300 – 900 cases per year [3]. *Mycobacterium leprae* is transmitted following prolonged close contact with untreated cases via droplets from the nose and mouth. *Mycobacterium leprae* replicates slowly and the incubation time of the disease on average is 5 years [4]. The precise mechanism of transmission of leprosy is obscure. The most commonly held belief was that the disease was switched by contact between the cases of leprosy and healthy subjects [5]. Clinically, the signs are easy to note. In a region with an increase incidence of leprosy, an individual should be considered as having leprosy if he or she appears one of these sings; skin lesion consistent with leprosy and with definite sensory loss, with or without thickened nerves as well as positive skin smear [6]. The skin lesion may be single or numerous, usually less pigmented. Occasionally, the lesion is reddish or copper-colored. The skin lesion may be shown macules (flat), papules (raised), or nodules. Sensory loss is regarded a typical feature of leprosy. Peripheral nerves comprise another feature of leprosy. In the lack of these signs, nerve thickening by itself without sensory loss and/or muscle weakness is overwhelming not a credible sign of leprosy [7]. The symptoms may have developed

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within the first year, but can also take as long as 20 years or more [3]. Lately, the possibilities of crossover by respiratory route is gaining ground as well as other possibilities such as transmission via insect which cannot fully eliminate [8]. Untreated cases lead to progressive permanent damage of skin, nerves, and limbs as well as eyes. Leprosy is a curable disease with multidrug therapy (MDT) [3].

2. Case Presentation

A 43 years-old male from rural eastern Sudan came downtown Port Sudan Dermatology Hospital, Sudan. He complained many facial and body nodules developed over the past months. He was given an antibiotic by a local rural clinic two weeks' ago with no any improvement in the nodules. He had no notifies on fever, chills, and night sweats. On examination the patient had thick skin, numbness in the hands and feet, and peripheral neuropathy. Since he admitted he had an ulcer seen in the uncle and he was not aware of his medical diagnosis. Furthermore, his blood pressure was 110/70, pulse rate 84, respiratory rate 16, and body temperature 37.4°C. There was also conjunctivitis and no corneal injury. The complete blood count (CBC) findings revealed an increased white cell count (12.6 x10^3/µl), many stab forms (unsegmented neutrophils) in the differential count and toxic granulation of neutrophil. Human immunodeficiency virus (HIV) was negative, erythrocyte sedimentation rate was elevated, increment in C-reactive protein (CRP), normal renal function and electrolytes. Urinalysis demonstrated a slight proteinuria (trace), no pus cells, dysmorphic erythrocytes (14 – 16/hpf), and erythrocytic cast (+) (Table 1). Skin smear treated with cold Zeihl-Neelsen stain disclosed a diffuse *Mycobacterium leprae* (Figure 1). The patient was ascertained as leprosy as defined by the World Health Organization's (WHO) standard diagnostic criteria illustrated in Table 2 [4]. Consequently, a course of multi-drug therapy combining Dapsone (100 mg / day), Clofazimine (50 mg / day) and Rifampicin (600 mg/monthly) was prescribed for a period of 2 years. The lesion nodules were exhibited well improvement after completion of therapy. Occasionally, Liver function tests (LFT) were ordered to ensure hepatotoxicity or adverse effect did not result from his concurrent use of other medications.

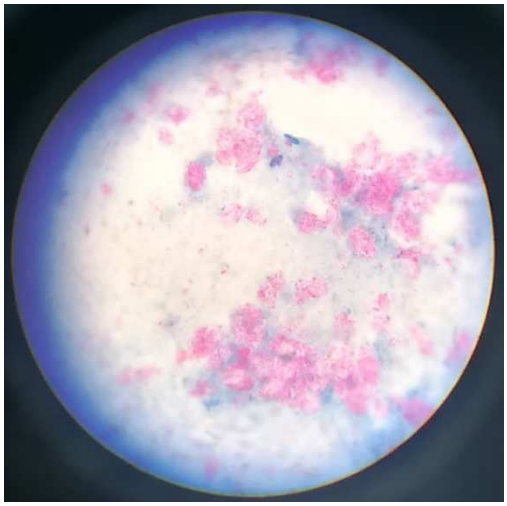

Figure 1. Skin smear with ZN stain highlighted a diffuse of AFB (original magnification X100).

