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Lucio Leprosy in Neglected Cases of Hansen's Disease: A Series of Three Cases

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ABSTRACT

Lucio Leprosy is a rare form of Lepromatous leprosy, which normally occurs in chronic untreated patients of Hansen's disease. Lucio Leprosy, was first reported from Mexico and henceforth commonly known as Mexican leprosy, but is now being increasingly reported sporadically across the globe. Lucio leprosy presents as slowly progressive diffuse infiltration of skin all over the body with shiny waxy thickened skin, loss of body hair including eyebrows and eyelashes, puffy hands and sensory loss due to involvement of dermal nerves. There will be thickening of upper eyelids that gives the patient a sleepy look (Melancholy look). This case series pertains to three such patients (58 year and 63 year old females and 55 year old male) of chronic lepromatous leprosy who had features suspicious of Lucio leprosy. Each case of the present series had a unique clinical presentation like diffuse infiltration of skin, total loss of eyebrows, oedema of hands, characteristic triangular ulcers with jagged borders and was posing challenge to the treating Dermatologist. Heavy colonisation of *Mycobaterium leprae* was observed in the endothelium of small capillaries in the superficial dermis. Involvement of these capillaries manifested as haemorrhage and infarction of the overlying epidermis. As this unusual variant of untreated chronic lepromatous leprosy leads to potentially lethal complications, which are irreversible therefore the present study has been designed to highlight the factors predisposing to Lucio leprosy and its impact on the quality of life.

Keywords: Distal tubular injury, False positive rheumatoid factor, Mexican leprosy, Vasculitis

INTRODUCTION

Lucio leprosy is a pure, diffuse, non nodular form of lepromatous leprosy. It is encountered exclusively in Mexico and Central America and is quite rare in rest of the world. But recently Lucio Leprosy cases are being reported in few Asian countries also [1]. Lucio Leprosy is a rare type of reaction seen in cases of diffuse lepromatous leprosy. It usually manifests as a painful purpuric spot that evolves into well defined, angulated, jagged ulcers involving limbs, but rarely involves trunk and face. Lucio phenomenon usually occurs in untreated patients of Hansen's disease and defaulters [2].

CASE SERIES

Case 1

A 58-year-old female, home maker, presented to the dermatology Outpatient Department (OPD) of a tertiary care centre, with complaints of multiple non healing ulcers, loss of eyebrows, diminished sensations over hands and feet since two months. Patient also complained of pain and foul smelling discharge from multiple non healing ulcers for the past 15 days. She was an undiagnosed, untreated case of Hansen's disease. She didn't have any other co-morbidities and was not taking any drugs.

Patient gave history of asymptomatic diffuse nodular lesions over anterior and posterior aspect of the trunk for the past one and half years [Table/Fig-1a,b]. Patient had diffuse infiltration of skin, coppery hue all over the skin surface, areas of blackish brown macular pigmentation [Table/Fig-2a,b], depressed bridge of the nose. Routine investigations revealed very low haemoglobin (8 gm%), patient was also positive for Anticardiolipin Antibody (ACL) and false positive Rheumatoid Factor (RF). Patient was subjected to thyroid profile screening and was found to be euthyroid. Patient was admitted and subjected to a slit skin smear, which was positive for *Mycobacterium leprae* and biopsy of the ulcer confirmed the diagnosis of vasculitic ulcer of Lucio Leprosy on HP examination with Haemtoxylin and Eosin (H&E) staining [Table/Fig-3].

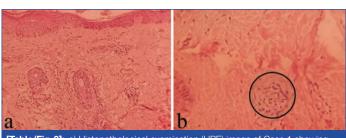
Since the pretreatment haemoglobin level was low around 8 gm%, patient was started on Dapsone free Multibacillary Multidrug Therapy (MB-MDT) (Rifampicin 600 mg once a month and Clofazimine 300 mg once a month and 50 mg daily without



[Table/Fig-1]: Case 1-a,b) Lesions of calciphylaxis before ulceration



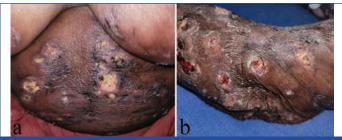
[Table/Fig-2]: Case 1- a) Posterior aspect; b) Anterior aspect of repromatous nodules before onset of Lucio leprosy.



