RESIDENT dream

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IADVL Newsletter For The Residents

RESIDENT DREAM

Dermatology Residents Education And Motivation Bulletin

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From the Editor's desk :

"Every day you may make progress. Every step may be fruitful. Yet there will stretch out before you an ever-lengthening, ever ascending, ever-improving path. You know you will never get to the end of the journey. But this, so far from discouraging, only adds to the joy and glory of the climb." - Sir Winston Churchill

The Residream Team presents to you the second issue of this year's newsletter. We have tried to keep the things short and sweet. Dr. Somodyuti Chandra from Kolkata and Dr. Pankaj Das from Pune have discussed an elaborate approach to a case of genital ulcer. This article will surely help the exam going post graduate students. Dr. Isha Narang from New Delhi takes us through a wonderful roller coaster ride of "What is Normal". It is an article which promises to bring a smile on the face of each and every reader. Dr. Komal Agarwal from Dibrugarh has penned down the current scenario of Dermatology which is a misunderstood branch of medicine. Dr. Seujee Das from Gauhati and Dr. Akshi Bansal from Bangalore present to us an interesting debate on the utility of dermoscopy. Dr. Soumya Jagadeesan and Dr. Sarah Elizabeth Dias from Cochin have drafted a beautiful brain tickling quiz followed by a comprehensive discussion. Besides, we also provide the answers to the Dermcross puzzle which was published in the previous issue along with the names of all the residents who had sent correct answers.

I am hereby signing off by wishing all the readers a happy and joyful festive season and request all to stay tuned for a blockbuster third anniversary issue of Residream, to be released in the Dermacon 2017 in the City of Joy, Kolkata.

Happy reading.
Signing off,
Dr. Anupam Das
Chief Editor, Residream

HIGHLIGHT

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APPROACH TO A CASE OF GENITAL ULCER

Genital ulcer is defined as ulcerative, erosive, vesicular or pustular lesion leading to a breach in the continuity of genital mucosa and/or skin.

Genital ulcers may be caused by infectious and non-infectious etiologies :

I. Infectious causes :

A. STI :

- Herpes simplex
- Syphilis
- Chancroid
- Lymphogranulomavenereum
- Granuloma inguinale

B. Non STI :

- Fungal – Candida, deep fungi (rare)
- Viral – Cytomegalovirus, Epstein-Barr virus, Varicella or Herpes Zoster
- Bacterial – Staphylococcus, Streptococcus, Salmonella, Pseudomonas, Mycobacteria
- Parasite – Amoebiasis, Leishmaniasis, Scabies, Phthiriasis

II. Non-infectious causes :

A. Trauma

B. Bullous dermatoses :

- Pemphigus
- Cicatricialpemphigoid

C. Drug reaction :

- Erythema multiforme
- SJS/TEN
- Fixed drug reaction
- Contact dermatitis

D. Inflammatory dermatoses :

- Non specificvulvitis/balanitis
- Erosive Lichen Planus
- Lichen sclerosus et atrophicus
- Behcet's disease
- Reiter's disease
- Plasma cell balanitis
- Pyodermagangrenosum
- Lupus erythematosus
- Crohn's disease
- Vasculitis – Wegener granulomatosis

E. Premalignant or malignant neoplasm :

- Erythroplasia of Querat
- Extramammary Paget's disease
- Bowen's disease
- Squamous cell carcinoma
- Basal cell carcinoma
- Lymphoma
- Leukaemia
- Histiocytosis X

The global incidence of genital ulcer is

estimated to be 20 million cases annually with sexually transmitted infections accounting for about 55% of the ulcers while the rest 45% are due to non-venereal causes.

Careful history taking and meticulous clinical examination are essential for correct diagnosis and management.

History

- Age of patient :
- Children –
  - ➔ Trauma
  - ➔ LSA
  - ➔ Contact dermatitis
  - ➔ Scabies
- Adults –
  - ➔ STI
  - ➔ Other infectious causes (EBV, TB)
  - ➔ Inflammatory dermatoses (Behcet's, Reiter's, Zoon's balanitis)
  - ➔ Pemphigus
  - ➔ Contact dermatitis, LP, LE
- Elderly –
  - ➔ Candidiasis, LSA, Cicatricialpemphigoid
  - ➔ Malignancy
- Sex :
- Male - Zoon's balanitis
- Female - LSA, LE, Extramammary Paget's disease
- Occupation :

STI's are more common in sea men, truck drivers & people with jobs that involve frequent travel.

- Onset and duration of complaint :
- Sudden onset and short duration –
  - ➔ Trauma
  - ➔ Infectious causes other than syphilis
  - ➔ Drug reaction
  - ➔ Contact dermatitis
  - ➔ Behcet's disease
- Insidious onset and protracted course –
  - ➔ Inflammatory dermatoses like Crohn's disease, LE
  - ➔ Erosive LP, LSA
  - ➔ PG
  - ➔ Zoon's balanitis
  - ➔ Pemphigus, cicatricialpemphigoid
  - ➔ Malignancy
- Associated pain :
- Painful –
  - ➔ Trauma
  - ➔ STI and other infections other than syphilis
  - ➔ Pemphigus, drug reaction, LP, Behcet's disease
- Painless –
  - ➔ Syphilis, granuloma inguinale
  - ➔ LSA, LE, papulonecrotictuberculid
  - ➔ Malignancy
- Associated pruritus :

Extreme pruritus is seen in scabies, phthyriasis, LSA

- Associated symptoms :
  - ➔ Retention of urine, meatal stenosis, phimosis LSA, malignancy
  - ➔ Dysuria, dyspareunia
  - ➔ Pain in lower abdomen PID (TB, chlamydia)
- Presence of oral ulceration :
  - ➔ Pemphigus, LE, LP, Behcet's disease
  - ➔ Drug reactions
- History of recurrence :

Present in Herpes, Behcet's disease, FDE, EM/SJS/TEN, pemphigus, papulonecrotictuberculid

- Sexual history :

Detailed sexual history should be enquired in all cases regarding –

- ➔ Sex of the partner.
- ➔ Type of exposure (oral, vaginal, anal).
- ➔ Use of condoms.
- ➔ Relationship to partner/s (spouse, regular non-spouse, casual).

➔ Problems or symptoms in the partner/s.

➔ Date of last sexual intercourse.

➔ Number of partners in the past three months.

➔ Travel to endemic area

➔ Serodiscordant partners

• Assessment of high risk behaviour :

➔ Marital status: married, living together, single, separated, widowed.

➔ Occupation: sex workers (male and female), seamen, workers in the hospitality industry,transport workers, migrant workers, etc.

➔ Unprotected casual sexual encounters (other than with regular partner).

➔ Previous history of STI.

➔ History of injections or blood transfusions.

➔ Substance use: alcohol, drugs (e.g. heroin).

➔ Tattooing.

➔ Partner with symptoms suggestive of STI.

➔ Multiple sexual partners.

