Introduction

Antibiotics are chemicals derived from microorganisms that have the capacity, in dilute solutions, to kill other microorganisms (bacteria, virus, fungi, and parasite) or inhibit their growth. In this study, antibiotics refer to collective term for antibacterial, antiviral, and antiparasitic agents. In routine clinical practice, antibiotics are chiefly used to eliminate various pathogens (bacteria, viruses, and parasites). Many antibiotics were later found to have anti-inflammatory properties apart from their antimicrobial action. We have discussed anti-inflammatory and immunomodulatory effects of various antibacterial and antiparasitic drugs. Antiviral and antifungal drugs are seldom used for their anti-inflammatory properties.

Antibacterial Agents

Clindamycin

Clindamycin is a synthetic derivative of lincomycin and isolated from the Streptomyces species. The drug has broad-spectrum antibacterial action by binding irreversibly to 50S subunit of bacterial ribosome and thereby inhibiting bacterial protein synthesis. In dermatology, clindamycin is being used for several indications for its both antibacterial and anti-inflammatory properties [Table 1].

Clofazimine

Clofazimine is a iminophenazine dye known for its antimycobacterial properties. Its absorption is increased with food. Because of slow elimination, the drug has long half-life of approximately 70 days. Metabolism of the drug occurs in liver and elimination occurs through sebum, sputum, tears, sweat, and urine. However, it also possesses good anti-inflammatory actions and is used in many dermatologic diseases for the same [Table 2].
Dapsone

Dapsone (4,4'-diaminodiphenylsulfone) is an aniline derivative belonging to the group of synthetic sulfones. Dapsone is absorbed rapidly and nearly completely from the gastrointestinal tract. Peak plasma concentration is reached within 2–8 h after administration. The mean half-life of elimination is about 20–30 h. It is metabolized in liver by two distinct routes, N-acetylation and N-hydroxylation. It has dual functions of both antimicrobial/antiprotozoal effects and anti-inflammatory features similar to nonsteroidal anti-inflammatory drugs. Dapsone has been used as a treatment option in various dermatological conditions because of its anti-inflammatory effects [Table 3].

Macrolides

Macrolides contain a macrocyclic lactone ring structure. These are of actinomycetes or semisynthetic derivatives of same bacteria. They are bacteriostatic antibacterial agents which bind irreversibly to the large (50S) ribosomal subunit of bacteria, thereby inhibiting RNA-dependent protein synthesis. However, there have been many dermatological uses of macrolides for their immunomodulatory action. Azithromycin (A), roxithromycin (R), erythromycin (E), and clarithromycin (C) are commonly used in dermatology practice for their immunomodulatory and anti-inflammatory potential [Table 4].

Metronidazole

Metronidazole is a synthetic nitroimidazole antibacterial drug. It acts by DNA disruption and nucleic acid synthesis inhibition. It acts against anaerobic bacteria and protozoa. However, it has many actions other than its antibacterial action for which it is being used in different dermatological diseases [Table 5].

Rifampicin

Rifampicin (R) is a semisynthetic derivative of rifamycin B, an antimicrobial agent produced by *Streptomyces mediterranei*. It is a broad-spectrum antimicrobial and inhibits the growth of most Gram-positive bacteria, as

| Disease/condition | Mechanism of action for anti-inflammatory property | Dose |
|------------------|-----------------------------------------------|-----|
| Acne             | Modulates cytokine production in LPS-stimulated macrophages Decreases TNF-α and IL-1β concentrations and increases serum IL-6 concentrations | Topical clindamycin 1% BD |
| Folliculitis decalvans | Suppresses the complement-derived chemotaxis of polymorphonuclear leukocytes *in vitro*, thereby reducing the potential for inflammation | Oral clindamycin 300 mg BD plus rifampicin 300 BD for 10 weeks |
| Fox-Fordyce disease | Suppresses the complement-derived chemotaxis of polymorphonuclear leukocytes *in vitro*, thereby reducing the potential for inflammation | Topical clindamycin 1% BD |
| Hidradenitis suppurativa | Inhibits complement-derived chemotaxis of polymorphonuclear leukocytes *in vitro* and reduces inflammation Immunomodulatory-clindamycin might enhance the uptake of microorganisms by the phagocytic cells of the host | Clindamycin 300 mg BD PO+ Rifampicin 300 mg BD PO for 10 weeks |
| Rosacea | Modulates cytokine production in LPS-stimulated macrophages Decreases TNF-α and IL-1β concentrations and increases serum IL-6 concentrations | Topical clindamycin 1% 5% benzoyl peroxide/1% clindamycin gel |

PO: Peroral, IL: Interleukin, TNF-α: Tumor necrosis factor-alpha, LPS: Lipopolysaccharide, BD: Twice a day

| Disease/condition | Mechanism of action for anti-inflammatory property | Dose |
|------------------|-----------------------------------------------|-----|
| Type 2 lepra reaction | Decrease in neutrophil mobility with consequent decreased influx of PMN Stimulating synthesis of PGE 2 by PMN monocytes and macrophages | Up to 300 mg daily in equal doses PO |
| Granulomatous cheilitis | Alters the function of monocytes and neutrophils (inhibits PMN motility and lymphocyte transformation) | 100-200 mg PO |
| Granuloma faciale | Inhibits lymphocyte transformation | 100 mg BID to TID |
| Lupus miliaris disseminatus faciei | Antigranulomatous effects | 100 mg 3 times/week PO |
| Pyoderma gangrenosum | Alters the function of monocytes and neutrophils (inhibits PMN motility and lymphocyte transformation) | 50-300 mg PO |

PMN: Polymorphonuclear neutrophils, PO: Peroral, PGE: Prostaglandin E
well as many Gram-negative microorganisms. However, it has other properties besides antimicrobial action for which it has been used in various dermatological conditions [Table 6].

**Tetracyclines**

The tetracyclines are broad-spectrum antibiotics and comprise four main drugs (tetracycline [T], doxycycline [D], minocycline [M], and lymecycline [L]). Tetracycline group of antibacterial agents are indicated in a wide range of infections including *Treponema pallidum* (syphilis), *Borrelia burgdorferi*, *Borrelia afzelii*, *Borrelia garinii* (Lyme disease), *Coxiella burnetii* (Q fever), *Rickettsia rickettsii* (Rocky Mountain spotted fever), and *Yersinia pestis* (Plague). Their antibiotic effect is primarily exerted by binding to the 3OS subunit of bacterial ribosomes, thereby halting protein synthesis. However, many tetracyclines have in addition anti-inflammatory properties. Table 7 discusses the role of tetracyclines chiefly for their anti-inflammatory properties.