[Table/Fig-3]: a) Histopathological examination (HPE) image of Case 1 showing small vessel vasculitis (H&E 10X). b) HPE image showing inflammatory infiltrate around blood vessel (H&E 40X).

Dapsone) as Dapsone has a potential to cause haemolytic anaemia, along with appropriate dose of systemic steroids as per World Health Organisation (WHO) guidelines [3,4]. Vasculitic lesions started healing well and patient was discharged, with the advice of regular follow-up of once in two weeks, for tapering of steroids and monitoring of disease activity. Patient was on MB-MDT without dapsone, since September 2021, with no known co-morbidities.

Patient after taking MB-MDT for six months, defaulted for three months, and stopped systemic steroids too, and came back to Dermatology OPD, with acute exacerbation and dissemination of the lesions in the form of painful multiple vasculitic ulcers of 15 days duration. On examination, multiple painful punched out ulcers of varying size over upper and lower extremities, trunk and lower back were present [Table/Fig-4a], lesions first started as fluid filled vesicles which then ruptured to form large triangular jagged ulcers. Patient was very weak, debilitated, with a Blood Pressure (BP) record of 70/50 mmHg.



[Table/Fig-4]: Case 1: a) Ulceronecrotic lesions of anterior trunk; b) Ulceronecrotic lesions of left elbow joint.

Dermatological examination revealed multiple punched out ulcers of varying sizes with erythematous edges, necrotic non viable tissue with slough, mostly present on the limbs and less on abdomen and face [Table/Fig-4b]. There was total loss of eyebrows and eyelashes with ear lobe infiltration (Buddha ear). Patient also had ulcers involving the malar area which showed the severity of Lucio phenomenon [Table/Fig-5a,b].



[Table/Fig-5]: Case 1: a) Malar area lesions indicating severity of Lucio phenomenon; b) Total loss of eyebrows, saddle nose deformity, necrotic ulcers of left malar area.

Laboratory investigations revealed low haemoglobin- 5.9 gm%, serum potassium- 3.4 mmol/lit, albumin- 2.9 gm/dL, other parameters were normal. As patient had anticardiolipin, antibody positive, areas of calciphylaxis, over a period of time ulcerated to form typical vasculitic ulcers of lucio phenomenon. Patient had features of septicaemia, because of chronic malnutrition patient was debilitated, hence couldn't even accomplish the task of day to day activities of life. Patient was started on Inj. noradrenaline drip as per physician's opinion for a period of three days. After three days patient was started on parenteral fluids to restore normal BP and maintain her basic fluid and electrolyte balance. i.v. Antibiotics (Inj piperacillin- tazobactum 4.5 gm i.v. tds and Inj. meropenam 1 gm i.v. tds for 14 days) were started according to pus Culture and Sensitivity (C/S). Two units of Packed Red Blood Cells (PRBCs) were transfused and patient Hb improved to 8.9 gm%. As the general condition of the patient improved, in order to halt the progression of Lucio phenomenon, the patient was started on Inj. methylprednisolone pulse 250 mg and 500 mg in 500 mL of normal saline, slow i.v., over a period of four hours, given three weeks apart, in order to improve the tolerance of the patient to supra pharmacological doses of super potent steroid. Even after Inj. methyl prednisolone was stopped and inspite of oral Potassium Chloride (KCL) two tsp tds, supplementation patient had persistent hypokalemia and hence Nephrologist opinion was sought. For correction of hypokalemia Inj. KCL two ampules, followed by two ampules of Magnesium Sulphate (MgSO₄), were given for five days along with oral syp KCL 30 mL QID daily.