• Drug history –

➔ Whether on immunosuppresants

➔ History of taking Anti tubercular drugs in the past

➔ History of recent drug intake preceding the onset of ulcer

Examination

General Survey :

• Nutrition- Poor in AIDS, chronic diseases, in oral mucosal involvement, Crohn's disease

• Fever – HIV infected, Leukemia, Lymphoma, TB, Behcet's disease

• Pallor - Chronic diseases

• Lymph node examination –

Number, location, size, tenderness, presence of bubo, consistency and attachment to underlying structure are to be noted

➔ Painful, firm, tender & nonsuppurative Herpes

➔ Painless, discrete, shotty, non tenderSyphilis

➔ Painful, very tender, suppurative, unilocularChanroid

➔ Tender, suppurative, soft/firm, multilocular LGV

➔ Matted, nontender, firm or rubbery, fixed to underlying structures Malignancy

➔ BubosLGV,Chanroid

➔ Pseudo-bubo-groove sign- Granuloma inguinale

➔ Associated with lymphadenopathy elsewhere- Leukemia, lymphoma

➔ Associated oral lesions may manifest as cervical lymphadenopathy.

Genital examination :

Examination of the ulcer(s) –

• Site :

➔ Glans penis FDR, Pemphigus

➔ Glans penis & prepuce Zoon's balanitis

➔ Primary chancre develops on site of primary exposure

➔ Scrotum & vulva, anywhere on the perianal skin Behcet's disease, aphthi

➔ Prepucial opening in men &vulval orifice in female LSA

• Size :

➔ Small - Herpes, Aphthi, Behcet's disease

➔ Large erosions - Pemphigus, erosive LP, FDR, SJS/TEN

• Number :

➔ Numerous – Herpes, Aphthi, Behcet's Disease, Chancroid

➔ Single – Primary chancre, Zoon's balanitis, LGV

• Edge :

➔ Sharply demarcated – Primary chancre, LSA, Zoon's balanitis, aphthous, LGV

➔ Irregular- Coalesced Herpes lesions,

Chancroid, pemphigus, Erosive LP, SJS/TEN

- Induration :

➔ Present in - primary syphilitic chancre, LSA, malignancy

- Base :

➔ Smooth, non-purulent, covered with serous exudate- primary chancre

➔ Covered with necrotic slough–Chancroid

➔ Beefy granulation tissue bleeds easily on touch- Donovanosis

➔ Sclerosis with epidermal atrophy- LSA

➔ Curdy white material that can be wiped off – Candida

Oral examination :

- Indurated, painless, button like lesion covered with serous exudate, usually single – Primary chancre
- Snail track mucosal lesions- Secondary syphilis
- Lacy white reticulate, erythematous (atrophic), Ulcerative (erosive) lesions – oral LP
- Painful erosions – Pemphigus
- Ulcers erosions almost always affecting the gingiva- Cicatricialpemphigoid
- Painful erosions on erythematous base – SJS/TEN,EM
- Oral aphthi in Behcet's disease
- Lips & oral mucosa involvement over hyperpigmented/erythematous patch- FDR
- Curdy white membrane that can be wiped off – oral candida
- Solitary, painless, large ulcer on the hard palate- SLE

Rectal examination :

- Primary chancre depending upon the primary exposure
- LSA – involves perianal area leading to fissures that causes painful defecation
- Traumatic receptive anal sex

Ophthalmological examination :

Important in SJS/TEN, Behcet's disease, cicatricialpemphigoid

Cutaneous examination :

Thorough cutaneous examination may provide a clue to diagnosis, as :

- Classic cutaneous lesions of LP present as purple, polygonal, pruritic papule and plaque and pterigium of nails.
- Behcet's – Erythema nodosum, pustules
- Cicatricialpemphigoid-occasional dome shaped bullae
- Pemphigus- Multiple flaccid bullae
- Target, targetoid lesions typically in palms & soles – Erythema multiforme
- Macular or atypical targetoid lesions with purpuriccentres that coalesce to form bullae & peels off – SJS/TEN
- Pruritic papular lesions, excoriations, burrows especially in the finger webs – Scabies
- Red patch that soon evolves an iris or target lesion eventually blisters & erodes healing with hyperpigmention-FDR
- Infected genito-crural& pubic eczema- Phthiriasis
- Diffuse hairloss, Raynaud's phenomenon, Photosensitivity, Joint pain in SLE,DLE.
- Swollen lymph nodes, chloromas in a cachectic individual in ulcers associated with leukaemia, lymphoma.

MANAGEMENT OF GENITAL ULCER DISEASE

The management of genital ulcer disease is unique as, the stake-holder is not only the patient, but the spouse and contacts as well. The possibility of more than one co-existing

causes of genital ulcers, the overlap of the clinical signs and symptoms, co-existing immune-suppression with altered clinical picture and unpredictable response to treatment, the stigma, stress and anxiety this syndrome complex brings with it, makes the management of genital ulcer disease tricky. However a step by step and syndromic approach allows physicians to diagnose and treat the genital ulcer diseases more efficiently. Given below is an outline of investigations and management of the commonly encountered causes of genital ulcers.

INVESTIGATIONS

1. Bed-Side Tests:

There are certain bedside tests which can be done like a simple Tzanck smear, which can clinch the diagnosis in case of genital herpes. Samples should be taken from a fresh vesicle, rather than a crusted one, to ensure the yield of a number of virus infected cells. The vesicle should be unroofed with the help of a scalpel blade or the crust removed, and the base scraped with the blunt edge of the blade. The material is transferred to a glass slide, fixed and stained with Giemsa stain. The typical features include characteristic multinucleated syncytial giant cells and acantholytic cells. The cells appear as if they have been inflated ("ballooning degeneration") and sometimes may grow tremendously, to 60-80 µ in diameter. Syncytial giant cells contain multiple nuclei (many with 8 or more) that exhibit nuclear molding, so that the nuclei fit together in a jigsaw puzzle like fashion.

A KOH mount may be done in a case of suspected Candidiasis, which would show the presence of budding yeast cells and pseudo-hyphae.

2. Collection of sample from ulcer for staining and culture:

In principle, it is always better to speak to the microbiologist/ lab-technician, to know and confirm, how a sample is to be collected and transported.

a. For Dark-Field Microscopy:

Clean the lesion only if it is encrusted or obviously contaminated. Use only tap water or physiological saline. Antiseptics or soaps should not be used as they may kill the treponemes. Use minimal amount of liquid for cleaning as it may dilute and reduce the yield of the organisms. Gently abrade the lesion with dry gauze, wipe away any blood-stained exudate and apply gentle pressure until only clear serum exudes. Collect the specimen directly on a cover slip or a clean glass slide by pressing directly onto the lesion. For cervical and vaginal lesions, identify the lesion by speculum examination, clean with saline, abrade with a gauze pad held in suitable forceps. Collect the serous exudates using a bacteriological loop or Pasteur pipette and transfer to a slide. If material is not sufficient, mix with a drop of saline. Seal the edges of the cover slip with petroleum jelly and examine immediately. If negative at first, dark-field examination should be repeated daily for at least three consecutive days. Treponemapallidum appear as brightly illuminated objects against a dark background. They are identified by their typical morphology, size, and movement. It is a 0.25-0.3 m wide and 6-16 m long organism with 8-14 regular, tightly wound, deep spirals. It exhibits quick and abrupt movements. The organism rotates slowly along the longitudinal axis (corkscrew motion) accompanied by bending and twisting in the middle.