**Antimalarials**

The parent molecule for the antimalarials is quinine. Among antimalarials, chloroquine (CQ) and hydroxychloroquine (HCQ) are used in various dermatological disorders. Both CQ and HCQ are alkylated 4-aminoquinolines. HCQ is a derivative of CQ and is nearly completely absorbed within 2–4 h of an oral dose and metabolized in liver by dealkylation. The drugs accumulate in thrombocytes, granulocytes, and erythrocytes; hence, their concentration in whole blood is 3–10 times higher than that of plasma. CQ has high affinity for melanin and gets accumulated in the eyes and the skin where the concentration is 100–200 times higher than that of plasma; in the epidermis, it is 3–7 times higher than that of the dermis. The maximum daily dosage is 3.5–4 mg/kg of body weight for CQ and 6–6.5 mg/kg body weight for HCQ. Various indications for antimalarials drug are shown in Table 8.
Table 4: Indications of macrolides in dermatological diseases for their anti-inflammatory and immunomodulatory properties

| Disease/condition                  | Drug | Mechanism of action for anti-inflammatory property                                                                 | Dose                                      |
|-----------------------------------|------|------------------------------------------------------------------------------------------------------------------|-------------------------------------------|
| Acne                              | A, E | Inhibits pro-inflammatory cytokines[52-54] Inhibits IL-8, neutrophil chemotaxis[49-51]                           | 500 mg thrice weekly consecutive days for 6 months |
| Adult onset Still's disease[55,56]| C    | Anti-inflammatory and immunomodulatory[49]                                                                   | 500 mg BD                                 |
| Bullous pemphigoid[57-59]         | E    | Anti-inflammatory and immunomodulatory[49]                                                                   | 1000-3000 mg/day                          |
| Confluent and reticulated papillomatosis[60] | A, R, C, E | Inhibits cytokines, such as TNF-α and IL-1α induced by staphylococcal enterotoxin B, which modulate epidermal keratinization, [55] inhibits lymphocytic activity, [61] immunomodulation of keratinocytes[62] | A - 500 mg/day R - 300 mg/day C - 500 mg/day E - 1000 mg/day |
| Gingival hyperplasia              | A    | Block cyclosporine A-induced cell proliferation and Type 1 collagen synthesis, [63-66] activates MMP-2 level[64-65] | Initial dose of 500 mg, followed by a daily dose of 250 mg for 4 days[64] |
| Granulomatous cheilitis            | R    | Inhibits inflammatory cytokines                                                                             | 150 mg/day                                |
| Immune thrombocytopenic purpura[67,68] | E, C | Immunomodulatory-eradication of bacteria or by modulation of the immune system involving the mucosa on which commensal bacteria reside[69] | E - 600 mg/day C - 500 mg/day             |
| Lupus miliaris disseminatus faciei[70] | R    | Anti-inflammatory activity,[63] inhibition of expression of vascular endothelial growth factor[71]           | 300 mg daily                              |
| Perioral dermatitis[72]           | A, E, C | Inhibits pro-inflammatory cytokines[52-54] | 500 mg OD for 7 days |
| Pityriasis rosea[73-75]           | A, E | Inhibits pro-inflammatory cytokines[52-54] Inhibits lymphocytic activity[64]                                  | E - 250 mg/400 mg QID                      |
| Pityriasis lichenoides[74,77]     | A, E | Inhibits pro-inflammatory cytokines[70]                                                                       | A - 500 mg on day 1 and 250 mg on days 2 through 5, to be taken on the 1st and 3rd weeks of the month |
| Psoriasis[78,79]                  | A, R, E | Inhibits the production of pro-inflammatory cytokines, such as IL-6, IL-8, and (TNF)-α perhaps by suppressing the transcription factors NF-κB or activator protein-1, and reduce neutrophil activity, [80] suppresses immunological events in interferon gamma-treated keratinocytes, including expression of MHC Class II, secretion of IL-1 alpha, and superantigen presenting ability[81,82] | R - 150 mg orally twice daily for 1-7 weeks A - 500 mg daily dose for 4 days with a gap of 10 days for 48 weeks E - 1000 mg/day for 4 weeks |
| Rosacea[83,84]                    | A    | Inhibits pro-inflammatory cytokines IL-1, -6, -8, and -10, GMCSF TNF-α, and LT-B4[40,46,65-87]               | 500 mg on 3 successive days for 4 weeks    |
| SAPHO syndrome[88-91]             | A    | Inhibits pro-inflammatory cytokines                                                                         | 500 mg on 6 successive days, followed by 500 mg twice a week[88] |

IL: Interleukin, TNF-α: Tumor necrosis factor-alpha, MMP: Matrix metalloproteinase, GMCSF: Granulocyte macrophage colony stimulating factor, LT: Leukotriene, SAPHO: Synovitis, acne, pustulosis, hyperostosis, and osteitis, OD: Once a day, BD: Twice a day, MHC: Major histocompatibility complex, A: Azithromycin, R: Roxithromycin, E: Erythromycin, C: Clarithromycin, NF: Nuclear factor

**Levamisole**

Levamisole is an anthelmintic agent with a wide range of immunomodulatory actions. It belongs to the class of imidazothiazolo derivative. It is water-soluble and gets rapidly absorbed from the gastrointestinal tract with peak blood levels achieved after 1.5–4 h. Metabolism of the drugs occurs mainly in liver and the plasma half-life is 16 h. Due to immunomodulatory properties, it has been widely used in various dermatological disorders. Usual dose of the drug is 150 mg/day for 2–4 days each week [Table 9].

**Side effects**

All the above-discussed drugs have variety of side effects in the therapeutic dose range. The treating skin physician must be aware of commonly encountered side effects which can enable him or her to rationalize the treatment protocol and manage the side effects with due care [Table 10].