Inspite of all these interventions serum potassium was 3.4, and thus the Nephrologist suspected Distal Tubular Injury of the kidneys, because of long standing untreated and defaulted lepromatous leprosy, which was confirmed by, acid loading test and urine pH (>5.5) was monitored along with urine creatinine (14 mg%) and potassium level estimation which finally confirmed distal tubular injury. In a long standing case of lepromatous leprosy down grading to Lucio Leprosy, irreversible distal tubal injury, is a new finding that was encountered in this case series of Lucio Leprosy. Patient's potassium level was stabilised at 4 meg/lit and patient was shifted to oral supplements syp. KCL, 2 tsp tds. As the patient has got irreversible distal tubular injury, Patient has to be on oral potassium supplements life long with regular monitoring of serum potassium levels. After all these interventions patient's condition improved, ulcers started healing [Table/Fig-6a-c]. Patient was discharged after three months of inpatient care and had been advised to attend the OPD for regular follow-up.



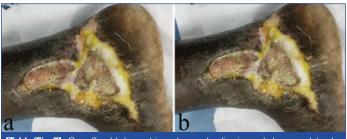
Case 2

A 63-year-old female, a known case of lepromatous leprosy, presented at the Dermatology OPD of a tertiary care centre, with the complaint of ulcer over both legs with maggots since two weeks, patient was declared Released From Treatment (RFT) in 2018 with no known co-morbidities.

Dermatological examination revealed triangular jagged ulcers present over both lower limbs with unhealthy granulation tissue, and morphology of the ulcers were compatible with vasculitic ulcers of Lucio leprosy. Patient had diffuse infiltration of skin with total loss of eyebrows. There was a large triangular jagged out ulcer, larger in dimension in left leg than right leg. The ulcer didn't show a tendency to heal inspite of standard treatment with both broad spectrum parenteral antibiotics along with topical human recombinant epidermal growth factor. After ensuring Blood C/S and Pus C/S negative patient was started on parenteral steroids viz., Inj dexamethasone 2 cc i.m. OD for a period of two weeks and then gradually tapered as 5 mg/wk and converted into oral steroids. Maggots were removed and she was treated with antibiotics (T.linezolid 600 mg BD for 14 days). Laboratory investigations revealed

Anticardiolipin antibodies, IgG (> 26 GPL U/mL) and IgM (>14 GPL U/mL), Erythrocyte Sedimentation Rate (ESR), and C-Reactive Protein (CRP) were elevated. Patient was started on phenytoin dressing of ulcer over right foot and three sittings of Platelet-Rich Fibrin (PRF) dressing for ulcer over left foot was given.

Lesions were healing only partially and fresh viable epithelial tissue growth was not observed even after three sessions of PRF dressing over left foot [Table/Fig-7a,b] with all these conservative management and special intervention of Platelet-Rich Plasma (PRP) and PRF nearly for two months, the ulcers didn't show signs of healing. Hence, the markers of acute inflammation were repeated, along with repeat ACL which still showed ACL positivity along with increased ESR, CRP.



[Table/Fig-7]: Case 2- a,b)- Large triangular non healing jagged ulcer over lateral malleolus of left foot (before starting steroids).

Hence, pus c/s was repeated and patient was started on Inj. piptaz as *Proteus mirabalis* isolated was sensitive to the same. Patient was started on parenteral steroid, inj.dexamethasone 2 cc I.M., od for two weeks. Doses were gradually tapered and converted into oral steroids for a period of eight weeks. Lesions started healing well [Table/Fig-8a,b], thereby justifying the diagnosis of Lucio Vasculitic ulcers. Patient was advised to come to Hansen OPD for regular follow-up, postdischarge for the purpose of tapering of steroids and to ensure complete healing of vasculitic ulcer.



[Table/Fig-8]: Case 2: a) Poststeroid 3 sittings of PRF; b) Poststeroid 7 sittings of PRF.