b. Gram Staining:

In context of genital ulcers, Gram staining is useful for the diagnosis of chancroid. Specimen is collected from the undermined edge or the base of the ulcer. Wipe the lesion with saline gauze followed by dry gauze to remove the superficial debris and crusts. Roll a sterile swab in one direction beneath the undermined edge of the ulcer. Re-roll the swab in the reverse direction at 180° on a clean glass slide to maintain the arrangement of the bacteria. The swab in transport medium (e.g. Amies or Amies

with charcoal) needs to reach the laboratory within 4 h because H ducreyi will not survive well beyond this time frame.Haemophilusducreyi, a short gram-negative bacillus appears in tissue as clusters of parallel bacilli as chains and resembles “school of fish”.

c. Giemsa/Leishman stain:

For Granuloma inguinale, a small piece of tissue is taken instead of a swab or smear; from the border of a well-defined ulcer using a curette or forceps. The specimen is placed on a clean glass slide and is crushed between two slides (Rajam and Rangiah method). Impression smears from the lower surface of the biopsy specimen may also be used. The specimen is air-dried and stained with Giemsa or Leishman stain. Klebsiellagranulomatis appears as pleomorphic, intracellular (macrophages > neutrophils), gram-negative bacillus surrounded by a well-defined bipolar staining capsule – 'Safety pin' appearance.

Though LGV can be diagnosed by the cytologicdetection of typical intra-cytoplasmic inclusions, it is not pathognomonic for LGV; using Giemsa or iodine stain fails to provide a high percentage of diagnoses. For Giemsa staining, the smear is air dried, fixed with absolute methanol for at least 5 min and dried again. It is then covered with the freshly prepared diluted Giemsa stain for at least 1 h. The slide is rapidly rinsed in 95% ethanol to remove excess dye and then dried and examined microscopically. The inclusions are basophilic and stain pinkish-blue.

d. Culture:

Culture is the gold standard for diagnosis of chancroid. In case of its suspicion, inform the lab prior to sending the sample, so that they have 2 to 3h to prepare the media (many laboratories may need even more time because they may not have media preparation facilities on site).

Candida can be cultured on a Sabouraud's Dextrose agar. LGV as well as Donovanosis, both need specialized tissue culture techniques.

3. Serological studies:

a. Herpes genitalis: Accurate type-specific HSV serologic assays (ELISA and IMMUNOBLOT) based on the HSV-specific glycoprotein G2 (HSV-2) and glycoprotein G1 (HSV-1) are available and are now recommended.

b. Syphilis: A presumptive diagnosis of syphilis requires use of two tests: a non-treponemal test VDRL or RPR and a treponemal test FTA-ABS, TP-PA assay, various enzyme immunoassays [EIAs], chemiluminescence immunoassays, immunoblots, or rapid treponemal assays. Use of only one type of serologic test is insufficient for diagnosis and can result in false-negative results in persons tested during primary syphilis and false-positive results in persons without syphilis. Therefore, persons with a reactive non-treponemal test should always receive a treponemal test to confirm the diagnosis of syphilis. Non-treponemal test antibody titers might correlate with disease activity and are used to follow treatment response. A fourfold change in titer, equivalent to a change of two dilutions (e.g., from 1:16 to 1:4 or from 1:8 to 1:32), is considered necessary to demonstrate a clinically significant difference between two non treponemal test results obtained using the same serologic test. Most patients who have reactive treponemal tests will have reactive tests for the remainder of their lives, regardless of treatment or disease activity.

c. Lymphogranulomavenereum: Specimens drawn too early during primary infection may not contain detectable antibodies. If chlamydial infection is suspected, a second specimen should be drawn 10 to 21 days later and tested in parallel with the original specimen. Serologic tests- complement fixation (CF) or microimmunofluorescence (MIF), are the most common means to diagnose LGV, but all have

limitations since they do not distinguish the serotypes and are insensitive for diagnosis. A positive serologic test for IgG antibodies, in the presence of a compatible clinical syndrome, is considered supportive of a presumptive diagnosis of LGV. It is generally accepted that a titer of >1:64 rules in the diagnosis, while a titer of <1:32 rules it out. Serial titers demonstrating a rise is confirmatory. MIF is more sensitive and specific than CF, but neither test can distinguish recent from past infection. Thus, the results of serologic testing must be correlated with the clinical manifestations.

d. Reactive Arthritis:

Serologic or other evidence of antecedent or concomitant infection-Laboratory tests, such as stool cultures to test for Salmonella, Shigella, Campylobacter, and Yersinia, can sometimes confirm a preceding or concomitant infection with one of the pathogens that classically induce reactive arthritis. However, by the time patients develop arthritis, the pathogens may no longer be retrievable. Urine and genital swab testing can sometimes detect Chlamydial infection using nucleic acid amplification techniques.

4. Rule out other Sexually Transmitted Diseases:

All cases of genital ulcers should be screened for HIV and in addition be investigated to rule out chronic HBV and HCV infection.

5. Other Investigations:

a. Routine hematology and biochemistry work up

b. Acute phase reactants

c. HLA studies for suspected Behcet's disease(HLA-B 51) and Reactive Arthritis(HLA-B27)

d. Ocular examination

e. Imaging of affected joints

f. Synovial fluid aspiration biochemistry and cytology in case of significant joint effusion

g. Pathergy test

h. Patch testing for suspected FDE may be done at a later date, after resolution of lesions

i. Rule out any organ system involvement

j. Biopsy from the ulcer for HPE and IHC, in case a malignancy is suspected.

TREATMENT

1. Herpes genitalis:

Management of genital HSV should address the chronic nature of the disease rather than focusing solely on treatment of acute episodes. Counselling of infected persons and their sex partners is the cornerstone of the therapy. The following points should be discussed when counseling persons with genital HSV infection-natural history, recurrent episodes, risks of sexual transmission, barrier contraception, importance of abstaining when lesions/prodromal symptoms are present, asymptomatic viral shedding, suppressive and episodic therapy, informing current and future sex partners and risk for neonatal HSV infection. Acyclovir 400 mg orally three times a day for 7–10 days or Acyclovir 200 mg orally five times a day for 7–10 days is the recommended regimen for the primary episode, whereas episodic therapy demands Acyclovir to be given at 400 mg orally three times a day for 5 days or Acyclovir 800 mg orally twice a day for 5 days. Suppressive therapy with Acyclovir is indicated in patients who experience frequent recurrences and is given in the doses 400 mg orally twice a day, which can be given for as long as 6 years. For treatment of acyclovir-resistant genital herpes, Foscarnet at the dose of 40–80 mg/kg IV every 8 hours is the preferred anti-viral drug.

