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Review Article

A review on lupus disease, symptoms and their treatment with traditional medicines

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ABSTRACT

Lupus erythematous is an autoimmune illness that mostly affects women and has no recognized cause. The classical period, the neoclassical period and the modern period are the three periods in the history of lupus erythematous. Important discoveries have occurred at each period allowing for a better understanding of a conditions.Pericarditis, valvular lesions and myocardial dysfunction, particularly mild pericarditis. As a result, echocardiography should be done on SLE patients on a regular basis. Vascular blockage, including coronary arteries can occur as a result of SLE related vasculitis early atherosclerosis or antiphospholipid antibodies. The immune system of the body becomes overactive and targets normal, healthy tissue. Inflammation, swelling and damage to the skin, joints, skin, kidney, blood, heart, and lungs are among the symptoms and medicinal treatment commonly used with the help of ayurvedic treatment, allopathic treatment and homeopathic treatment.

1. Introduction

Lupus affect skin, joint, blood vessels, tissue and other organ. It has a genetic component, is often triggered by viruses, and can be life threatening. A similar disease occurs in a strain of mice and ancient time 2003 onwards this has generated result that have greatly increased the understanding of human disease. There is strain of mice with lupus and these have been shows to lack and enzyme called DNase1, which removes rubbish from cells22. Removal of an immune system signaling protein, SLAM associated protein or SAP also cause lupus23. In 2001 a lupus causing gene was identified in mice. It causes a fault in enzyme called alpha-mannosidase 2^{nd}. Scientists now need to know more about how these substances interact. Scientists have used gene therapy to prevent the development of lupus in mice by boosting level in an immune system component. A receptor gene knows as Fc acts like a gatekeeper, helping to maintain a healthy immune system instead of one that turns on itself, as is the case in autoimmune disease. In mice and in human with lupus production of the Fc receptor is reduced. Gene therapy reversed this and prevented the disease in the lupus-susceptible strain of mice, but not in untreated mice. The scientists expect their funding to apply to humans, since there seemed to be no serious adverse effect from the therapy in mice. A protein molecule that interferes with the site where antibodies at reduce the mortality in mice with genetic lupus from 80% down to 10% and virtually eliminates the kidney damage 25 and a tranquilizer like drug Bz-423, reduces the incidence of renal complications by 85%26 as does an anti-cancer drug.[1]

Auto antibodies, complement activation and immune complex deposition produce systemic lupus erythematosus (SLE), a multisystem disease with a wide range of clinical manifestations.[2] With a female to male ratio of 9 to 1, the disease primarily affects women of childbearing age.

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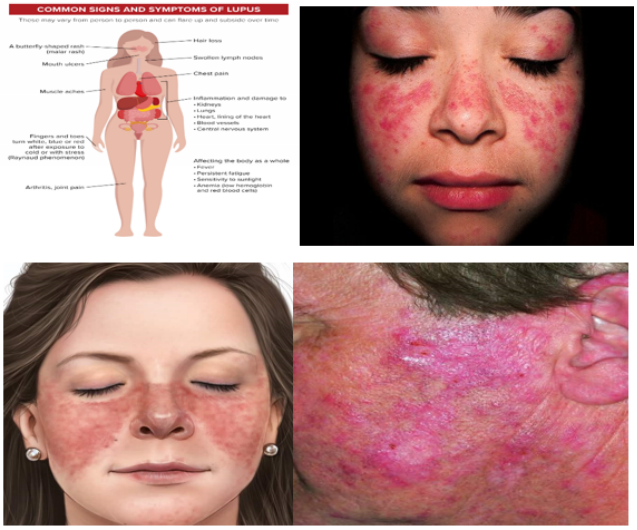

Fig. 1: Common Sign and symptoms of lupus disease.