Case 3

A 55-year-old male presented to the dermatology OPD with the complaint of ulcer over both lower limbs, upper limbs and ears associated with fever since 15 days. History of dark coloured skin patches which progressed to form multiple ulcers over bilateral upper and lower limbs was present, also patient had total loss of eyebrows. He also had associated glove and stocking type of anaesthesia. Patient didn't have any other co-morbidities.

Dermatological examination revealed bilateral ciliary and superciliary madarosis, saddle nose deformity [Table/Fig-9], extensive ulcerations with necrotic slough, overlying adherent blackish crust over bilateral upper and lower limbs [Table/Fig-10a,b] and necrosis of bilateral ear helices. Resorption of digits over right hand was seen [Table/Fig-11]. Peripheral nerve examination showed bilateral non tender enlargement of ulnar nerves. On investigating Slit skin smear showed 3+, biopsy from the ulcer showed features suggestive of vasculitis [Table/Fig-12]. Special staining of vascular endothelial cell revealed, intracellular *Mycobaterium leprae*, with Fite-Faracco stain [Table/Fig-13] and diffuse macrophage granulomas loaded with Acid Fast Bacilli (AFB) and hence the diagnosis of Lucio Leprosy with Lucio's phenomenon was confirmed. He was started on parenteral antibiotics according to pus C/S (Inj. meropenam 1 gm i.v. TDS,

Inj. cefoperazone sulbactum 1.5 gm i.v. BD). Then MB-MDT with parenteral steroids (Inj. decadron 2 cc im OD for a period of two weeks were started and then gradually tapered as 5 mg/wk and converted into oral steroids. Lesions started healing well then oral steroids were stopped. Patient was advised to attend Hansen OPD for follow-up and tapering of steroids. The details of the patients of Lucio Leprosy are summarised in [Table/Fig-14,15].



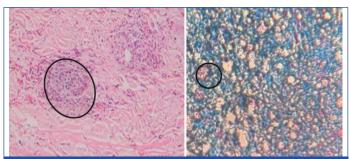
[Table/Fig-9]: Case 3 Total loss of eyebrow, saddle nose deformity, nasal ulcers, diffuse infiltration of skin.



[Table/Fig-10]: Case 3 a) Extensive vesiculonecrotic lesions of both legs; b) Close-up view of ulceronecrotic lesions with eschar posterior aspect left foot.



[Table/Fig-11]: Case 3- Short stumpy banana fingers with ulcero-necrotic lesions



[Table/Fig-12]: Case 3 picture showing vasculitis and occlusion of dermal blood vessel (H&E 40X). [Table/Fig-13]: Case 3 intravascular AFB under special stain-Fite-Faraco staining for tissue section (Carbol Fuchsin stain counter stain with haematoxylin viewed under 100X Oil immersion). (Images from left to right)

Case	Age/Sex	Initial spectrum of Hansen disease	Duration of parent spectrum	Treatment given in our institution	H/o previous treatment (before coming to our institution) and default,	Reactions Yes/no	Co-morbidities
1	58/F	58/F Lepromatous leprosy 1.5 years dapsone) with N		Treated at Tiruvallur General Hospital with MB-MDT with steroids for two months, (In our centre) defaulter for three months	No	No	
2	63/F	Lepromatous leprosy	1 year	MB-MDT	No	No	No
3	55/M	55/M Lepromatous leprosy 3 years		MB-MDT with steroids	No previous treatment history	No	No

Ī	Table/Fig	1-141:	Details of	Lucio	leprosy	patients
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				ACL (Anticardiolipin	Serum (sr). Electrolytes (meq/L)	
Case	Hb (gm%)	ESR (mm/hr)	CRP (mg/L)	antibody) and RF (Rheumatoid Factor)	Sr. Potassium	Sr. Sodium
1	5.9	140	132.5	ACL- Positive RF- Positive	3.4	136
2	10.8	74	57	ACL- Positive RF- Negative	4.3	144
3	11	98	126	ACL- Negative RF- Positive	4	138

[Table/Fig-15]: Investigation profile of patients of Lucio leprosy.