2. Syphilis:

Primary syphilis (Chancre) is treated with Benzathine Penicillin G 2.4 million units IM in a

single dose. Persons who have had sexual contact with a person who receives a diagnosis of primary, secondary, or early latent syphilis within 90 days preceding the diagnosis should be treated presumptively for early syphilis, even if serologic test results are negative. Persons who have had sexual contact with a person who receives a diagnosis of primary, secondary, or early latent syphilis >90 days before the diagnosis should be treated presumptively for early syphilis if serologic test results are not immediately available and the opportunity for follow-up is uncertain. Clinical and serologic evaluation should be performed at 6 and 12 months after treatment. Serologic response (i.e.titer) should be compared with the titer at the time of treatment. Failure of non-treponemal test titers to decline fourfold within 6–12 months after therapy for primary or secondary syphilis might be indicative of treatment failure. At a minimum, these persons should receive additional clinical and serologic follow-up and be evaluated for HIV infection. If additional follow-up cannot be ensured, re-treatment is recommended. Because treatment failure might be the result of unrecognized CNS infection, CSF examination can be considered in such situations. For retreatment, weekly injections of benzathine penicillin G 2.4 million units IM for 3 weeks is recommended, unless CSF examination indicates that neurosyphilis is present. In case of penicillin-allergic persons who have primary or secondary syphilis, regimens of Doxycycline 100 mg orally twice daily for 14 days may be given. Ceftriaxone and Azithromycin have been effective for treating primary and secondary syphilis, however, there are no sufficient evidence regarding the cure rates. Careful clinical and serologic follow-up of persons receiving any alternative therapies is essential.

3. Chancroid:

Any one of the following- Azithromycin 1 g orally in a single dose or Ceftriaxone 250 mg IM in a single dose or Ciprofloxacin 500 mg orally twice a day for 3 days may be administered. Patients should be tested for HIV infection at the time chancroid is diagnosed. If the initial test results were negative, a serologic test for syphilis and HIV infection should be performed 3 months after the diagnosis of chancroid. Patients should be re-examined 3–7 days after initiation of therapy. If treatment is successful, ulcers usually improve symptomatically within 3 days and objectively within 7 days after therapy. If no clinical improvement is evident, the clinician must consider whether 1) the diagnosis is correct, 2) the patient is co-infected with another STD, 3) the patient is infected with HIV, 4) the treatment was not used as instructed, or 5) the H. ducreyi strain causing the infection is resistant to the prescribed antimicrobial. The time required for complete healing depends on the size of the ulcer; large ulcers might require >2 weeks. Regardless of whether symptoms of the disease are present, sex partners of patients who have chancroid should be examined and treated if they had sexual contact with the patient during the 10 days preceding the patient's onset of symptoms.

4. Donovanosis:

Azithromycin 1 g orally once per week or 500 mg daily for at least 3 weeks and until all lesions have completely healed, is the first line of therapy. Alternatively, Doxycycline 100 mg orally twice a day for at least 3 weeks or Ciprofloxacin 750 mg orally twice a day, for at least 3 weeks and until all lesions have completely healed. If improvement is not evident within first few days of therapy, addition of another antibiotic, preferably an aminoglycoside to these regimens is an option (Gentamicin 1 mg/kg IV every 8 hours). Persons who have had sexual contact with a patient who has granuloma inguinale within the 60 days before onset of the patient's symptoms should be examined and offered therapy.

5. Lymphogranulomavenereum:

At the time of the initial visit (before

diagnostic tests for chlamydia are available), persons with a clinical syndrome consistent with LGV, including proctocolitis or genital ulcer disease with lymphadenopathy, should be presumptively treated for LGV. Treatment cures infection and prevents ongoing tissue damage, although tissue reaction to the infection can result in scarring. Buboes might require aspiration through intact skin or incision and drainage to prevent the formation of inguinal/ femoral ulcerations. Doxycycline 100 mg orally twice a day for 21 days is the preferred treatment. Azithromycin 1 g orally once weekly for 3 weeks may also be given. Patients should be followed clinically until signs and symptoms resolve. Persons who have had sexual contact with a patient who has LGV within the 60 days before onset of the patient's symptoms should be examined and tested for urethral, cervical, or rectal chlamydial infection depending on anatomic site of exposure. They should be presumptively treated with a chlamydia regimen (Azithromycin 1 g orally single dose or Doxycycline 100 mg orally twice a day for 7 days).

6. Genital Candidiasis:

Mild cases can be managed by topical Azole antifungals, whereas severe and recurrent Candidiasis warrants systemic azole antifungals.

7. Treatment of other causes of genital ulcers:

a. Behcet's Disease: Topical and Systemic steroids, Colchicine, Dapsone, Azathioprine, Methotrexate, Cyclosporine, Thalidomide.

b. Reiter's Syndrome: Rest, NSAIDs, DMARDs (Most commonly Sulfasalazine), Methotrexate.

c. FDE: Topical and/or systemic corticosteroids, depending on severity.

d. Zoon's Balanitis: Advice on hygiene, Topical corticosteroids and calcineurin inhibitors, Ablative lasers, circumcision.

e. Erosive Lichen planus: Topical corticosteroids and calcineurin inhibitors, systemic steroids.

f. Pyodermagangrenosum:Topical and Systemic steroids

g. Crohn's disease:Topical and Systemic steroids, Sulfasalazine, Azathioprine, Methotrexate, Cyclosporine.

h. Vasculitis: Topical and Systemic steroids, Colchicine, Dapsone, Azathioprine, Methotrexate, Cyclosporine.

i. Bullous dermatoses:Topical and Systemic steroids, Dapsone, Cyclophosphamide, Azathioprine, Methotrexate, Cyclosporine.

j. Cutaneous TB: ATT

k. Leishmaniasis: Na stibogluconate, Meglumineantimoniate, PentamidineAminosidine, Interferon, Cryotherapy, Electrosurgery.

l. Pre-malignant and Malignant lesions: 5-FU, Cryotherapy, Photodynamic therapy, Ablative lasers, Circumsicion, Excision, Moh's Micrographic surgery.

- H/O kala-azar in the past, residence in kala-azar endemic area, presence of photosensitivity (for PKDL)
- H/O high risk sexual behaviour and genital ulcer in the past (for secondary syphilis)
- H/O exertional breathlessness (relevant for sarcoidosis,)

History of other systemic involvement –

- H/O decreased lacrimation, inability to close eyes, deep pain with redness and foreign body sensation in both eyes, diminished vision
- H/O testicular pain
- H/O swelling of breast tissue, infertility, decreased libido
- H/O bone pain particularly involving small bones of hands and feet
- H/O hoarseness of voice

History of any possible precipitating event for Type 2 reaction–

- Whether spontaneous or precipitated following-
- H/O intercurrent infection, physical or mental stress, surgery, drug intake

Treatment history –

- H/O treatment with injectables for 21 days (suggestive of kala-azar)
- H/O treatment received for this condition in the form of blister pack and adherence or compliance to the treatment

Past history –

- Similar episode in the past
- H/O tuberculosis, diabetes, hypertension, bronchial asthma (treatment with steroid for Type 2 reaction may flare up these conditions)

Family history –

- H/O family members or close contacts having hypopigmented-hypoaesthetic patch
- H/O similar disease in the family or neighbourhood and whether they treatment received for the same

Q2. What are the relevant clinical examination needed?