SLE is a complicated and complex immune pathogenesis. Despite advance in therapy choice and increase survival rates, SLE remains an incurable disease. Given the recent explosion in the number of scholarly articles and the numerous studies on SLE, gaining a complete overview of the leading research in the field is difficult.[3] There are two basic methods for acquiring a comprehensive overview of a research subject both of which can be used in tandem to detect potential knowledge gaps. First both narrative and systemic literature reviews concentrateon research finding with the goal of drawing a board conclusion.[4] Systemic literature reviews differ from narrative literature reviews in that they have a well stated goal and search strategy, as well as specified inclusion and exclusion criteria.[5] The second way is to employ bibliometrics, which is an analytical and mapping method for assessing the links and influence of published papers and citations quantitatively.[6] Lupus is a disease with an unpredictable course that includes flares and remissions and where cumulative damage over time significantly reduce quality of life and impairs organ function. This disease can damage multiple cells, tissue, organ and the clinical picture varies widely between people. Indeed, even within a single patient, the clinical picture may change with time. Joints, skin, mucous membrane, blood cells, brain and kidney are the organ system most typically implicated in lupus patients.[7] An immune response by auto-antibodies against a person's own tissues is involved in the mechanism. Anti-nuclear antibodies are the most prevalent type and they cause inflammation. The diagnosis is based on a mix of symptoms and laboratory tests and it can be complex.

1.1. Classical period (1230-1856)

The thirteenth century physician Rogerius is created with coining the term "lupus" (Latin for "wolf") to describe erosive facial lesions that resembled wolf bites. Thomas Bateman, a student of the British dermatologist Robert William, in the early 19th century; **Cazenave**, a student of the French dermatologist Laurent Biett, in the mid nineteenth century and Moriz Kaposi (born Moriz khon) a student and son in low of the Austrian dermatologist Ferdinand von Hebra, in the late nineteenth century, were the first to describe the various dermatologic features of lupus.[8]

1.2. Neoclassical period (1872-1948)

It was contested for many years whether lupus was a manifestation of tuberculosis, another disease that was still being described at the time. Because tuberculosis was regarded to be significantly different in those pre bacteriological days, the misconception developed. Lupus was defined by Erasmus Wilson (1809-1884) as follows:

The dominant character of lupus then could be destruction. However additional research into the aetiology of lupus revealed that this debilitating disease was preceded by a confined thinking and prominence of the skin typically referred to as a tubercle and hence lupus is regarded as a skin tuberculosis action. In the first occurrence the term lupus signified a destructive action. Intended to be limited to the type of tubercle that cause destructive ulceration; nevertheless, as cutaneous disease become more understood, it was discovered that there was a type of tubercle that did not ulcerate but was chronic and long lasting in its essence and which has a deep pit or a powerful force behind its cicatrix with a distinct pattern. His particular type of cutaneous illness has been identified. "Lupus erythematous" was coined by Cazenave.[9] The photosensitivity of lupus erythematous rashes was discovered by Jonathon Hutchinson (1828-1913). Kaposi originally defined the disease systemic nature in 1872. The neoclassical phase of lupus began as result of this. According to him, "Lupus erythematosus can be managed, according to experience". By far more severe pathological alterations, and even potential deadly the procedure may be intricately linked to constitutional symptoms. In question and that death may occur as a result of situations that must be addressed. It thought to be caused by a local ailment.[10] Moriz Kaposi (1837-1902), von Hebra's student and son-in-low was the first to distinguish between the two forms of lupus discoid and disseminated lupus.[11] During the years 1895-1904, Sir William Osler (1849-1919) described systemic lupus erythematosus in three articles. Only a few of the skin disease he studied were lupus erythematosus.[12] In 1923, Emanuel Libman (1876-1946) and Benjamin sacks (1896-1971) published a report on four patients with non-

infectious endocarditis. Two of these patients had the classic lupus erythematosus malar rash. These individuals appeared to be comparable to the individuals appeared to be comparable to the erythema patients in Osler's writings, according to Libman and sacks. More individuals were suspected of having systemic lupus erythematosus, but due to the absence of a rash, they were diagnosed with polyserositis and glomerulonephritis.[13] Sulfonamides were first used in 1938 to treat discoid LE, which was followed by SLE a few years later. Although it did not cure the condition, it helped alleviate the symptoms.[14]This theory was proposed by Arnold Rich (1893-1968). Rich felt that anaphylaxis caused the collagen and endothelium of patients to be affected by the primary lesions of SLE, but he didn't know how this happened.[15]This chemical was also discovered to cause thrombosis in 1963.[16] This chemical was later discovered to cause spontaneous miscarriages in SLE patients in 1975.[17]Anti-phospholipid syndrome is the name to the condition.[18]