DISCUSSION

The present case series even though carried out with a small sample size of three patients, throws significant light into the dark areas of Lucio Leprosy, which is now being sporadically reported from across the Globe, outside Mexico too. Long standing patients of Lepromatous leprosy, usually downgrade and land up either with Histoid Hansen or Lucio Leprosy, wherein Tenosynovitis, spontaneous skin ulceration, localised lepromatous nodules are the other unusual manifestation of Lepromatous Leprosy [5].

All the three patients responded to daily dose of parenteral steroids (Inj. dexamethasone) along with MB-MDT. Inj. methyl prednisolone, if started after appropriate investigations like Complete blood count, fasting blood sugar, fasting lipid profile, renal and liver function test, serum electrolytes, and adequate monitoring of vitals, could be a wonderful tool in the dermatologist armamentarium to achieve quick control of disease activity. Also, add on take home message from first case of this series is that, in individuals not responding to oral or parenteral potassium supplements alone, the potential advantage of concomitant administration of parenteral magnesium, thereby, exploiting the "Potassium-Magnesium symport" pathway of potassium absorption in the ascending loop of Henley, can be accomplished.

Patient of Lucio leprosy who defaulted in taking MB-MDT, (case 1), posed a huge challenge to the treating Dermatologist as she was recording a low blood pressure of 70/50 mmHg, along with an extremely low Hb of 5 gm%, at the time of hospitalisation. Greatest task in resuscitating patient of Lucio Leprosy is the tight corner of blood dyscrasia and severe form of vasculitis, adding fuel to the fire will be the superadded bacterial infections in a patient of Lucio vasculitic ulcers, wherein they are prone for septicaemia and septic shock. This particular spectrum of Hansen's disease is notorious for its Rheumatological manifestations in the form of arthritis and arthralgia with the fact many a times as these Lucio patients have false positive RF, land up for treatment with Rheumatologist [6]. Anticardiolipin antibodies predispose them to painful vasculitic ulcers, which very much mimics Rheumatoid arthritis and there are cases on the records where Lucio Leprosy patients have been started on tab. methotrexate by Rheumatologist without much benefits. Also the Melancholy look (sleepy look) of these patients will mimic myxoedema, hence a thyroid profile work-up is necessary to rule out hypothyroidism [6].

Characteristic lesions of Calciphylaxis- Black areas of macular pigmentation [Table/Fig-1] occurs prior to the onset of frank punched out vasculitic ulcers which over a period of time enlarge to become large triangular jagged out ulcers that hardly show a tendency to

heal without oral or parenteral steroids. Apart from systemic steroids along with MB-MDT, drugs such as Tumour Necrosis Factor alpha (TNF- α) inhibitors- Inj. etanercept, tab. pentoxifylline have been found to have a crucial role in the treatment of vasculitic ulcers. Tab. pentoxifylline 400 mg twice a day not only increases the self-life of RBCs by increasing membrane pliability but also antagonises the inflammatory cytokine TNF- α [7-9]. Inj. etanercept 50 mg. s.c., also has a promising role to play in the healing of vasculonecrotic ulcers and prevention of further progression of Lucio phenomenon.

Meticulous saline soaks, hydrogen peroxide debridement to remove the necrotic slough to prevent maggots and cutaneous myiasis is of paramount significance. The foot vasculitic ulcers on the dorsal aspect of both feet were recalcitrant to heal even with systemic steroids, which finally healed with Tab. colchicin 0.5 mg OD for three weeks. As these patients are relatively non ambulant for a long time due to vasculitic ulcers and debilitation, in the background of ACL positivity, it becomes mandatory to maintain these patients on prophylactic low dose Aspirin (T. Aspirin 75 mg/day) [10], as guarding against Deep vein thrombosis and hence pulmonary embolism. Many long standing untreated and treatment defaulting patients of lepromatous and Lucio leprosy patients develop involvement of kidneys in the long-term sequel of the disease. Spectrum of renal involvement can be of glomerulonephritis, nephrosclerosis, tubulointerstitial nephritis, granulomas and renal amyloidosis [11]. Microcytic and hypochromic anaemia of erythropoietin deficiency can also occur [12,13].