**Conclusions**

The study aims to highlight the role of various antibiotic drugs in the management of noninfectious diseases of
### Table 5: Indications of metronidazole for its anti-inflammatory properties

| Disease/condition                  | Mechanism of action for anti-inflammatory property                                                                 | Dose                                           |
|------------------------------------|---------------------------------------------------------------------------------------------------------------------|------------------------------------------------|
| Acne                               | Anti-inflammatory\(^{[92,93]}\)                                                                                      | Topical - 0.75% gel or cream, 1% gel, 2% gel   |
|                                   | Immunosuppressive\(^{[89,90]}\)                                                                                     |                                                 |
|                                   | Antipruritic, inhibition of free radical generation by neutrophils\(^{[94]}\)                                      |                                                 |
| Cutaneous metastatic Crohn’s disease\(^{[95]}\)                  | Antibacterial action and possible anti-inflammatory                                                               | 20 mg/kg                                       |
| Periorificial dermatitis\(^{[96-99]}\)                        | Antipruritic, inhibition of free radical generation by neutrophils                                                | Oral - 250 mg BD                               |
| Rosacea\(^{[100]}\)                                                                | Anti-inflammatory mediated through inhibition of release of reactive oxygen species from neutrophils\(^{[101,102]}\) | Topical - 0.75% gel or 1% cream               |
| Seborrheic dermatitis               | Anti-inflammatory\(^{[103-117]}\)                                                                                    | Topical - 1% gel or 0.75% gel                  |
|                                   | Inhibition of free radical generation and oxidative tissue damage                                                  |                                                 |

BD: Twice a day

### Table 6: Indications of rifampicin for its anti-inflammatory and immunomodulatory properties

| Disease/condition                  | Mechanism of action for anti-inflammatory property                                                                 | Dose                                           |
|------------------------------------|---------------------------------------------------------------------------------------------------------------------|------------------------------------------------|
| Hidradenitis suppurativa\(^{[108]}\) | Immunomodulatory effects through its capacity to alter the secretion of cytokines by human monocytes                  | Clindamycin 300 mg BD PO+ Rifaxemicin 300 mg BD PO for 10 weeks |
| Pruritus due to cholestasis\(^{[109]}\)  | Induces Phase I, II, and III biotransformation enzymes and transporters such as CYP3A4, UGT1A1, SULTA1, and MRP2\(^{[110-112]}\), Enhances the metabolism of bilirubin and its breakdown products | 300 mg/day                                      |
| Psoriasis\(^{[113]}\)                                             | Immunosuppressive properties (both humoral and cellular immunity \textit{in vivo} and \textit{in vitro})\(^{[114-116]}\) | 300 mg BD                                      |

PO: Peroral, BD: Twice a day

### Table 7: Indications of tetracyclines in dermatology for their anti-inflammatory properties

| Disease/condition                  | Agent | Mechanism of action for anti-inflammatory property                                                                 | Dose                                           |
|------------------------------------|-------|---------------------------------------------------------------------------------------------------------------------|------------------------------------------------|
| Acne                               | D, M  | Inhibits IL-8\(^{[117]}\)                                                                                          | D-200 mg/day, M-200 mg/day                     |
|                                   |       | Inhibits MMP-1\(^{[118]}\)                                                                                         |                                                 |
|                                   |       | ROS scavenging\(^{[119,120]}\)                                                                                     |                                                 |
|                                   |       | Inhibiting bacterial products that stimulate inflammation\(^{[121,122]}\)                                         |                                                 |
| Confluent and reticulated papillomatosis\(^{[123]}\) | M     | Inhibit T-cell proliferation and granuloma formation\(^{[125]}\)                                                   | D-200 mg/day, M-200 mg/day                     |
| Granulomatous diseases\(^{[126-128]}\)  | D, M  | Inhibit MMP and mast cell activation\(^{[113]}\)                                                                    | T-1500 mg/day, M-100 mg/day, D-100 mg/day      |
| Immunobululous diseases\(^{[126-128]}\) | T, M, D | Inhibit MMP and mast cell activation\(^{[113]}\)                                                                    | T-1500 mg/day, M-100 mg/day, D-100 mg/day      |
| Kaposi’s sarcoma\(^{[129]}\)                                | Chemically modified tetracycline                                                                                 |                                                 |
|                                   |       | Inhibits MMP-2 and MMP-9 \(^{[130]}\)                                                                               |                                                 |
| Lichen planus\(^{[131]}\)                                                   | T, D  | Inhibition of the T-lymphocyte response\(^{[132]}\)                                                               | T - 500 mg BD, D-100 mg BD                     |
| Lupus miliaris disseminatus faciei\(^{[133]}\) | T, M  | Inhibits T-cell proliferation and granuloma formation\(^{[120]}\)                                                  | 100 mg/day                                     |
| Neutrophil disorders\(^{[134,135]}\) | D, M  | Inhibit IL-8 and neutrophil activation\(^{[112]}\)                                                                | D - 200 mg daily, M - 200–300 mg/day          |

Contd...
| Disease/condition                  | Agent | Mechanism of action for anti-inflammatory property | Dose |
|-----------------------------------|-------|----------------------------------------------------|------|
| Pityriasis lichenoides[136]        | T     | Inhibition of the T-lymphocyte response[127]       | 500 mg BD |
| Prurigo pigmentosa[137,138]        | D, M  | Inhibits the migration and/or function of neutrophils[112] | D - 200 mg/day |
|                                   |       | Scavenging ROS                                      | M - 200 mg/day |
| Red scalp disease[139]             | L     | Inhibits matrix metalloproteinases                   | Lymecycline - 300 mg/day |
| Red scrotum syndrome[140]          | D     | Inhibits matrix metalloproteinases and thereby preventing tissue destruction | 100 mg/day |
| Rosacea[141]                       | D, M  | Decreases ROS damage, including inhibiting neutrophils, direct scavenging of ROS, and inhibiting reactions that lead to ROS generation[112] | D - 100 mg/day |
|                                   |       | Act on VEGF, iNOS, and NO and contribute to preventing excessive vascular dilatation and angiogenesis in rosacea[143] | M - 100 mg/day |
|                                   |       | Improve epidermal hydration level[143]              |      |
|                                   |       | Inhibit granuloma formation in vitro[120]          |      |

BD: Twice a day, OD: Once a day, D: Doxycycline, M: Minocycline, ROS: Reactive oxygen species, NO: Nitric oxide, iNOS: Inducible nitric oxide synthase, VEGF: Vascular endothelial growth factor, IL: Interleukin, MMP: Matrix metalloproteinase, T: Tetracycline, L: Lymecycline

| Disease                                         | Agent | Mechanism of anti-inflammatory and immunomodulatory property | Dose |
|-------------------------------------------------|-------|----------------------------------------------------------------|------|
| Autoimmune diseases (lupus erythematosus, dermatomyositis) | HCQ, CQ | Immunomodulating action[144-147] | HCQ - 6.5 mg/kg |
|                                                 |       | Inhibition of lysosomal acidification, phagocytosis, proteolysis, antigen presentation by altering the cleavage of peptides in preparation for binding and presentation by MHC Class II molecules | CQ - 3.5-4 mg/kg |
|                                                 |       | Chemotaxis inhibition | |
|                                                 |       | Decreasing the production of pro-inflammatory cytokines and prostaglandins | |
|                                                 |       | Inhibition of matrix metalloproteinases | |
|                                                 |       | Blocking T and B-cell receptor and toll-like receptor signalling | |
|                                                 |       | DNA stabilization, absorption, and preventing ultraviolet light cutaneous reactions | |

