DISSIMINATED CUTANEOUS LEISHMANIASIS IN A MIDDLE AGE LADY FROM AFGHANISTAN

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ABSTRACT

Objective: The objective of this study was to compare the frequency of adverse events (hypoglycemia and hypocalcaemia) in Jaundiced Neonates (having bilirubin level of >20mg/dL) with and without co-morbid conditions (anemia (Hb<10mg/dl) and sepsis (culture proven)

Material and Methods: This study was conducted at Special Care Baby Unit Khyber Teaching Hospital, Peshawar. Duration of study was one year and study design was prospective observational study. Patients without co-morbid conditions were included in group "A" and those with co-morbidity were in group "B". 62 patients in each group were enrolled. Inclusion criteria of the study was Jaundiced neonates having total serum bilirubin >20mg/dl and Jaundiced neonates with and without comorbid condition while exclusion criteria was exchange transfusion for polycythemia and birth asphyxia grade III.

Results: Hypocalcaemia was analyzed showing that in Group "A" out of n=38(61.29%) male neonates, only n=13(34%) had Hypocalcaemia which ranged from 6.5-8.9 mg/dl with a mean Hypocalcaemia level of 7mg/dl with SD±0.81mg/dl while out of n=24(38.71%) female neonates only 5(20%) had Hypocalcaemia ranging from 6.2-8.8 mg/dl with a mean Hypocalcaemia level of 7mg/dl with a SD ±0.76 mg/dl. Where as in Group "B" out of 30(48.38%) male neonates only 3(10%) had Hypocalcaemia ranging from 6.6-8.8 mg/dl with a mean hypocalcaemia level of 7mg/dl with a SD ±0.68 mg/dl while n=32(51.61%) female neonates only 3(9%) had hypocalcaemia which ranged from 6.1-8.9 mg/dl with a mean hypocalcaemia level of 7mg/dl with a SD ±0.69 mg/dl.

Hypoglycemia among two groups was analysis as in Group "A" n=38(61.29%) male neonates only 13 had Hypoglycemia which ranged from 18-45 mg/dl with a mean Hypoglycemia level of 33mg/dl with a SD±5.23 mg/dl while n=24(38.71%) female neonates only 10 had hypoglycemia which ranged from 16.2-44.5 mg/dl with a mean Hypoglycemia level of 31mg/dl with a SD ±6.01 mg/dl. Where as in Group "B" n=30(48.38%) male neonates only 5 had Hypoglycemia which ranged from 20-44.6 mg/dl with a mean hypoglycemia level of 33mg/dl with a SD ±6.22 mg/dl while 32(51.61%) female neonates only 4 had Hypoglycemia which ranged from 19-45 mg/dl with a mean Hypoglycemia level of 30mg/dl with a SD ±6.39 mg/dl.

Conclusion: It was concluded from the study that adverse events of hypocalcemia and hypoglycemia are significantly higher in neonates with comorbid conditions relative to ones without comorbid conditions. Moreover number of female neonates suffering from these conditions is higher than male neonates, in our study which is irrespective of the comorbid conditions.

Key Words: Exchange transfusion, Neonatal jaundice, hypocalcemia, hypoglycemia

INTRODUCTION

Cutaneous leishmaniasis is a protozoal disease caused by Leishmanial parasite.[1] It is transmitted by bite of Sandfly. Presentation of this disease ranges from asymptomatic nodule to multiorgan fatal disease. It is a rapidly emerging disease & constitutes a public health problem in endemic areas. Diffuse cutaneous leishmaniasis is a disseminated variant of cutaneous leishmaniasis which is chronic, progressive and has treatment-refractory nature.[2] It constitutes approximately 20% of cutaneous leishmaniasis.[3] We report case of a patient presenting with chronic disseminated lesions.