1.3. Modern period (1948-present)

With the discovery of the LE cell, Hargraves and his colleagues hered in the contemporary era. Hargraves found LE cells in patients with acute disseminated lupus erythematosus and proposed that the LE cell was formed by the phagocytosis of free nuclear material and contained a vacuole containing partially lysed and digested nuclear material. "Peculiar fairly structureless globular entities taking purple stain" were found in the marrow aspirate of a young infant with an unexplained illness, according to Hargraves. This happened two more patients before they were finally diagnosed with SLE.[19] In 1950, it was discovered that this is a gamma factor globulin. "Hematoxylin bodies" that appeared to be identical to the material phagocytosed by the LE cell.[20] The first known case of placental transmission of the LE factor, the previously stated anti-DNA antibody, occurred in 1954.[21] In 1963, the lupus band test was created to diagnose lupus. In this test a skin biopsy is done and then immunofluorescent microscopy is used to see if immunoglobulin's are deposited at the dermo epidermal junction.[22]Cyclophosphamide is an alkylating chemical that limits DNA synthesis and thus cell division by cross linking DNA strands. Inosine monophosphate dehydrogenase an enzyme involved in the rate limiting stage of the de-novo purine synthesis is inhibited by mycophenolate mofetil. Azathioprine is an imidazolyl mercaptopurine derivative that inhibits purine metabolisms.[23] Immunomodulation is becoming a common treatment option for managing and treating this condition. Cyclophosphamide, mycophenolate mofetil and azathioprine are three instances of this method. Biological medicine like rituximab and lymphostat B are another option for treatment. Rituximab is a monoclonal antibody that targets the antigen CD20 on B-lymphocytes. Lymphostat B lymphocyte stimulator monoclonal antibody.[24]

1.4. Sign and Symptoms

SLE receives its name from the fact that it usually affects multiple organ system in your body including:

1. Age and Sex.
2. Skin.
3. Pregnancy.
4. Muscles and Bones.
5. Kidney.
6. Heart.Lungs.
7. Nervous system.

1.4.1. Age and Sex

Women, especially those of childbearing age are more likely to develop SLE. This increased incidence could be due to hormones, specifically oestrogen as studies have revealed that women who had an early menarche or used oral contraceptives or hormonal therapy were more likely to develop SLE.[25] Men have a lesser risk than women in their adolescent or postmenopausal years. Klinefelter's syndrome, which is characterised by an extra X chromosome in males, has been related to an increase of SLE, adding to the evidence for a hormonal aetiology in SLE.[26]

1.4.2. Skin

After joint involvement, the skin is the second most usually affected organ and skin lesions are the second most prevalent manner that this disease manifests itself.[27]In nearly 80% of patients with SLE, the skin and mucous membrane are symptomatic at some point.[28] Alopecia, scaring lesions, deformity and other skin lesions cause significant morbidity in these individual and as a result roughly 45% of patients have some degree of vocational handicap.[29] Skin lesions in SLE patients are divide between those that are caused by lupus specific disease such as malar mash and those that are caused by lupus non-specific disease such as alopecia.[30]

1.4.3. Pregnancy

Thrombosis, infection, thrombocytopenia, transfusion, pre-eclampsia and mortality are all major medical and pregnancy issue that women with SLE face.[31] If a woman has active SLE or major organ involvement, it is suggested that she not become pregnant due to increased risk of miscarriage, stillbirth, premature, delivery and SLE exacerbation. Because excessive amount of oestrogen might trigger SLE exacerbations, oral contraceptive must be used with caution.[32]Placental transfer of maternal antibodies, which might cause cutaneous or cardiac problems should be carefully monitored in new-borns (e.g., congenital

heart block and cardiomyopathy). Prophylaxis with aspirin, low molecular weight heparin or both is recommended for women with SLE and antiphospholipid antibodies to prevent foetal loss.[33]