Granulomatous disorders[144-150] | HCQ, CQ | Anti-inflammatory | HCQ - 6.5 mg/kg |
|                                 |       | Stabilization of lysosomal membranes, inhibiting PGE synthesis, and possibly, other enzyme system[140] | CQ - 3.5-4 mg/kg |
|                                 |       | Inhibition of toll-like receptor 9 signal pathway-diminished antigen presentation and immune stimulation | |

Lichen planus[151] | HCQ | Anti-inflammatory | 200 mg BD |
|                   |       | Decreasing the production of pro-inflammatory cytokines[140] | |
|                   |       | Immunomodulatory | |
|                   |       | Reduced stimulation of autoreactive CD4+T cells[152] | |

Contd...
Table 8: Contd...

| Disease                                     | Agent | Mechanism of anti-inflammatory property                                                                 | Dose                                                                 |
|---------------------------------------------|-------|---------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------|
| Acne vulgaris                               |       | Immunomodulatory-Restoration of impaired T-cell function                                                | 2.5 mg/kg/week (up to 150 mg/week)                                  |
| Collagen vascular diseases                  |       | Restoration of delayed hypersensitivity responses and improvement of impaired T-cell function and defective macrophage activity | 150 mg/week for 3-24 months                                          |
| Erythema multiforme                         |       | Sequestration or elimination of persistent antigen                                                      | 150 mg thrice weekly                                                 |
| Human immunodeficiency virus infection      |       | Increase IL-2 production by T lymphocytes                                                              | 2 mg/kg/day for 3 days each week for 24-52 weeks                     |
| Leprosy                                     |       | Immunostimulation-rapid bacterial clearance                                                             | 150 mg thrice weekly                                                 |
| Lichen planus                               |       | Decreases the levels of tumor necrosis factor-α, IL-6, and IL-8                                        | 150 mg thrice weekly                                                 |
| Vitiligo                                    |       | Inhibits the action of endogenous immunosuppressive factors such as                                     | 150 mg on 2 consecutive days every week for periods varying from 4 to 48 months |

BD: Twice a day, PGE: Prostaglandin E, HCQ: Hydroxychloroquine, CQ: Chloroquine, MHC: Major histocompatibility complex

Table 9: Indications of levamisole in dermatology

| Disease                                      | Mechanism of anti-inflammatory and immunomodulatory action                                                                 | Dose                                                                 |
|----------------------------------------------|--------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------|
| Urticaria                                    | Immunomodulatory: Inhibition of by altering the cleavage of peptides in preparation for binding and presentation by MHC Class II molecules | 200 mg/day for a duration of at least 12 weeks                        |
| Acne vulgaris                                | Anti-inflammatory: Inhibition of mast cells-diminished leukotriene synthesis and histamine release                         | 2.5 mg/kg/week (up to 150 mg/week)                                  |
| Collagen vascular diseases                   | PGE: Prostaglandin E, HCQ: Hydroxychloroquine, CQ: Chloroquine, MHC: Major histocompatibility complex                   | 150 mg/week for 3-24 months                                          |
| Erythema multiforme                          | Sequestration or elimination of persistent antigen                                                                       | 150 mg thrice weekly                                                 |
| Human immunodeficiency virus infection       | Increase IL-2 production by T lymphocytes                                                                              | 2 mg/kg/day for 3 days each week for 24-52 weeks                     |
| Leprosy                                      | Immunostimulation-rapid bacterial clearance                                                                              | 150 mg thrice weekly                                                 |
| Lichen planus                                | Decreases the levels of tumor necrosis factor-α, IL-6, and IL-8                                                         | 150 mg thrice weekly                                                 |
| Vitiligo                                     | Inhibits the action of endogenous immunosuppressive factors such as                                                     | 150 mg on 2 consecutive days every week for periods varying from 4 to 48 months |

skin and its appendages. In future, many more cutaneous diseases will be treated and managed with various antibiotics tapping their anti-inflammatory properties. We would like to highlight that in future, these antibiotics will be used albeit in continuous low-dose in various noninfectious dermatoses, thereby minimizing the incidence of side effects.

Financial support and sponsorship

Nil.
### Table 10: Commonly encountered side effects of various antibiotics

| Drugs   | Side effects                                                                                                                                 |
|---------|--------------------------------------------------------------------------------------------------------------------------------------------|
| Clindamycin | Cutaneous: Maculopapular or urticarial eruptions. Older reports have suggested anaphylaxis, erythema multiforme, and Steven–Johnson syndrome  
Gastrointestinal: Antibiotic-associated colitis |
| Clofazimine | Cutaneous: Reversible orange-brown discoloration of the skin, generalized xerosis/ichthyosis  
Gastrointestinal: Enteropathy, splenic infarction, and eosinophilic enteritis |
| Dapsone | Cutaneous: Dapsone hypersensitivity syndrome  
exanammatous eruption, Stevens-Johnson syndrome, toxic epidermal necrolysis  
Photosensitivity  
Hematological: Agranulocytosis  
Neurological: Peripheral neuropathy and psychosis  
Hypothyroidism  
Renal: Nephritis and renal failure  
Hepatic: Hepatitis, cholestasis, cytolytic, and mixed |
| Macrolides | Cutaneous: Fixed drug eruption, leukocytoclastic vasculitis, and hypersensitivity reactions, photosensitivity, angioedema  
Hepatotoxicity  
Exacerbation of myasthenia gravis  
Gastrointestinal: Nausea, abdominal pain, and diarrhea |
| Metronidazole | Topical: Dryness, itching, burning, and stinging |
| Rifampicin | Cutaneous: Orange-red discoloration of liquid body excretions (i.e., urine, sweat, tears, breast milk)  
Anaphylaxis  
Serum sickness such as reaction, disseminated intravascular coagulopathy, conjunctival congestion, linear IgA bullous dermatosis, pemphigus foliaceus, and pemphigus vulgaris  
Neurological: Headache, dizziness, ataxia, inability to concentrate, and fatigability  
Hepatotoxicity |
| Tetracycline | Cutaneous phototoxicity  
Dyspigmentation (hyperpigmentation of skin, nail bed, teeth, bone, and mucous membranes)  
Vasculitis: Cutaneous polyarteritis nodosa (minocycline)  
Lupus-like syndrome  
Drug hypersensitivity syndrome  
Autoimmune hepatitis  
Gastrointestinal  
Benign intracranial hypertension |
| HCQ | Cutaneous: Bluish-gray hyperpigmentation  
Bleaching of hair roots  
Hypersensitivity reactions: Morbilliform, lichenoid, eczematous, urticaria, and exfoliative erythroderma  
Exacerbation and induction of psoriasis  
Transverse pigment bands on nails  
Gastrointestinal: Nausea, vomiting, and diarrhea  
Hematologic: Hemolysis and agranulocytosis  
Neuromuscular: Irritability, nervousness, psychosis, headache, vertigo, tinnitus, nystagmus, and skeletal muscle weakness  
Ocular: Corneal deposition and true retinopathy, “bull’s eye” pigment deposition  
Contd... |
Table 10: Contd...