In individuals not responding to potassium supplements alone, the potential advantage of concomitant administration of parenteral magnesium, thereby, exploiting the "Potassium-Magnesium symport" pathway of potassium absorption in the ascending loop of Henley can be accomplished. As she had, distal tubular injury, that was irreversible in nature, patient warrants life time supplementation of potassium, as persistent hypokalemia, could predispose to cardiac arrhythmia. Urine acidification test is of huge significance in diagnosing the same.

An early diagnosis of Lepromatous leprosy, adequate health education, counselling regarding the potential hazards of defaulting treatment and high-risk of involvement of kidneys in long standing cases of Lepromatous spectrum are to be explained to the patients in detail, during the administration of first blister pack of MB-MDT. Paramount importance of concomitant steroids as per WHO Regime, along with MB-MDT, could be a practical feasible strategy to prevent many a potential irreversible complications of Lucio leprosy. Treating the patients to the point of smear negativity, customising the duration of treatment of individual patients as per bacterial load and immunity of the patient, rather than a fixed schedule of treatment is going to offer a promising strategy to prevent all the sequel of Lucio leprosy. As per recent guidelines of WHO recommendations, multibacillary patients of Hansen's disease are to be kept under surveillance [14] for a period of nine years, from the day they are declared RFT, with a confirmation negative slit skin smear at the end of treatment [15].

Bacterial load, immunity of the patient, ability of *Mycobaterium leprae*, to manage gaining access into vascular endothelial cells, transepidermal elimination of *Mycobaterium leprae*, as documented by Ghorpade AK, secondary bacterial resistance to MB-MDT,

brought about by, inadequate and irregular treatment, are crucial epidemiological factors predisposing a patient of Hansen's disease into Lucio leprosy and hence Lucio phenomenon [16].

CONCLUSION(S)

Distal tubular injury can occur in long standing cases of Lucio/lepromatous leprosy in the absence of glomerular involvement. Malar area lesions in Lucio indicate severity of the disease. Complete treatment of leprosy to the point of smear negativity or adequate treatment of Lepromatous leprosy (As per WHO recommendations 12 months within 18 months) would be a promising strategy to prevent patients of Lepromatous leprosy getting into Lucio leprosy and hence, Lucio phenomenon. Steroids will be the sheet anchor of treatment of Lucio leprosy along with MB-MDT to prevent complications like vasculitis, blood dyscrasias and renal failure.

REFERENCES

- [1] Choon SE, Tey KE. Lucio's phenomenon: A report of three cases seen in Johor, Malaysia. Int J Dermatol [Internet]. 2009;48(9):984-88.
- [2] Ya SNC, Muhamad R, Zakaria R, Ishak A, Abdullah WNHW. Lucio phenomenon: Sequelae of neglected leprosy. Korean J Fam Med. 2021;42(3):245-49. Doi: 10.4082/kjfm.19.0068. Epub 2020 Jan 23. PMID: 31968408; PMCID: PMC8164933.
- [3] Deps P, Guerra P, Nasser S, Simon M. Hemolytic anemia in patients receiving daily dapsone for the treatment of leprosy. Lepr Rev. 2012;83(3):305-07. PMID: 23356031
- [4] WHO expert committee on Leprosy: Eight report(WHO technical report series; no 968). WHO expert committee on leprosy 2010 Geneva, switzerland, editor. www.who.int. Italy: World Health Organization; 2012, Pp. 17-36.
- [5] Das P, Arora S, Singh GK. An unusual case of tenosynovitis in Hansen's disease. Eur J Rheumatol. 2022. Doi: 10.5152/eurjrheum.2022.21125.
- [6] Gao W, Chen Z, Jiang H, Shi Y, Zhang W, Wang H. Diffuse multibacillary leprosy patient with Lucio's phenomenon and positive anticardiolipin antibody misdiagnosed as lupus erythematosus panniculitis in the People's Republic of China. Infect Drug Resist. 2019;12:741-44. Doi: 10.2147/IDR.S202386. PMID: 31114260: PMCID: PMC6497491.