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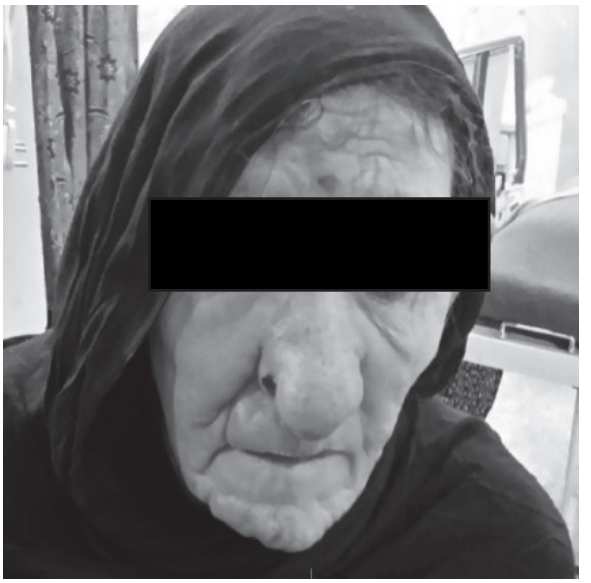
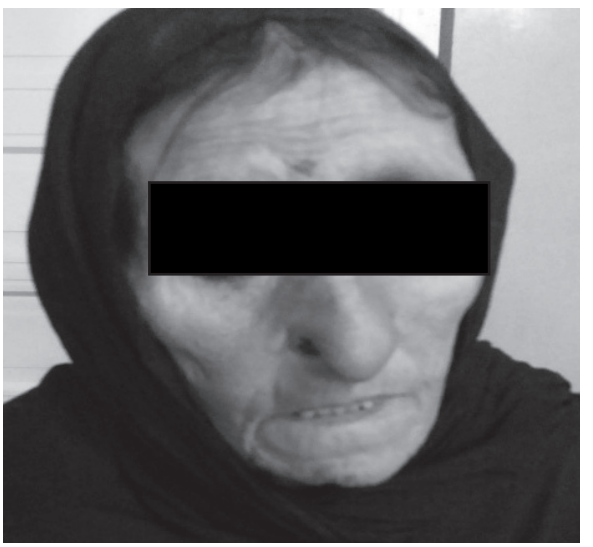
CASE PRESENTATION

A 60 years old lady from Afghanistan, farmer by occupation presented to outpatient department of Dermatology unit Hayatabad Medical Complex Peshawar on 1st April 2016 with three years history of slowly progressive erythematosus Infiltrated plaques and nodules involving forehead, cheeks, nose right ear lobe and upper and lower limbs asymmetrically. These lesions

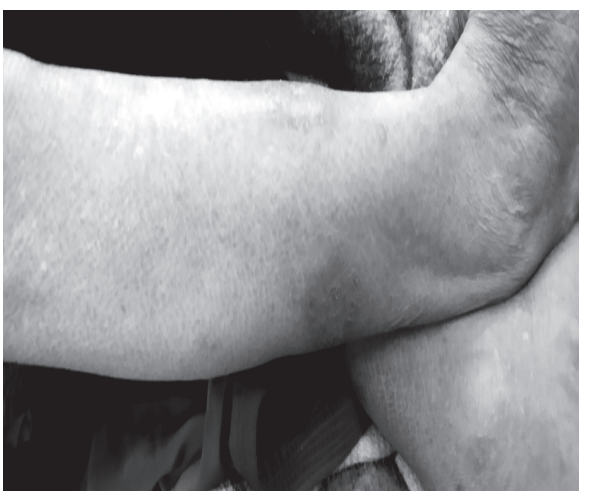
started as erythematosus nodule on forehead gradually increased in size leading to erythematosus infiltrated plaques without developing any ulceration and extending to involve the above mentioned sites over a period of three years. It was associated with mild pruritus otherwise no cutaneous or systemic symptoms.

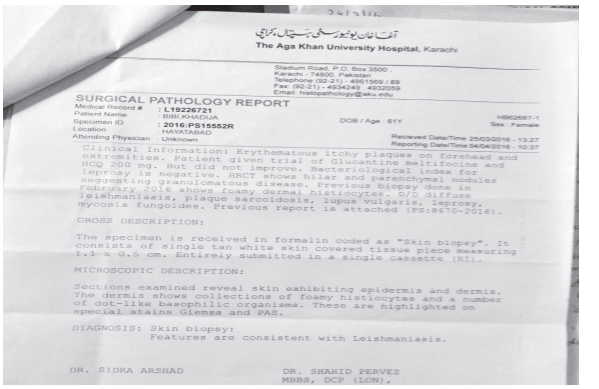


Before treatment



After treatment





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SURGICAL PATHOLOGY REPORT

Medical Record # : L19226721
Patient Name : BIBI KHADIJA
Specimen ID : 2016;PS15552R
Location : HAYATABAD
Attending Physician : Unknown
DOB / Age : 61Y
Sex : Female
Recieved Date/Time: 25/03/2016 - 13:27
Reporting Date/Time: 04/04/2016 - 10:37

Clinical Information: Erythematous itchy plaques on forehead and extremities. Patient given trial of Glucantine meltifocine and HCQ 200 mg, but did not improve. Bacteriological index for leprosy is negative. HRCT shows hilar and parenchymal nodules suggesting granulomatous disease. Previous biopsy done in February, 2016 shows foamy dermal histiocytes. D/D diffuse leishmaniasis, plaque sarcoidosis, lupus vulgaris, leprosy, mycosis fungoides. Previous report is attached (PS:8670-2016).