General survey

Detailed examination to be done including

♦ Presence of bilateral pitting edema (relevant to lepromatous leprosy)

♦ Palpation of lymph nodes (in relevance to lepromatous leprosy, secondary syphilis, cutaneous T-cell lymphoma)

♦ Temperature will be raised during the period of Type 2 lepra reaction

Cutaneous examination

♦ Multiple erythematous tender nodules with smooth shiny surface present over face, ears, trunk and upper and lower extremities with characteristic sparing of the scalp, axillae and groins.

♦ Numerous small irregular hypopigmented macules with ill-defined margins present over back, buttocks and thighs with tendency to symmentry.

♦ Facial skin thickened and thrown into folds (leonine facies) with nodules over both ears including ear lobes. Broad nose with collapsed nasal bridge.

♦ Both testes are soft and shrunken with reduced testicular sensation

♦ Gynaecomastia (secondary to testicular atrophy)

♦ Hair – Loss of Eyebrows, eyelashes, axillary and pubic hair

Examination of mucosae

♦ Nasal mucosa – to look for crusting, presence of erosion over nasal septum and inferior turbinate

♦ Oral mucosa – to look for lip swelling, nodules and erosions over tongue, palate, uvula, loss of upper incisor teeth

Examination of peripheral nerves

♦ Peripheral nerves are palpated to note thickening, tenderness or nodularity, and compared with the opposite side; though in lepromatous leprosy they may be equally thickened on both sides. Nerves to be examined include:

♦ Supraorbital nerves
♦ Supratrochlear nerves
♦ Infraorbital nerves
♦ Temporal nerves
♦ Great auricular nerves
♦ Supraclavicular nerves
♦ Radial nerves
♦ Ulnar nerves
♦ Median nerves
♦ Radial cutaneous nerves

- Common peroneal
- Anterior tibial nerves
- Posterior tibial nerves
- Sural nerves
- Sensory examination – over the lesions as well as on the area supplied by the nerves
- Motor examination – of muscles of hands, forearm, legs

Examination of eyes

- Presence of peri-orbital skin lesions, frequency of blinking, excess lacrimation, conjunctival congestion is to be looked for.
- Corneal reflex and light reflex examination.

Examination of hands and feet

- Examination of hands and feet Presence of trophic ulcer (over pressure point) and deformities (claw hand, wrist drop, foot drop etc.) are to be looked for. In the present case non-healing, well defined, round/oval ulcers over the ball of great toe was present. The ulcer was surrounded by a rim of callus and floor is covered by slough with scanty sero-purulent discharge.
- Presence of muscular atrophy, loss of digits, obvious deformity are to be noted

Q. What are the atypical variants of lepromatous leprosy?

A. Lepromatous leprosy may have certain unusual presentations, which are as follows –

1. Localised lepromatous leprosy – It is characterised by a single nodule or a localised area of papulo-nodular lesions, having very high bacterial index while the rest of the skin is clinically and microbiologically normal.

2. Histoid leprosy – It is characterised by sudden appearance of dome-shaped, shiny, erythematous papulo-nodules mainly involving posterior and lateral aspect of arms, buttocks, thighs, bony prominences and dorsum of hands. Bacteriological and morphological indices of the lesions are very high (5+ or 6+) while it is negative in the uninvolved skin.

3. Spontaneous skin ulceration – It may occur without the presence of any reaction and may be the presenting feature of severe, long-standing and untreated LL. These mainly occur over the anterior thighs, calf, posterior arms and dorsum of forearms.

4. Lucio leprosy - It is characterised by uniformly diffuse, shiny infiltration of the entire skin of the body, thickened eyelids with madarosis, epistaxis, hoarseness of voice and peripheral edema and numbness of hands and feet. This variant is mostly encountered in Latin America.

Q. How will you treat this case?

A. Treatment consists of 3 parts :

a. Treatment of the disease

b. Treatment of reaction

c. Prevention and treatment of deformities

a. Treatment of the disease

- Standard adult treatment regimen for MB leprosy is:

Rifampicin: 600 mg once a month

Clofazimine: 300 mg once a month, and 50 mg daily

Dapsone: 100 mg daily

Duration: 12 months.

- Standard child treatment regimen for MB leprosy is:

Rifampicin: 450 mg once a month

Clofazimine: 150 mg once a month, and 50 mg every other day

Dapsone: 50 mg daily

Duration: 12 months.

b. Treatment of reaction of Type 2 Lepra reaction- ENL

Reactions require urgent treatment as they can lead to irreversible deformities. Thus, early diagnosis and the timely initiation of anti-inflammatory measures are crucial. MDT should be continued at full dosage without interruption.

Q. What are the late complications in Lepromatous leprosy?

A. I. Complications associated with steroid therapy to manage type 2 reaction:

- Metabolic : hypertension, hyperglycemia, weight gain
- GI : peptic ulcer, bowel perforation
- Osteoporosis, avascular necrosis of femoral head
- Proximal muscle myopathy
- Growth retardation
- Psychosis, depression, mood disorders
- Reactivation of pulmonary TB, herpes zoster
- HPA axis suppression

II. Complication of "delayed nerve fibrosis": Neural deficit continues even after complete cure.

Q. What are the histological finding expected in lepromatous leprosy?

A.

- Epidermal atrophy
- Well formed Grenz zone
- Diffuse granuloma extending upto subcutaneous fat. Granuloma contains profuse foamy macrophages and plasma cells with scanty lymphocytes and epitheloid cells
- Loss of appendages
- Clumps of bacilli (globi)

Q. What are the histological differential diagnosis ?

A.

I. Conditions where grenz zone may be found other than LL:

- BL, BB, BT
- Pseudolymphoma
- B-cell lymphoma
- Granuloma faciae

II. Conditions where foamy macrophages may be found :

- Atypical mycobacterial infection
- Xanthoma
- Necrobiotic xanthogranuloma
- Langerhans cell histiocytosis
- Sebaceous gland tumor

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DERMOSCOPY: BOON OR BANE

Knowledge isn't life changing, application of knowledge is
-Todd Stocker

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As doctors, our most important goal is the provision of best possible care for our patients, and having a correct diagnosis is critical to it. The technique of dermatoscope has provided us with a simple, handheld instrument linking the worlds of macroscopic clinical dermatology with microscopic dermatopathology, enabling us to visualize and evaluate the skin lesions in a way, unimaginable, with naked eye examination.

While dermoscopy was initially used primarily to differentiate cutaneous melanomas from benign nevi, it has now gained traction in the diagnosis and management of a host of inflammatory and infectious conditions ranging from psoriasis to lichen planus, and scabies to tineanigra. Not only have the improvements in diagnostic accuracy afforded by dermoscopy, translated into earlier detection of skin cancer, it has also helped reduce the number of unnecessary biopsies of benign lesions, resulting in lowering of benign to malignant biopsy ratio.

And now, this procedure has become an integral part of a dermatologist's routine diagnostic armamentarium as a trichoscope, onychoscope, inflammascope and an entomodermoscope.