- [7] Cogen AL, Lebas E, De Barros B, Harnisch JP, Faber WR, Lockwood DN, et al. Biologics in leprosy: A systematic review and case report. Am J Trop Med Hyg. 2020;102(5):1131-36. Doi: 10.4269/ajtmh.19-0616. PMID: 32157993; PMCID: PMC7204592.
- [8] Sampaio EP, Moraes MO, Nery JA, Santos AR, Matos HC, Sarno EN. Pentoxifylline decreases in vivo and in vitro tumour necrosis factor-alpha (TNF-alpha) production in lepromatous leprosy patients with erythema nodosum leprosum (ENL). Clin Exp Immunol. 1998;111(2):300-08. Doi: 10.1046/j.1365-2249.1998.00510.x. PMID: 9486396; PMCID: PMC1904921.
- [9] Yuniati R, Firdaus Munita F, Dayana B. Difference of tumor necrosis factor levels in multibacillary leprosy between reversal reactions and non reversal reaction patients. PJMHS. 2018;12(3):1381-83.
- 10] Ruiz-Irastorza G, Crowther M, Branch W, Khamashta MA. Antiphospholipid syndrome. Lancet. 2010;376(9751):1498-509. Doi: 10.1016/S0140-6736(10) 60709-X. Epub 2010 Sep 6. PMID: 20822807.
- [11] Silva Junior GB, Daher Ede F, Pires Neto Rda J, Pereira ED, Meneses GC. Leprosy nephropathy: A review of clinical and histopathological features. Rev Inst Med Trop Sao Paulo. 2015;57(1):15-20. Doi: 10.1590/S0036-46652015000100002. PMID: 25651321; PMCID: PMC4325518.
- [12] Sen R, Yadav SS, Singh U, Sehgal P, Dixit VB. Patterns of erythropoiesis and anaemia in leprosy. Lepr Rev. 1991;62(2):158-70. Doi: 10.5935/0305-7518.19910019. PMID: 1870378.
- [13] Amalia H, Tabri F, Muchtar SV, Petellongi I, Sjahril R, Amin S. Hemoglobin and ferritin serum levels on leprosy patients before Multi Drug Therapy-World Health Organization (MDT-WHO) compared with healthy control group. IJSBAR [Internet]. 2017 Oct. 14 [cited 2022 Dec. 12];36(4):74-82. Available from: https://www.gssrr.org/index.php/JournalOfBasicAndApplied/article/view/8270.
- [14] Hazarika N, Gupta PK, Dhanta A, Singh A, Mohanty A, Gupta P. Renaissance of Hansen's disease in post-elimination era in north india: A retrospective clinicobacteriological study. Cureus. 2021;13(8):e17514. Doi: 10.7759/cureus.17514. PMID: 34603886; PMCID: PMC8476198.
- [15] Barbosa CC, Bezerra GSN, Xavier AT, de Albuquerque M de FPM, do Bonfim CV, de Medeiros ZM, et al. Systematic review of survival analysis in leprosy studies-including the following outcomes: Relapse, impairment of nerve function, reactions and physical disability. International Journal of Environmental Research and Public Health [Internet]. 2022;19(19):12155. Available from: http://dx.doi.org/10.3390/ijerph191912155.
- [16] Ghorpade AK. Transepidermal elimination of Mycobacterium leprae in histoid leprosy: A case report suggesting possible participation of skin in leprosy transmission. Indian J Dermatol Venereol Leprol. 2011;77:59-61.

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