Table 1. Laboratory findings of the case.

Test	Patient results	Reference interval
White blood cells /µl	12.6	4 – 10
Red blood cells /µl	3.72	3.5 – 5.5
Hemoglobin g/dl	12.0	12 – 16
Hematocrit %	33	35 – 47
MCV fl	88	78 – 98
MCH pg	32	26 – 35
MCHC %	37	30 – 36
RDW-CV	14.3	11.5 – 14.5
Absolute lymphocyte count /µl	4.0	1 – 3.4
Absolute neutrophil count /µl	6.4	2 – 7.0
Stab form /µl	1.16	Up to 0.6
Platelet count /µl	239	150 – 400
MPV fl	9.6	7.5 – 10.4
PDW fl	12.2	9 - 17
P-LCR %	22.3	15 – 35
RBC Morphology	Normal	—
Rouleaux formation	Moderate	—
HIV screening	Negative	—
ESR mm/hr	84	Up to 30
CRP mg/l	23.4	Up to 10
Blood Urea level mg/dl	27.5	10 – 50
Blood Urea Nitrogen mg/dl	11.5	7 – 21
Serum Creatinine level mg/dl	1.24	0.4 – 1.6
Serum Uric acid level mg/dl	5.7	3.4 – 7.0
Serum Phosphorus level mg/dl	2.56	2 – 5

Test	Patient results	Reference interval
Serum Calcium level mg/dl	11.0	8.5 – 11.0
Serum Potassium level mmol/l	5.2	3.5 – 5.5
Serum Sodium level mmol/l	134.6	135 – 145
Random Blood Glucose level mg/dl	81	80 - 180
Proteinuria	Trace	—
Erythrocyturia hpf	16 (dysmorphic)	—
Erythrocyte cast	+	—

Table 2. WHO diagnostic guidelines for Leprosy.

World Health Organization, operational diagnostic criteria for leprosy
1. Skin lesion consistent with leprosy and with definite sensory loss, with or without thickened nerves
2. Positive skin smears

3. Discussion

Leprosy is a chronic infection that is endemic in tropical countries, causing a broad-spectrum clinical manifestation [1]. The causative agent is *Mycobacterium leprae* or Hansen's bacillus spirilly [3]. The clinical remarks of leprosy are vastly variable. Ridley and Jopling were describing the leprosy spectrum criteria as tuberculoid leprosy, borderline conditions, and lepromatous leprosy [9]. On the other hand, WHO also suggested leprosy classification based on the number of AFB detected in the skin smear as either paucibacillary or multibacillary. The paucibacillary categorize associates with tuberculoid, borderline tuberculoid, and indeterminate leprosy, and multibacillary consensual with borderline lepromatous and lepromatous leprosy. Paucibacillary leprosy usually displays an unwired clinical picture than multibacillary leprosy [10]. The clinical presentation is dependent on the extent of immune response to *Mycobacterium leprae* [1].

In this setting, the social, environmental details and contact history thoroughly predict the risk of exposure. Therefore, the bacterial load also can predict the transmission risk. Our patient had a diffuse number of AFB (multibacillary) which is consistent with WHO categories [4]. Underlying diseases such as scleroderma, mycosis fungoides, pellagra, asteatosis, ichthyosis and eczema or contact dermatitis should be differentiated from multibacillary lepromatous skin lesions. Due to inadequacy, laboratory skin biopsy was not performed. This biopsy investigation is not critically required to make a formal diagnosis (Table 2). This patient was firstly misdiagnosed at the rural health clinic, which indicates that many cases are still missing in this region of Sudan. This misdiagnosis probably due to some attributable factors such as insufficient knowledge and skills of the health staff, poor registration and recording system and lack of decentralization of health centers. However, to our knowledge, there are very few recent studies in this country, although the disease still existed.

4. Conclusion

Proper diagnosis and treatment of leprosy can inhibit incapacitating disease. In Sudan, especially eastern region, the future researches that implicate the impact and clinical course of the disease should be viewed. Overall, this report highlights the demand for continued efforts that encourage awareness programs and conserve, control activities in eastern Sudan.

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