| Drugs               | Side effects                                                                 |
|---------------------|-------------------------------------------------------------------------------|
| Levamisole          | Cutaneous: Lichenoid eruptions, leg ulcers, fixed drug eruptions, necrotizing vasculitis, and retiform purpura |
|                     | Gastrointestinal: Nausea and abdominal cramps                                  |
|                     | Hematological: Agranulocytosis                                                 |
|                     | Musculoskeletal: Myopathy and arthralgia                                       |
|                     | Neurological-multifocal leukoencephalopathy, ataxia, psychosis                 |
|                     | Others: Flu-like syndrome                                                      |

HCQ: Hydroxychloroquine

Conflicts of interest

There are no conflicts of interest.

What is new?

- Antibiotics refer to collective term for antibacterial, antiviral, and antiparasitic agents
- Antibiotics have multifaceted actions besides killing the infectious organisms
- Anti-inflammatory and immunomodulatory effects of antibiotics make them eligible to be used in various non-infectious conditions in dermatology
- Anti-parasitic drugs are also used in dermatology for their anti-inflammatory and immunomodulatory properties.

References

1. Del Rosso JQ, Schmidt NF. A review of the anti-inflammatory properties of clindamycin in the treatment of acne vulgaris. Cutis 2010;85:15-24.
2. Nakano T, Hiramatsu K, Kishi K, Hirata N, Kadota J, Nasu M. Clindamycin modulates inflammatory-cytokine induction in lipopolysaccharide-stimulated mouse peritoneal macrophages. Antimicrob Agents Chemother 2003;47:363-7.
3. Thomas DR, Raimer S, Smith EB. Comparison of topical erythromycin 1.5 percent solution versus topical clindamycin phosphate 1.0 percent solution in the treatment of acne vulgaris. Cutis 1982;29:624-5, 628-32.
4. Gold MH, Korotzer A. Sub-group analyses from a trial of a fixed combination of clindamycin phosphate 1.2% and benzoyl peroxide 3.75% gel for the treatment of moderate-to-severe acne vulgaris. J Clin Aesthet Dermatol 2015;8:22-6.
5. Webster G. Cutaneous safety and tolerability of a fixed combination of clindamycin phosphate (1.2%) and benzoyl peroxide (3.75%) aqueous gel in moderate-to-severe acne vulgaris. J Clin Aesthet Dermatol 2015;8:22-8.
6. Powell JJ, Dawber RP, Gatter K. Folliculitis decalvans including tufted folliculitis: Clinical, histological and therapeutic findings. Br J Dermatol 1999;140:328-33.
7. Yost J, Robinson M, Meehan SA. Fox-fordyce disease. Dermatol Online J 2012;18:28.
8. George A, Bhatia A, Thomas E. Fox-fordyce disease: A report of 2 cases responding to topical clindamycin. Indian J Dermatol Venereol Leprol 2015;81:87-8.
9. van der Zee HH, Boer J, Prens EP, Jemec GB. The effect of combined treatment with oral clindamycin and oral rifampicin in patients with hidradenitis suppurativa. Dermatology 2009;219:143-7.
10. Pasquale TR, Tan JS. Nonantimicrobial effects of antibacterial agents. Clin Infect Dis 2005;40:127-35.
11. Weinkle AP, Doktor V, Emer J. Update on the management of rosacea. Clin Cosmet Investig Dermatol 2015;8:159-77.
12. Breneman D, Savin R, VandePol C, Vamvakias G, Levy S, Leyden J. Double-blind, randomized, vehicle-controlled clinical trial of once-daily benzoyl peroxide/clindamycin topical gel in the treatment of patients with moderate to severe rosacea. Int J Dermatol 2004;43:381-7.
13. Fdez-Freire LR, Serrano Gotarredona A, Bernabeu Wittel J, Pulpiio Ruiz A, Cabrera R, Navarrete Ortega M, et al. Clofazimine as elective treatment for granulomatous cheilitis. J Drugs Dermatol 2005;4:374-7.
14. Arbiser JL, Moschella SL. Clofazimine: A review of its medical uses and mechanisms of action. J Am Acad Dermatol 1995;32(2 Pt 1):241-7.
15. Gómez-de la Fuente E, del Rio R, Rodriguez M, Guerra A, Rodriguez-Peralto JL, Iglesias L. Granuloma faciale mimicking rhinophyma: Response to clofazimine. Acta Derm Venereol 2000;80:144.
16. Seukeran DC, Stables GJ, Cunliffe WJ, Sheehan-Dare RA. The treatment of acne agminata with clofazimine. Br J Dermatol 1999;141:596-7.
17. Goihman-Yahr M. Malignant pyoderma gangrenosum responding to clofazimine. Int J Dermatol 1996;35:757-8.
18. Kaplan B, Trau H, Sofer E, Feinstein A, Schewach-Millet M. Treatment of pyoderma gangrenosum with clofazimine. Int J Dermatol 1992;31:591-3.
19. Tan BB, Lear JT, Smith AG. Acne fulminans and erythema nodosum during isotretinoin therapy responding to dapsone. Clin Exp Dermatol 1997;22:26-7.
20. Prendiville JS, Logan RA, Russell-Jones R. A comparison of dapsone with 13-cis retinoic acid in the treatment of nodular cystic acne. Clin Exp Dermatol 1988;13:67-71.
21. Sharqie KE, Najim RA, Abu-Raghif AR. Dapsone in Behçet’s disease: A double-blind, placebo-controlled, cross-over study. J Dermatol 2002;29:267-79.
22. Jacyk WK. Behçet’s disease in South African blacks: Report of five cases. J Am Acad Dermatol 1994;30(5 Pt 2):869-73.
23. Harvath L, Yancey KB, Katz SI. Selective inhibition of human neutrophil chemotaxis to N-formyl-methionyl-leucyl-phenylalanine by sulfones. J Immunol 1986;137:1305-11.
24. Thuong-Nguyen V, Kadunce DP, Hendrix JD, Gammon WR, Zone JJ. Inhibition of neutrophil adherence to antibody by dapsone: A possible therapeutic mechanism of dapsone in the treatment of IgA dermatoses. J Invest Dermatol 1993;100:349-55.
25. Booth SA, Moody CE, Dahl MV, Herron MJ, Nelson RD. Dapsone suppresses integrin-mediated neutrophil adherence function. J Invest Dermatol 1992;98:135-40.
26. Debol SM, Herron MJ, Nelson RD. Anti-inflammatory action of dapsone: Inhibition of neutrophil adherence is associated with inhibition of chemoattractant-induced signal transduction. J Leukoc Biol 1997;62:827-36.
27. Person JR, Rogers RS 3rd. Bullous pemphigoid responding to sulfapyridine and the sulfones. Arch Dermatol 1977;113:610-5.
28. Jeffes EW 3rd, Ahmed AR. Adjuvant therapy of bullous pemphigoid with dapsone. Clin Exp Dermatol 1989;14:132-6.
29. Hall RP, Lawley TJ, Smith HR, Katz SI. Bullous eruption of systemic lupus erythematosus. Dramatic response to dapsone therapy. Ann Intern Med 1982;97:165-70.
30. Burrows NP, Bhogal BS, Black MM, Rustin MH, Ishida-Yamamoto A, Kirtschig G, et al. Bullous eruption of systemic lupus erythematosus: A clinicopathological study of four cases. Br J Dermatol 1993;128:332-8.
31. Rao CL, Hall RP. Linear immunoglobulin a dermatosis and chronic bullous disease of childhood. In: Wolff K, Goldsmith LA, Katz SI, Gilchrist BA, Faller AS, Leffell DJ, editors. Fitzpatrick's Dermatology in General Medicine. New York: McGraw Hill Medical; 2003. p. 485-90.
32. Knudson RM, Kalaji AN, Bruce AJ. The management of mucous membrane pemphigoid and pemphigus. Dermatol Ther 2010;23:268-80.
33. Bolotin D, Petronic-Rosic V. Dermatitis herpetiformis. Part II. Diagnosis, management, and prognosis. J Am Acad Dermatol 2011;64:1027-33.
34. Antiga E, Caproni M. The diagnosis and treatment of dermatitis herpetiformis. Clin Cosmet Investig Dermatol 2015;8:257-65.
35. Kim JH, Kim YH, Kim SC. Epidermolysis bullosa acquistia: A retrospective clinical analysis of 30 cases. Acta Derm Venereol 2011;91:307-12.
36. Krahe J, Galal O, Kordass U, Bornemann P. Epidermolysis bullosa atrophicans gravis. Report of a therapeutic trial with dapsone. Monatschr Kinderheilkd 1988;136:140-2.
37. Fort SL, Rodman OG. Erythema elevatum diutinum: An unusual reaction to streptococcal antigen and response to dapsone. Br J Dermatol 1971;84:393-9.
38. Nanda KB, Saldanha CS, Jacintha M, Kamath G. Hailey-Hailey disease responding to thalidomide. Indian J Dermatol 2014;59:190-2.
39. Beutner EH, Chorzelski TP, Wilson RM, Kumar V, Michel B, Helm F, et al. IgA pemphigus foliaceus. Report of two cases and a review of the literature. J Am Acad Dermatol 1989;20:89-97.
40. Fredenberg MF, Malkinson FD. Sulfone therapy in the treatment of leukocytic vasculitis. Report of three cases. J Am Acad Dermatol 1987;16:772-8.
41. Rao CL, Hall RP. Linear immunoglobulin a dermatosis and chronic bullous disease of childhood. In: Wolff K, Goldsmith LA, Katz SI, Gilchrist BA, Faller AS, Leffell DJ, editors. Fitzpatrick's Dermatology in General Medicine. New York: McGraw Hill Medical; 2003. p. 485-90.
42. Chopra A, Mittal RR, Kaur B. Dapsone versus corticosteroids in lichen planus. Indian J Dermatol Venereol Leprol 1982;7:504-10.
43. Stendahl O, Molin L, Lindroth M. Granulocyte-mediated release of histamine from mast cells. Effect of myeloperoxidase and its inhibition by antiinflammatory sulfone compounds. Int Arch Allergy Appl Immunol 1983;70:277-84.
44. Macmillan AL, Champion RH. Generalized pustular psoriasis treated with dapsone. Br J Dermatol 1973;88:183-5.
45. Woodward FC, Collins S. Pyoderma gangrenosum. Clin Dermatol 2000;18:283-93.
46. Martin J, Roenigk HH, Lynch W, Tingwald FR. Relapsing polychondritis treated with dapsone. Arch Dermatol 1976;112:1272-4.
purpura showing an increase in the platelet count following clarithromycin treatment. Rinsho Ketsueki 2003;44:1044-6.