Besides its relevance for diagnostic purposes, this technique has been employed for monitoring skin reactions to treatment and/or treatment response. Recently, in patients with port-wine stains (PWS), the pretreatmentdermoscopic evaluation helped clinicians to estimate and predict the response to laser treatment. Furthermore, application of dermoscopy has been suggested in monitoring the outcome of topical treatments, aiding in early recognition of unwanted side effects. Recognition of telangiectasias for steroid induced atrophy is one amongst the many conditions where dermatoscopy has helped in better management of patients and overall treatment outcomes.

In addition, dermoscopy has provided researchers the insights into natural biology of certain lesions that has enabled better understanding and lead to questioning of the conventional concept of nevogenesis.

The appeal and diffusion of dermoscope is not limited to dermatologists alone. The advantage of dermatoscope has been discovered in urology to study the local tolerability of antiseptic treatments of genitals by applying criteria of reddening, erosions and microbleeding. Recently, in another study by orthopaedicians, the dermoscopic finding of skin on the stump of patients with prosthesis intolerance were compared to patients with prosthesis tolerance, in terms of microvascular changes.

This 'sub-macroscopic' view of skin lesions in a branch of medicine where thorough visual analysis of lesions is imperative for diagnosis has definitely resulted in more confident clinicans. With novel data being increasingly gathered and standardization of guidelines, the dermatoscope is gradually becoming equivalent to the dermatologist's stethoscope, acquiring an irreplaceable role in clinical diagnosis

"The greatest enemy of knowledge is not ignorance; it is the illusion of knowledge"
-- Stephen Hawking

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Dermoscopyor skin surface microscopy or epiluminescence microscopy involves an evaluation of the skin surface for visualizing subtle clinical patterns of skin lesions and subsurface skin structures that are not normally visible to the unaided eye.

It provides a valuable aid in diagnosing pigmented skin lesions by studying the pigment network and vascular patterns, but, because of the complexity involved, this methodology is reserved for experienced clinicians.Dermoscopy is an evolving science in the field of dermatology and at present in its nascent stage. Dermatologists are not fully acquainted with the instrument and it is not yet widely used.

Mainly used in white skinned people for the study of melanocytic nevi and melanoma, it certainly has its limitations.Though it serves as an ancillary aid in the diagnosis of melanoma; it is not confirmatory. Histopathological examination is required for confirmation and staging based on which treatment is started accordingly. Dermoscopy therefore, can never replace dermatopathology.

Moreover, dermoscopic diagnosis for melanoma is difficult in cases lacking specific features for melanoma. Some authors have demonstrated the limitations of dermoscopy in the detection of early melanomas that present with an uncharacteristic dermoscopic appearance. Some melanomas, the so-called "featureless melanomas," may lack specificdermoscopic features and may even appear as benign melanocytic lesions or as atypical nevi, so that the diagnosis is impossible to make on dermoscopic grounds alone. Thus, this technique cannot differentiate early, featureless melanoma from dysplastic nevi.

Dermoscopes are not commonly used in developing countries like India because they are expensive and are not readily available.

Proper training and expertise is required for identifying various dermoscopic patterns in different pigmentary and inflammatory skin disorders. There is no universal method of teaching yet and risk of observer variation is also high.

The potential of dermoscopy for study of inflammatory and pigmentarydermatoses is waiting to be tapped.Predictive powers of patterns seen in dermoscopy in different disorders of the skin, hair and nail are yet to be quantified. Also, clinicians should be properly acquainted to identifying these patterns. No standardized criteria has been formulated that can be applied universally in routine clinical practice, resulting in a questionable diagnostic accuracy.

Thereby, there is definitely a need to make the learning of dermoscopy easier and to establish a global and international method of teaching.

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What's Normal?

'Normal' is something that is engrained in everyone who enters this enigmatic field of medicine.

'Normal' is the background music that syncs itself with the harmony of our daily chores and our role as a physician. Right from the day one we are introduced to normal anatomy, normal physiology and normal histology to be gradually taken over by concepts of pathology. From there on, that very moment our lives never become normal.

The pendulum swings each side to return to normal position and so does our lives. Long hours of study and duties become normal. Skipping meals, family obligations become normal. Failures become normal too. We adapt and our definitions of normal change.

This reflects on our attitude towards patients as well. We start imposing this ideology on them as well. From patients on steroids developing moon facies, stria and weight gain after recovering from the wrath of pemphigus, the post inflammatory hyperpigmentation after Stevens-Johnsons Syndrome, the polka-dots after vitiligo surgeries, normal scaling after retinoid application to normal hairfall or normal acne.

Do we take patients casually and expect them to understand that this is normal or are we impatient to explain the patient that this is a side effect and probably a disease in itself that they will need treatment for. Too complacent to explain that they may not be normal anytime soon or maybe ever.

We try to align them in our ever changing 'dynamic concepts of normalcy'. It's probably our callousness or helplessness that makes us assume that the patient will accept what's normal by our definitions.

But since medicine is ever changing and progressing due to demands of the patients than anything else. Our patients refuse to accept us and our concepts, no matter how patronizing our species is. Pushing us to bring this normal to 'more normal'. Scientists, physicians , academicians work day and night to reduce side effects of the therapy and increase clinical efficacy and bridge this gap.

This urge for being normal, knowing normal and realization when it's not, inspires us to turn nothing into something. This urge is everything that drives us to 'be better'.

So how normal are you? And how normal are you willing to be?

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DERMATOLOGY~ THE MISUNDERSTOOD BRANCH OF MEDICINE.

Dermatology is one specialization in medical field that has been highly misunderstood not only by laymen but also sometimes by medical students.

Thats partly because during the M.B.B.S. period the exposure to dermatology is limited, sometimes just in the form of 'compulsory' rotational ward classes(where most students are disinterested) and also because of time constraints and given the other major humongous subjects, no final year student really gets a chance to explore dermatology.

Coming from a non-medical background deciding to specialize in dermatology over branches like medicine, surgery or obgy is never easy. People frown and have this shocked and bewildered expression swept over their face. Its not uncommon to hear them say "why do you want to waste your degree being a beautician?"

Because if you aren't a surgeon or internal medicine practicioner, you aren't a doctor. The other side of dermatology that deals with all kinds of immunobullous disease and life threatening emergencies like ten and sjs is only best known to us.

Moreover even now in the 21st century there is so much taboo associated with leprology and venerology as branches. Most of the times these patients do not know where to go(again because of the lack of awareness and the preconceived notions about dermatology being just about cosmetics) they keep circling around the medicine departments accounting to increased morbidity and disease prevalence.

Nowadays in the modern technological era dermatology has been equated to "Whatsapp Medicine", yes thats what some like to call it. Friends, relatives, acquantainces everybody would want you to diagnose their disease with just a not-so-clear whatsapp picture (because if you are a good doctor, a picture should be enough).

People, Its science, not magic!