1.4.4. Muscles and bones

In people with SLE, involvement of the musculoskeletal system quite common.[34] Joint discomfort is the most common reason for seeking medical help, with tiny joint of the hand and wrist being the most commonly impacted, though any joint can affect. One of the most common causes of early clinical presentation in people with SLE is joint discomfort.[35] The most common symptoms are arthralgia, arthritis, osteonecrosis (avascular necrosis of bone) and myopathy. Arthritis and arthralgias have been reported in as many as 95% of SLE patients.[36] These symptoms can be mistaken for another type of inflammatory arthritis and can occur months or years before SLE is diagnosed. The small joints of the hands, wrists and knees can be affected by arthralgia, myalgia and frank arthritis. SLE arthritis or arthralgia unlike rheumatoid arthritis can be asymmetrical with pain that is disproportionate to swelling.[37] SLE arthritis and arthralgias are usually migratory with morning stiffness measured in minutes. SLE arthritis is seen as nondeforming in most cases. Antibodies against the citrulline containing peptide (anti-CCP) were detected in 8% of patients with SLE.[38] Osteoporosis which is frequently caused by glucocorticoid medication, raises the risk of fractures. Myositis is present in certain SLE patients as evidence by biopsy.[37]

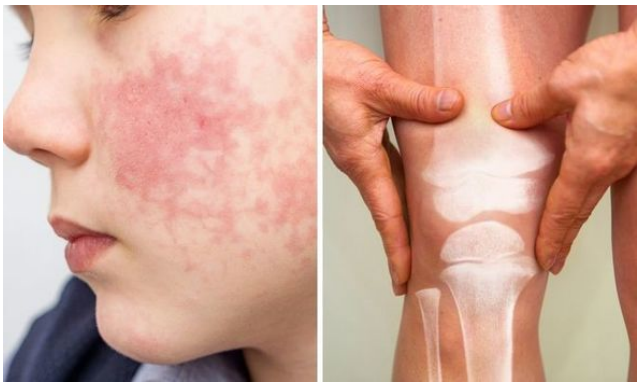

Fig. 2: Lupus in joints.

1.4.5. Kidney

Kidney inflammations due to increased lupus in some people called nephritis. The kidney filters toxins and waste materials from the blood which are very difficult in times of inflammation.

Symptoms include:

1. High blood pressure.
2. Darker urine.
3. Pain in your side.
4. Having to urinate more frequently at night.
5. Blood in your urine.
6. Swelling in your lower legs and feet.

Early warning signs and symptoms may go missed. Kidney function should be monitored after diagnosis. End-stage renal disease can develop if lupus nephritis is left untreated.[39]

1.4.6. Heart

In systemic lupus erythematous, the heart is frequentlyimplicated (SLE). The prevalence of cardiac involvement in SLE has been determined to be higher than 50% using highly sensitive method of cardio vascular research. The pericardium, endocardium, myocardium, coronary arteries and conduction tissue are all possible targets.

1.5. Pericarditis

Acuteand chronic inflammatory changes can affect the pericardium. Sero fibrinous pericarditis and fibrinous pericarditis are two types of acute pericarditis. The fibrous or fibro fibrinous characteristics of chronic pericarditis predominate. Direct immunofluorescence 4 shows granular immune and C3 deposition, indicating that immunocomplex that play a role in the development of pericarditis.[40] Pericardial involvement is more common at the outset of SLE or during relapses. Cardiac tamponade, constrictive pericarditis and purulent pericarditis are very uncommon pericarditis complications. When a pericardial effusion is large, a chest x-ray is used to document it. The usual method for investigating pericardial abnormalities is echocardiography, which can show modest effusions or thickening of the pericardial layers. In most cases, effusions are linked to active disease in other organs.[41]

1.6. Myocarditis

Myocarditis is the most common symptoms of SLE related cardiac involvement. Myocardial dysfunction in SLE patients on the other hand, may be caused by various factors such as coronary artery disease (CAD) caused by early atherosclerosis, hyper tension, renal failure, valvular disease and pharmaceutical toxicity such as cyclophosphamide and chloroquine.[42] SLE myocarditis has symptoms that are similar to myocarditis caused by other causes, such as viral myocarditis and can lead to ventricular dysfunction, dilated cardiomyopathy and heart failure. There are a number of non-invasive tests that can be used to diagnose cardiac involvement in SLE. Echocardiography reveals findings such as global, regional and segmental wall motion abnormalities, decreased ejection fraction, increased chamber size and delayed isovolumic relaxation time

that while not specific are symptomatic of myocardial inflammation and dysfunction.[43]