69. Tiaskalová-Hogenová H, Stepánková R, Hudcovíc T, Tucková L, Cukrová B, Lodinová-Zádníková R, et al. Commensal bacteria (normal microflora), mucosal immunity and chronic inflammatory and autoimmune diseases. Immunol Lett 2004;93:97-108.

70. Koizumi N, Ratafomi A, Shinahkai H. Treatment of lupus miliaris disseminatus faciei with roxithromycin. Nishinijh J Dermatol 2003;65:70-3.

71. Aoki D, Ueno S, Kubo F, Oyama T, Sakuta T, Matsushita K, et al. Roxithromycin inhibits angiogenesis of human hepatoma cells in vivo by suppressing VEGF production. Anticancer Res 2005;25:133-8.

72. Goel NS, Burkhart CN, Morrell DS. Pediatric periocular dermatitis: Clinical course and treatment outcomes in 222 patients. Pediatr Dermatol 2015;32:333-6.

73. Ehsani A, Esmaily N, Noormohammadpour P, Toosi S, Hosseinipour A, Hosseini M, et al. The comparison between the efficacy of high dose acyclovir and erythromycin on the period and signs of pityriasis rosea. Indian J Dermatol 2010;55:246-8.

74. Miranda SB, Lupi O, Lucas E. Vesicular pityriasis rosea: Response to erythromycin treatment. J Eur Acad Dermatol Venerol 2004;18:622-5.

75. Sharma PK, Yadav TP, Gautam RK, Tanega N, Satyanarayana L. Erythromycin in pityriasis rosea: A double-blind, placebo-controlled clinical trial. J Am Acad Dermatol 2000;42(2 Pt 1):241-4.

76. Trihan AP, Hebert AA, Esterly NB. Pityriasis lichenoides et varioliformis acuta with azithromycin. J Am Acad Dermatol 1986;15:66-70.

77. Skinner RB, Levy AL. Rapid resolution of pityriasis lichenoides et varioliformis acuta with azithromycin. J Am Acad Dermatol 2008;58:524-5.

78. Ohshima A, Takigawa M, Tokura Y. CD8+ cell changes in psoriasis associated with roxithromycin-induced clinical improvement. Eur J Dermatol 2001;11:410-5.