Even amongst the medicos, dermatology is mostly equated to easy lifestyle and easy money. Thats a generalization which isnt all inclusive for all dermatologists in practice. Though its mostly out patient work, with less number of emergencies and a better family life but there is much more hardwork than what meets the eyes. There is much more to dermatology than just acne and eczema and

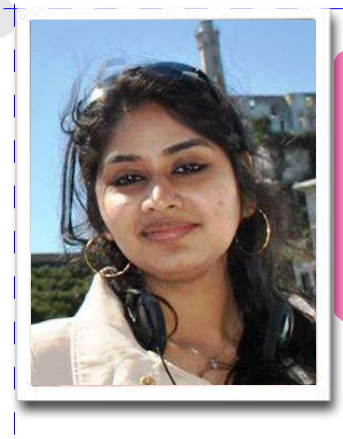
everything pretty. Dealing with the high chronicity of skin disease and other autoimmune diseases, bullous disorders, collagen vascular disease is not always such a pretty sight. Even on an average day a dermatology opd has much more flow of patients than most other specialities.

Inspite of being one of the most competitive residency programmes with limited number of seats, taking up dermatology over other branches is still frowned upon. Part of this problem likely stems from our poor ability to convey to medical students the breadth and importance of dermatological disease and how deeply can it affect the patients physical and mental health.

The general scenario of dermatology in the indian society needs to improve, and change should begin from us, the medicos.

A better exposure to the subject during undergraduate days and spreading awareness about more serious skin diseases amongst the general population is the need of the hour.

And someday dermatology will be accepted as much more than 'just acne, eczema and everything pretty'!



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SLOWLY ENLARGING RING-SHAPED LESION ON THE CHEST

A 48 year old man was seen in the dermatology clinic with a gradually enlarging reddish ring like lesion on the right side of the chest of 2 months duration. He had no complaints other than a low grade fever and malaise at the time of onset of the lesion. The patient revealed a history of a tick bite in a forest area, 2 weeks preceding the onset of lesions. Cutaneous examination revealed a single erythematous annular plaque on the right lateral thoracic area with central lichenification and peripheral erythema, giving a targetoid appearance (Figure 1).

Bacterial and fungal cultures from the lesion did not yield any growth. Mantoux was indeterminate (10mm). Histopathologic examination from the margin of the lesion showed superficial and deep perivascular, perineural and periappendageal cuffing by lymphocytes, histiocytes and a few eosinophils (Figure 2a,b).

PROBABLE DIAGNOSIS?

Ans: Erythema chronicum migrans

DISCUSSION

Erythema chronicum migrans/ Erythema migrans (EM), which is the early cutaneous form of Lyme disease, presents as a gradually enlarging gyrate erythema at the site of a tick bite. In majority of the patients, the lesion appears within 1-3 weeks of bite, though the history may not be forthcoming in all. The morphology of the lesion is characteristic, either with expanding erythema (> 5cm) or with central clearing and the tick bite mark at the exact center, giving a bull's eye or target like appearance, as in our case.[1]

Although the disease derives its name from Lyme, Connecticut in USA, it is a global disease with increasing number of cases reported in India. Lyme disease is caused by the spirochaete Borrelia burgdorferi, primarily by the strains B. burgdorferi sensu stricto, afzelii and garinii (the latter two more common in Asia). In Asia, the most common vector is the Ixodes persulcatus tick and one case in South India involving the Haemophysalis species. Common hosts implicated in transmission of vectors include deer, rodents, sheep, cattle and birds.[2]

The cutaneous manifestations of Lyme disease is classically described in three stages:

1. Acute localized stage
2. Disseminated stage
3. Late chronic stage

The first localized stage is characterized by the pathognomic erythema chronicum migrans as described above, and is seen in 90% of patients.

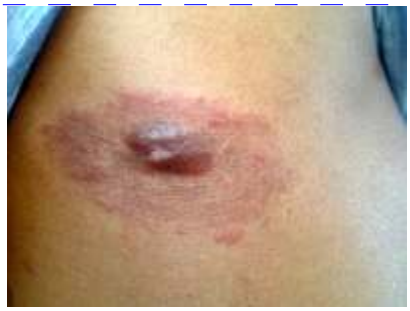

Figure 1: Reddish targetoid lesion on the lateral chest with central lichenification and peripheral erythema.

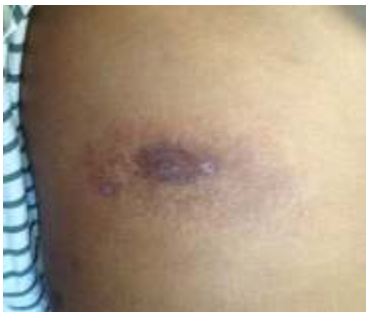

Figure 3: The resolving lesion after 2 weeks of treatment with Doxycycline.

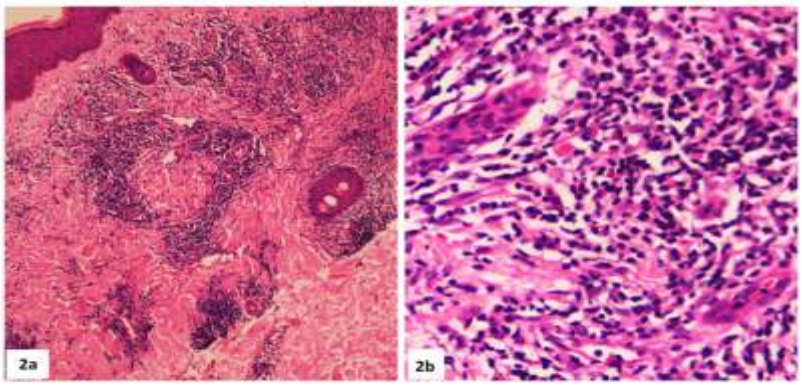

Figure 2a, b: Histopathology showing the superficial and deep perivascular, perineural and periappendageal cuffing by lymphocytes and histiocytes with a few eosinophils. 2a- magnification 20X. 2b- Magnification 400X. H &E staining.

It is usually associated with constitutional symptoms and lymphadenopathy. Aside from the classical annular lesions, atypical variations include vesicles, papules and purpura. Even if left untreated, the lesions resolve spontaneously after few months.[3,4]

Borrelial lymphocytoma is a rare localized lymphoreticular proliferation, presenting as small (5mm) erythematous papules or nodules seen over earlobes or nipple about 30-45 days after infection. The patient may not give a history of tick bite or preceding erythema migrans lesion. Biopsy reveals dense lymphocytic infiltrate with nodules and germinal centers in the dermis and subcutis, and demonstration of the organism on Warthin-Starry stain.[3]

The second disseminated stage (seen in 17-57% of patients) develops weeks to months after the initial infection and is characterised by multiple, smaller, annular lesions in polycyclic pattern over sites distant from the tick bite (signifying dissemination of the infection).[3] Systemic features in this stage include:

- Migratory arthritis, joint effusions and deformities, myalgia
- Cardiac involvement: myocarditis, pericarditis, conduction blocks
- Neurological involvement: cranial nerve palsies, peripheral neuropathy, meningitis, encephalitis[4]