1.7. Coronary artery disease (CAD)

Large transmural infractions frequently due to atherosclerotic plaque of at least one of the three major extramural coronary arteries and embolism small areas of necrosis adjacent to small intramural coronary arteries whose lumen appears restricted and walls infiltered by inflammatory cells are two major histological findings in CAD. The development of CAD is influenced by a number of processes. Atherosclerosis, coronary arteries, thrombotic events with or without aPL, vasospasm or valvular embolization and hypertension are all example. A vasculitis process can affect the minor coronary arteries.[44] Angina pectoris, myocardial infraction, and sudden death are all symptoms of CAD. The difference between atherosclerosis and coronary vasculitis in SLE patients is difficult but crucial for therapeutic decisions. Young people with active disease which is often of short duration are more likely to develop ischemia due to vasculitis although atherosclerosis related ischemia occurs earlier in SLE patients than in the general populations, it affects older SLE patients who have had the disease for a longer time and have taken corticosteroids for a longer period of a time. Ischemic cardiopathy caused by APS can occurs at any age and at any stage of disease's progression.[45]

1.8. Lungs

SLE may affect any organ; the lungs are usually implicated in other organ involvement later during the disease. The SLE related lungs illness is the most prevalent pulmonary manifestation, while pleura, parenchyma, lung, diaphragmatic dysfunction can be observed in other cases. High level of pulmonary infection makes the detection of the true prevalence of lung involvement with SLE complex. Lung involvement is currently regarded to be mostly a consequence of infection and not directly SLE in 80% of SLE patients with autopsy and chart analysis from earlier findings.[46]

Table 1: Most common pulmonary manifestation of SLE

Pleural disease	Pleural effusion pleurisy.
Vascular involvement	Acute reversible hypoxemia. Pulmonary embolism disease. Pulmonary arterial hypertension.
Parenchymal disease	Acute lupus pneumonitis. Acute respiratory distress chamber. Diffuse alveolarhaemorrhage. Shrinking lung syndrome.
Airways disease	Obstructive lung disease. Upper airways disease.

1.9. Nervous system

In order to encourage its clinical and scientific research the diversity of neurological disease n lupus generated a classification system.[47]They differentiate generally between issues affecting the central nervous system. These criteria have been offered with slight amendments, but for almost two decades they have stayedmostly constant. Neurological events as well as outcome indicators such as SLICC/ACR damage index were also integrated into lupus diagnosis criteria. The formulation of the definitions of ACR neurolepts contributed to encourage the epidemiological study of lupus neurology and proved the participation of the nervous system as an important negative factor of quality of life.However, such research has brought to light one of the field most pressing issues: demonstrating a causal link between a neurological condition and lupus. The ACR criteria for example include phrases like headache and mood problem, which are common in the general populations and seen at similar rates in healthy, matched controls and patients with other chronic inflammatory disorders. As a result, they are less likely to be directly caused by lupus. When "minor events" like headaches and anxiety problems are included in population's studies, 40% of patients had experienced at least one neuropsychiatric event. The criteria's specificity is much improved when mild symptomatology is excluded.[48] As a result, they are less likely to be directly caused by lupus. When "minor events" like headaches and anxiety problems are included in population studies 40% of patients had experienced at least one neuropsychiatric event.[49]

Types of lupus: Lupus are four types.

1. **Systemic lupus erythematous**
2. **Cutaneous lupus**
3. **Neonatal lupus**
4. **Drug induced lupus**

1.9.1. Systemic lupus erythematous

The most frequent kind of lupus is systemic lupus erythematous (SLE). When someone says they have lupus, they are almost certainly referring to SLE. The severity of SLE can range from mild to server. Symptoms may worsen and then improve as the illness progress. Flares are when your symptoms get worse, whereas remissions are when they get better or go away.

1.9.2. Cutaneous lupus

This kind of lupus usually affects only your skin. It can cause rashes and scars from permanent lesions. The following are some of the different kinds of cutaneous lupus:

1. **Acute cutaneous lupus:** This kind results in a characteristic "butterfly rash". This is a rash that occurs on the cheeks and nose and is red in colour.