79. Saxena VN, Dogra J. Long-term oral azithromycin in chronic plaque psoriasis: A controlled trial. Eur J Dermatol 2010;20:329-33.

80. Komine M, Tamaki K. An open trial of oral macrolide treatment for psoriasis vulgaris. J Dermatol 2000;27:508-12.

81. Konno S, Adachi M, Asano K, Okamoto K, Takahashi T. Inhibition of human T-lymphocyte activation by macrolide antibiotic, roxithromycin. Life Sci 1992;51:PL231-6.

82. Wakita H,Tokura Y, Furukawa F, Takigawa M. The macrolide antibiotic, roxithromycin suppresses IFN-γ-mediated immunological functions of cultured normal human keratinocytes. Biol Pharm Bull 1996;19:224-7.

83. Bakar O, Demircay Z, Yuksel M, Haklar G, Sanisoglu Y. The effect of azithromycin on reactive oxygen species in rosacea. Clin Exp Dermatol 2007;32:197-200.

84. Fernandez-Obregon A. Oral use of azithromycin for the treatment of acne rosacea. Arch Dermatol 2004;140:489-90.

85. Rubin BK. Immunomodulatory properties of macrolides: Overview and historical perspective. Am J Med 2004;117 Suppl 9A: 25-45.

86. Levert H, Gressier B, Moutard L, Brunet C, Dine T, Lyuexx M, et al. Azithromycin impact on neutrophil oxidative metabolism depends on exposure time. Inflammation 1998;22:191-201.

87. Kadota J, Iwashita Y, Matsubara Y, Ishimatsu Y, Yoshinaga M, Abe K, et al. Inhibitory effect of erythromycin on superoxide anion production by human neutrophils primed with granulocyte-colony stimulating factor. Antimicrob Agents Chemother 1998;42:1866-7.

88. Colina M, Lo Monaco A, Khodeir M, Trotta F. Propionibacterium acnes and SAPHO syndrome: A case report and literature review. Clin Exp Rheumatol 2007;25:457-60.

89. Schaefferkebe T, Lequen L, de Barbeyrac B, Labbé L, Bébéar CM, Morrier Y, et al. Propionibacterium acnes isolated from synovial tissue and fluid in a patient with oligoarthritis associated with acne and pustulosis. Arthritis Rheum 1998;41:1889-93.

90. Kirchoff T, Merkdesal S, Rosenthal H, Prokop M, Chavan A, Wagner A, et al. Diagnostic management of patients with SAPHO syndrome: Use of MR imaging to guide bone biopsy at CT for microbiological and histological work-up. Eur Radiol 2003;13:2304-8.

91. Assmann G, Kuech T, Kirchoff T, Rosenthal H, Voswinke J, Pfunderschuh M, et al. Efficacy of antibiotic therapy for SAPHO syndrome is lost after its discontinuation: An interventional study. Arthritis Res Ther 2009;11:R140.

92. Nishimuta K, Ito Y. Effects of metronidazole and tindazole ointments on models for inflammatory dermatitis in mice. Arch Dermatol Res 2003;294:544-51.

93. Khodaeiani E, Fouladi RF, Yusufi N, Amirmia M, Babaeinejad S, Shokri J. Efficacy of 2% metronidazole gel in moderate acne vulgaris. Indian J Dermatol 2012;57:279-81.

94. Akamatsu H, Oguchi M, Nishijima S, Asada Y, Takahashi M, Ushijima T, et al. The inhibition of free radical generation by human neutrophils through the synergistic effects of metronidazole with palmitoleic acid: A possible mechanism of action of metronidazole in rosacea and acne. Arch Dermatol Res 1990;282:449-54.

95. Khan EJ, Ullman TA, Ford AC, Abreu MT, Abadir A, Marshall JK, et al. Antibiotic therapy in inflammatory bowel disease: A systematic review and meta-analysis. Am J Gastroenterol 2011;106:661-73.

96. Green B, Morrell DS. Persistent facial dermatitis: Pediatric periocular dermatitis. Pediatr Ann 2007;36:796-8.

97. Vanderweil SG, Levin NA. Perioral dermatitis: It’s not every rash that occurs around the mouth. Dermatol Nurs 2009;21:317-20, 353.

98. Bocek K, Abeck D, Werfel S, Ring J. Perioral dermatitis in children – Clinical presentation, pathogenesis-related factors and response to topical metronidazole. Dermatology 1997;195:231-6.

99. Rodriguez-Caruncho C, Bielsa I, Fernandez-Figueras MT, Ferrándiz C. Childhood granulomatous periorificial dermatitis with a good response to oral metronidazole. Pediatr Dermatol 2013;30:e98-9.

100. Schmadel LK, McEvoy GK. Topical metronidazole: A new therapy for rosacea. Clin Pharm 1990;9:94-101.

101. Del Rosso JO, Baum EW. Comprehensive medical management of rosacea: An interim study report and literature review. J Clin Aesthet Dermatol 2008;1:20-5.

102. Del Rosso JO. A status report on the medical management of rosacea: Focus on topical therapies. J Drugs Dermatol 2008;7:125-33.

103. Zip CM. Innovative use of topical metronidazole. Dermatol Clin 2002;70:271-5.

104. Miyachi Y, Imamura S, Niwa Y. Anti-oxidant action of metronidazole: A possible mechanism of action in rosacea and acne. Arch Dermatol Res 1989;282:449-54.