The late, chronic stage of Lyme disease is characterized by Acrodermatitis chronica atrophicans (ACA). It is a chronic T-cell mediated immune reaction that can occur years after the initial infection, and most commonly seen in the elderly. Lesions usually occur in elbows, knees, and distal extremities. There is an initial inflammatory stage, with small papules or nodules that progress to erythematous plaques. ACA differs from the earlier stages in that it does not heal spontaneously. The lesion progresses to an atrophic phase, with central athropy over the plaque, induration and ulceration. Squamous

cell carcinoma and sarcoma can arise from the lesions. Systemic complications include arthritis, neurological and neuropsychiatric symptoms and ocular complaints. [3,4]

Other less common cutaneous manifestations or associations of Lyme disease include:

- Morphea
- Lichen sclerosus et atrophicus,
- Cutaneous B-cell lymphoma,
- Eosinophilic fasciitis
- granuloma annulare
- erythema multiforme,
- Urticaria/urticarial vasculitis
- Gianotti-Crosti syndrome and panniculitis[3]

Because of the limitations of laboratory testing for Lyme disease, the diagnosis is primarily based on clinical findings. The classical histopathologic findings of erythema migrans include a superficial and deep perivascular lymphocytic infiltrate in which plasma cells are identified at the periphery of the lesion and eosinophils in the center, in an angiocentric, interstitial or perivascular pattern. But wide variations have been reported including an absence of plasma cells and nonspecific spongiotic and interface changes. Warthin-Starry stain is used to demonstrate the spirochaetes. Culture of the biopsy specimen taken from the ECM lesions also yield specific results.5 Serologic tests are not required for diagnosis in a patient who presents with erythema migrans like rash from an endemic area, but a 2-step serological testing is recommended in other cases.6 Our patient had tested positive for Ig G Lyme antibody by Enzyme immunoassay, but immunoblot could not be done due to technical and financial constraints. PCR testing is more sensitive and specific that serology and cultures, but should be reserved for atypical presentations.[5]

The drug of choice is generally Doxycycline (100 mg twice per day) for 14 days for the treatment of adult patients with early localized or early disseminated Lyme disease associated with erythema migrans, following which there is an immediate response, as was seen in our patient (Figure 3). Treatment for other stages or complications of Lyme disease is given in Table 2. Oral Doxycycline 4 mg/ kg per day BD (maximum of 100 mg per dose) can be used in children above 8 years. Pregnant and lactating women should be treated with recommended medication for their stage of disease, but doxycycline is contraindicated. [7]

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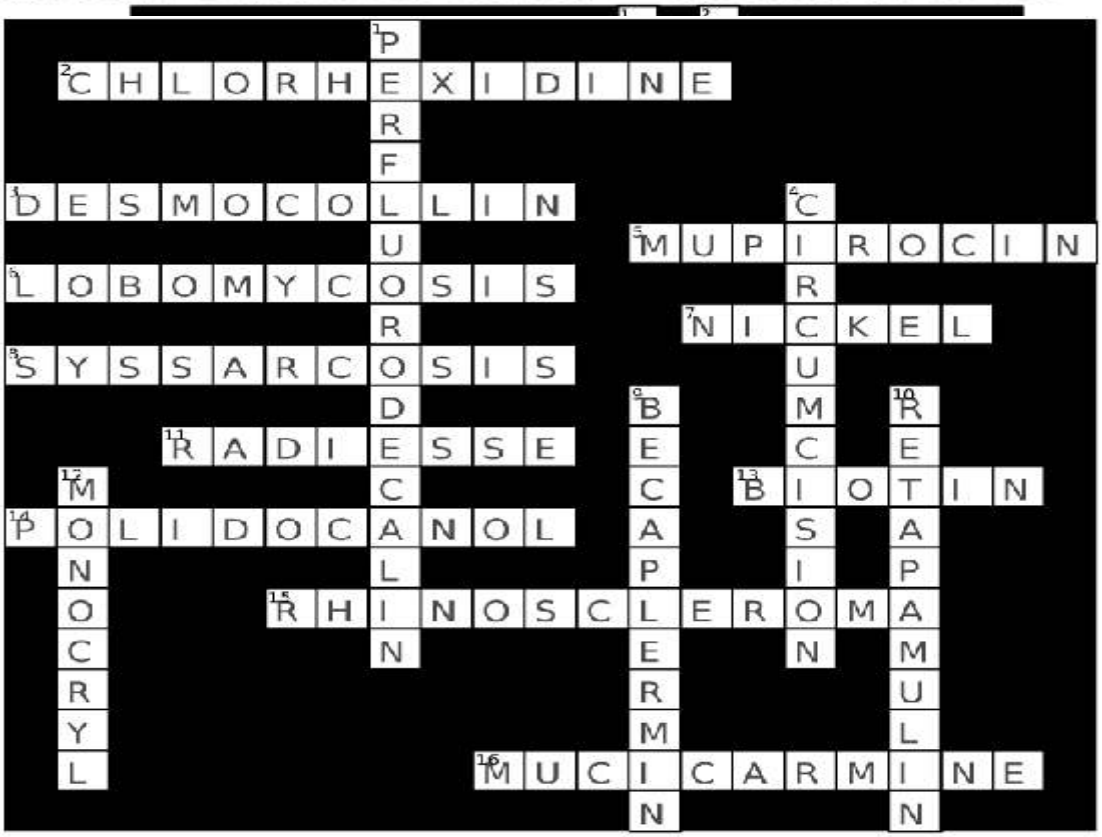
DERMCROSS

Major Pankaj Das, Col Rajesh Verma

Residream DermCross Series. Crossword Puzzle 1



Across

6 Antibiotic derived from Pseudomonas fluorescence
8 This filler was FDA approved in 2015, for volume-loss over dorsa of hands
10 A fungal infection common to humans and bottle nosed dolphins, endemic in South and Central America
12 Biguanide agent used for hand scrub
13 Characteristic "Shadow" or "Ghost cells" in histopathology are a characteristic feature of
14 Recombinant human platelet-derived growth factor used in diabetic foot ulcers
16 Post-Fumigation, the Formaldehyde vapour is neutralized with this agent
17 Derived from brown sugar, this peeling agent is preferred for peri-orbital hypermelanosis

Down

2 This topical agent is used in R-0 technique for Tattoo removal
3 It's mutation causes Sub-Corneal Pustular Dermatosis
5 Initially developed as an anaesthetic, later used as a sclerosant
6 Polyglecaprone-25 is popularly known as
7 Selective PDE-4 inhibitor, only oral biologic FDA approved for moderate to severe plaque psoriasis
9 Mikulicz Cells are seen in this disease
11 FDA approved in 2007, a pleuromutilin class of topical antibiotic derived from edible mushroom Clitopilus scyphoides
15 Vitamin 'H'

CORRECT ENTRIES :

1. Dr. Ayush Bindal, Burdwan Medical College And Hospital
2. Dr. Shreya Dass, Dr. R.N. Cooper Municipal General Hospital, Mumbai.
3. Dr. Kenit Ardeshna, MGM Medical College and Hospital, Navi Mumbai
4. Dr. Praneet Awake, Care Hospital Hyderabad
5. Dr. Anup Kumar Tiwary, RIMS, Jharkhand