2. **Subacute cutaneous lupus:** A rash that is red, raised and scaly forms on the body as a result of this type of cutaneous lupus. It usually occurs on places that have been exposed to sunlight and does not result in scarring.
3. **Chronic cutaneous lupus:** This type generates a rash that is purple or red. Skin discolouration, scarring or hair loss is possible side effects. Discoid lupus is another name for it. While acute cutaneous lupus is frequently linked to systemic lupus; subacute and chronic cutaneous lupus usually affects the skin solely.

1.9.3. Neonatal lupus

This is a very rare illness that affects infants whose mothers have certain autoimmune antibodies are passed down through the placenta from mother to foetus. Not all mothers with these antibodies experience lupus symptoms. In fact, nearly 25% of mothers who give birth to a child with neonatal lupus have no symptoms of the disease. However, it is estimated that 50% of these moms will have symptoms within 3 years. The following are some the symptoms of these conditions:

1. Liver problem after birth.
2. A skin rash.
3. Low blood cell count.

While some babies may suffer cardiac problems, the majority of them will experience symptoms that will subside within a few months. Autoantibodies (SSA/B) can however, the placenta and cause issues with heart conduction (heart block). During pregnancy, patients with these antibodies must be extensively monitored by experts, including a rheumatologist and a high-risk obstetrician (fatal-maternal medicine).

1.9.4. Drug induced lupus

Drug induced lupus can be caused by using certain prescription drugs (DIL). Drug-induced lupus erythematous (DIL) is another name for DIL (DILE).DIL can develop as a result of long-term usage of some prescription drugs, usually after only a few months. There is a verity of medicines that can cause DIL. Here are a few examples:

1. Terbinafine (an antifungal) and pyrazinamide (an antibacterial) are example of antimicrobials (a tuberculosis medication).
2. Phenytoin (Dilan tin) and valproate are anticonvulsant medicines.
3. Drugs to treat arrhythmia, such as quinidine and procainamide.
4. High blood pressure medicators such as timolol (Tim optic, istlol) and hydroxyzine.
5. Anti-TNF-alpha medicines, such as infliximab (Remicade) and etanercept, are biologics (Enbrel). While DIL has symptoms that are similar to SLE, the disease seldom affects major organs. It can however, cause pericarditis and pleurisy. DIL usually disappears within a few weeks of discontinuing the medicine that caused it.[50]

1.9.5. Treatment of lupus disease

As we all know that lupus disease is a very dangerous disease. That's why we have put its medicinal terms in three steps for its treatment.

1. Ayurvedic treatment.
2. Allopathic treatment.
3. Homeopathic treatment.

1.10. By Ayurvedic treatment

1.10.1. Oral medication

Oral drugs that are indicated in vatarakta illness were chosen. Because vata and rakta dusti were present, the favoured remedy was tablet kaishora guggulu which included the medicinal Guduchi (tinospora cordiofolia).[51] Because the main complaint was toe gangrene, tablet gandhaka rasyana was administration to aid wound healing and infection control.[52] Rasyana mediations were recommended because this is an autoimmune and avarana condition conditions, hence tablet shivagutika was added.[53] Orally Mahamanjistadi Kashaya was administration to normalise rakta vitiation.[54] Avipattikara churna, a laxative was suggested since the doshas were badly vitiated and regular cut cleansing was required.[54]

1.10.2. Panchakarma treatment

There are several processes panchakarma that will help you get out of it, which are detailed below:

1. **Patra potli pind sweda :** A potli is wrapped in herbs such as Nir gundi, Eranda and neem during this process. The potli is then soaked in herbal oil, which is then heated for a few minutes. The potli is applied to the injured body part. This treatment is used to relieve the discomfort in the joints caused by systemic lupus erythematosus.
2. **Shashti shali pind sweda:** A potli loaded with Shashti shali rice is cooked in medicinal oil in this technique. This potli is then used to massage the affected portion of the body. This treatment is used to treat skin irritation, inflammation and itching in people with systemic lupus erythematosus.
3. **Abhyanga :** After the massage the medicated oil is put on the patient's body. In individuals with systemic lupus erythematosus, this massage helps to reduce irritation, discomfort, stiffness and swelling.
4. **Basti :** Patients with SLE are given the medicinal oil or decoction in the form of enema. The body's total

detoxification is aided by this technique. It aids in the elimination of all poisons from the body.