105. Bikowski J. Facial seborrheic dermatitis: A report on 222 patients. Pediatr Dermatol 2005;22:353.

106. McFald WL, Roebuck HL. Rational management of papulopustular rosacea with concomitant facial seborrheic
dermatitis: A case report. J Clin Aesthet Dermatol 2011;4:40-2.
107. Seckin D, Gurbuz O, Akin O. Metronidazole 0.75% gel vs. ketocanozole 2% cream in the treatment of facial seborrhoeic dermatitis: A randomized, double-blind study. J Eur Acad Dermatol Venereol 2007;21:345-50.
108. Mendonça CO, Griffiths CE. ClindAMYcin and rifampicin combination therapy for hidradenitis suppurativa. Br J Dermatol 2006;154:977-8.
109. Mela M, Mancuso A, Burroughs AK. Review article: Pruritus in patients with chronic liver disease and its management. J Hepatol 2005;42:96-107.
110. LeCluyse EL. Pregnane X receptor: Molecular basis for species differences in CYP3A induction by xenobiotics. Chem Biol Interact 2001;134:283-9.
111. Marshall HU, Wagner M, Zollner G, Fickert P, Diczfalusy U, Gunhold J, et al. Complementary stimulation of hepatobiliary transport and detoxification systems by rifampicin and ursodeoxycholic acid in humans. Gastroenterology 2005;129:476-85.
112. Khurana S, Singh P. Rifampin is safe for treatment of pruritus due to chronic cholestasis: A meta-analysis of prospective randomized-controlled trials. Liver Int 2006;26:943-9.
113. Kazandjieva J, Kamarshev J, Hinkov G, Tsanov K. Rifampicin and psoriasis. Akt Dermatol 1997;23:78-81.
114. Paunescu E. In vivo and in vitro suppression of humoral and cellular immunological response by rifampicin. Nature 1970;228:1188-90.
115. Nilsson BS. Rifampicin: An immunosuppressant? Lancet 1971;2:374.
116. Dajani BM, Canady MS, Thompson JS, Kasik JE. Rifampicin: An immunosuppressant? Lancet 1972;2:1094.
117. Monk E, Shalita A, Siegel DM. Clinical applications of non-antimicrobial tetracyclines in dermatology. Pharmacol Res 2011;63:130-65.
118. Griffin MO, Fricovsky E, Ceballos G, Villarreal F. Tetracyclines: Drugs with huge therapeutic potential. Mini Rev Med Chem 2011;11:38-41.
119. Bahrami F, Morris DL, Pourgholami MH. Tetracyclines: Drugs with huge therapeutic potential. Mini Rev Med Chem 2012;12:64-52.
120. Rao TN, Guruprasad P, Sowjanya CH, Narasirdevi I. Confluent and reticulated papillomatosis: Successful treatment with minocycline. Indian J Dermatol Venereol Leprol 2010;76:725.
121. Bachelez H, Senet P, Cadranel J, Kaukhov A, Dubertret L. The use of tetracyclines for the treatment of sarcoidosis. Arch Dermatol 2001;137:69-73.
122. Webster GF, Toso SM, Hegemann L. Inhibition of a model of in vitro granuloma formation by tetracyclines and ciprofloxacin. Involvement of protein kinase C. Arch Dermatol 1994;130:748-52.
123. McSweeney TP, Sadowski PL, Veitch JM. Hydroxychloroquine: From malaria to autoimmunity. Clin Rev Allergy Immunol 2012;42:145-53.
124. Aburientos C, Sperber K, Shapiro DL, Aronow WS, Chao CP, Ash JY. Hydroxychloroquine in systemic lupus erythematosus and rheumatoid arthritis and its safety in pregnancy. Expert
Pradhan, et al.: Anti-inflammatory and immunodulatory actions of antibiotics

146. Sisó A, Ramos-Casals M, Bové A, Brito-Zerón P, Soria N, Muñoz S, et al. Previous antimalarial therapy in patients diagnosed with lupus nephritis: Influence on outcomes and survival. Lupus 2008;17:281-8.

147. Costedoat-Chalumeau N, Dunoqué B, Morel N, Le Guern V, Guettrot-Imbert G. Hydroxychloroquine: A multifaceted treatment in lupus. Fresse Med 2014;43(6 Pt 2):e167-80.

148. Carlín MC, Ratz JL. A case of generalized granuloma annulare responding to hydroxychloroquine. Cleve Clin J Med 1987;54:229-32.

149. Babuna G, Buyukbabani N, Yazganoglu KD, Baykal C. Effective treatment with hydroxychloroquine in a case of annular elastolytic giant cell granuloma. Indian J Dermatol Venereol Leprol 2011;77:110-1.

150. Jones E, Callen JP. Hydroxychloroquine is effective therapy for control of cutaneous sarcoidal granulomas. J Am Acad Dermatol 1990;23(3 Pt 1):487-9.

151. Atzmony L, Reiter O, Hodak E, Gdalevich M, Mimouni D. Treatments for cutaneous lichen planus: A systematic review and meta-analysis. Am J Clin Dermatol 2016;17:11-22.

152. van Loosdregt J, Spreafico R, Rossetti M, Frakken BJ, Lotz M, Albani S. Hydroxychloroquine preferentially induces apoptosis of CD45RO effector T cells by inhibiting autophagy: A possible mechanism for therapeutic modulation of T cells. J Allergy Clin Immunol 2013;131:1643-6.e1.

153. Reeves GE, Boyle MJ, Bonfield J, Dobson P, Loewenthal M. Impact of hydroxychloroquine therapy on chronic urticaria: Chronic autoimmune urticaria study and evaluation. Intern Med J 2004;34:182-6.

154. Ochsendorf FR. Use of antimalarials in dermatology. J Dtsch Dermatol Ges 2010;8:829-44.

155. Rassai S, Mehri M, Yaghoobi R, Sina N, Mohhebbipour A, Feily A. Superior efficacy of azithromycin and levamisole vs. azithromycin in the treatment of inflammatory acne vulgaris: An investigator blind randomized clinical trial on 169 patients. Int J Clin Pharmacol Ther 2013;51:490-4.

156. De Cree J, De Cock W, Verhaegen H. Levamisole treatment of inflammatory acne. Restoration of impaired T-cell function accompanied by clearing of the lesions. Biomedicine 1979;31:95-9.

157. Scherak O, Smolen J, Kolarz G, Kojer M, Menzel J. Clinical experience with levamisole treatment of patients with systemic lupus erythematosus (author’s transl). Wien Klin Wochenschr 1979;91:758-62.

158. Rovenský J, Cebeocauer L, Zitnan D, Lukáč J, Ferencík M. Levamisole treatment of systemic lupus erythematosus. Arthritis Rheum 1982;25:470-1.

159. Lozada F, Spiller L, Silverman S Jr. Clinical and immunologic responses to levamisole in 13 patients with erythema multiforme. Int J Immunopharmacol 1980;2:63-8.

160. Lozada F. Levamisole in the treatment of erythema multiforme: A double-blind trial in fourteen patients. Oral Surg Oral Med Oral Pathol 1982;53:28-31.

161. Castro Garzón M, Mubita M, Kachinka L. Levamisole treatment in HIV-infected Zambian children. Lancet 1992;340:1099-100.

162. Kar HK, Bhatia VN, Kumar CH, Sirumman P, Roy RG. Evaluation of levamisole, an immunopotentiator, in the treatment of lepromatous leprosy. Indian J Lepr 1986;58:592-600.

163. Lu SY, Chen WJ, Eng HL. Response to levamisole and low-dose prednisolone in 41 patients with chronic oral ulcers: A 3-year open clinical trial and follow-up study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;86:438-45.

164. Khondker L, Khan SI. Efficacy of levamisole for the treatment of slow spreading vitiligo. Mymensingh Med J 2013;22:761-6.

165. Renoux G. Modulation of immunity by levamisole. J Pharmocol Ther 1978;2:288-96.