GROSS DESCRIPTION:

The specimen is received in formalin coded as "Skin biopsy". It consists of single tan white skin covered tissue piece measuring 1.1 x 0.5 cm. Entirely submitted in a single cassette (B1).

MICROSCOPIC DESCRIPTION:

Sections examined reveal skin exhibiting epidermis and dermis. The dermis shows collections of foamy histiocytes and a number of dot-like basophilic organisms. These are highlighted on special stains Giemsa and PAS.

DIAGNOSIS: Skin biopsy:
Features are consistent with Leishmaniasis.

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Mucosae were spared and she was not having any visceral abnormality. Her personal and family history was negative for tuberculosis. She has used injection sodium stibogluconate intramuscularly for 3 weeks without any response. Her bacteriological index was negative. HIV serology was negative and smear from lesion for LD (leishmen donovan) bodies was positive. Biopsy taken from involved skin showed foamy macrophages full of amastigotes with normal epidermis. Clinical and histopathological evidence was suggestive of diffuse cutaneous leishmeniasis. She was started on low dose (50mg BD) Meltifosine after doing baseline investigations including Full blood count, liver and renal function tests. She was followed for 2 months she was lesion and symptoms free after using Meltifosine for 2 months.

DISCUSSION

Cutaneous leishmaniasis is a protozoal disease transmitted by sandfly vectors. There are many different species of Leishmania, each confined to a geographical region of the world and transmitted by the bite of the female sand fly vector. [2] Bites usually occur at night on exposed skin and are not normally painful, therefore patients rarely recall being bitten. Clinical manifestations of leishmaniasis ranges from non aggressive cutaneous ulcers to systemic multiorgan disease. There are 3 types of leishmaniasis, cutaneous mucocutaneous and visceral leishmaniasis. Different clinical presentations of leishmaniasis depend on Immune status of person. Leishmania-specific anergy leads to developmentof DCL. Poor immune response or immunosuppression (HIV/AIDS) leads to visceral leishmaniasis. Hyperergic variant: Leishmaniasis recidivans is caused by L.tropica.[3]

Estimated prevalence of CL is upto12 million people infected worldwide with annual incidence of about 2 million. [1, 4] Disease is endemic in 90 countries mostly developing countries[1].More than 80% of cases of CL occur in Afghanistan, Algeria, Iran, Iraq, Saudi Arabia, Syria, Brazil, Peru. Climate in these countries range from deserts to rain forests, rural to urban. [1] The number of DCL cases has shown a statistically significant increase in the past 20 years. [5, 6,7]

Diffuse cutaneous leishmaniasis(DCL) occurs in approximately 20%of individuals with widespread skin involvement resembling lepromatous leprosy.It occurs in immunocompetent individuals in which cellular immunity fails to clear Leishmania parasite. It is caused by L. aethiopica & is characterised by small initial lesion, spreading locally, disseminating to larger areas of skin without involving internal organs. Llesions do not ulcerate & have superabundance of parasites in the lesions.[3] Histology shows masses of macrophages full of amastigotes , with little or no lymphocytic infiltrate, a normal or attenuated epidermis. The leishmanin test and other tests of specific cellular immunity are negative. The disease has chronic progressive course with slow and poor response and relapse is the rule. [1]

Lesions of DCL commonly involve central on the face. Satellite papules aggregates to form into a large non ulcerating seldom inflammed nodules and plaques. Mucocutaneous leishmaniasis may develop if bite is on mucocutaneous junction. [1]

Currently, first-choice treatment for cutaneous leishmaniasis is pentavalent antimony compounds meglumineantimoniate or sodium stibogluconate[8,9]. Drugs like miltefosine ,amphotericin B and Ketoconazole can be used as second line.[10,11,12.]The dose of pentavalent antimonial for all forms of leishmaniasis is 20 mg/kg/day.[13] DCL treatment is associated with worse prognosis owing to a high probability of recurrence. [14]

CONCLUSION

DCL appears to be increasingly occurring because the causative agent along with vector are freely available in endemic areas. We consider the diffuse cutaneous leishmaniasis is under reported.

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