5. **Lepa :** Licorice (yashtimadhu) and milk are used to make Lepa. This Lepa is applied on your faces butterfly rash. Itching, irritation and swelling of the skin are reduced by the herb's cold powder.[55]

1.10.3. Ulcer management

Dry gangrenous foot ulcers were treated conservatively with Gomutra arka (distilled cow's urine) wash and jatyadi taila dressing. The terminal phalanx of the left second toe was on the point of the falling off after 6 days of treatment and was surgically removed without anaesthetic or antibiotic protection. The dressing of the ulcers was done on a daily basis. She was instructed to eat solely a vegetarian diet that was low in spice and oil. She was told to stay away from pickles, brinjal, cabbage and cauliflower. During the entire treatment, no concomitant allopathic medication was given.

1.11. Allopathic treatment: commonly used for medication in the treatment of systemic lupus erythematous

1. **Drug class:** NSAIDs [including salicylates]
   Mechanism of action: Block prostaglandin synthesis through inhibition of cyclooxygenase enzymes, producing anti-inflammatory, analgesic and anti-pyretic effect.
   Adverse effects: GIT irritation, bleeding, renal toxicity, hepatic toxicity and hypertension
2. **Drug class:** Antimalarials [Hydroxychloroquine]
   Mechanism of action: Unknown may reduce cytokine activity and interfere with T-cell activation also thought to inhibit intracellular TLRs.
   Adverse effect: Macular damage, muscle weakness
3. **Drug class:** Corticosteroids [Prednisone, Methylprednisolone]
   Mechanism of action: Multiple immune system impacts (stopping cytokine activation and suppressing interleukins, interferon and tumour necrosis factor.
   Adverse effect: Hyperlipidaemia, hypertension, cataracts, hypokalaemia, osteoporosis and increased risk of infection
4. **Drug class:** Immunosuppressants [Cyclophosphamide, Azathioprine, Mycophenolate]
   Mechanism of action: Multiple suppressive effect on immune system (DNA and RNA disruption
   Adverse effect: Renal dysfunction, infertility, myelosuppression hepatotoxicity and cancer
5. **Drug class:** Monoclonal antibodies [Belimumab].
6. **Mechanism of action:** B-cell survival is inhibited and B-cell differentiation into immunoglobulin-producing cells is reduced when BLYs binds to receptors on B-cells
   Adverse effect: Insomnia, depression, diarrhoea and pyrexia[56]
7. Adverse effect: Insomnia, depression, diarrhoea and pyrexia $

1.12. Homeopathic treatment: commonly used for medication in the treatment of systemic lupus erythematous

1. **Belladonna:** Mallar rash or bitter fly rash of systemic lupus erythematous with symptoms of neuropsychiatric SLE (NPSLE) where CNS involvement is prominent as well as PNS symptoms of NPSLC.
2. **Borax:** Again, this is cure is helpful for mucosal ulcers although the ulceration in this therapy is more pronounced in the oral mucosa than in the nasopharynx.
3. **Ferrum Phosphoricum :** When a patient has a fever due to as illness that cause malaise, exhaustion, hair loss or anaemia due to lupus or its medications, low potencies of biochemic form might be given along with other medicines.
4. **Ferrum Metal licum:**A red acute rash with involvement of the oral mucosa is typical of acute lupus erythematous although it can also be seen in later choric phases when there are symptoms of haemolyticanaemia.

2. Conclusion

The history of lupus erythematous can be traced back to 400Bc, thanks to Hippocrates writings. Hebra was also the first to include the drawing of LE in its publications. The LE cell was found was Hargraves. Antinuclear antibodies were first detected vis immune fluorescence, which was later used to demonstrate their presence. Later, immunofluorescent microscopy was employed to perform "lupus band test" on skin samples. It's possible that early one-set SLE isn't a single disease, but rather a heterogeneous collection of single gene abnormalities linked to infection susceptibility and affecting several organs. SLE still linked with severe co-morbidity and has an influence on health-related quality of life despite advance in our understanding of its aetiology, path physiology and disease management. The anti-phospholipids syndrome is a condition that can coexist with SLE and cause morbidity and mortality. The SLICC/ACR criteria are used to describe damage and several approaches have been validated for defining disease activity.

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4. Conflict of Interest

There are no any conflict of interest by authors.

5. Author Contribution

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