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ERYTHRODERMIC PSORIASIS: CURRENT PERSPECTIVES WITH AN EMPHASIS ON TREATMENT

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ABSTRACT

Psoriasis has improved knowledge on skin biology. It is a genetic disease with chronic, inflammatory, hyperproliferative skin disease, it is protype of a group of intradermal disease, defined as regular elongation of the rete ridges with preservation of the rete ridges & dermal papillae pattern. Psoriasis affects people of all ages, &in all countries. The psoriasis reported frequency in countries ranges between 0.09%-11.43%, by which 100 million people individually affected making psoriasis a serious global problem Depending on whether the lesion is early or resolving, psoriasiform epidermal changes may be refined or bulging. It puts negative impact on life & feeling of condemn is a central consequence for example already emerge with only small patches of skin being affected. While treating this disease dermatologist should address possible effects by problematic encounters with the public & in sexual relationship even if the severity of disease is low because lesions on invisible parts of body can already cause serious impairment. Self-help organization & by taking part in an inter-disciplinary patient education program can help grave psychosocial consequence it is led by dermatologist. In 1st & 2nd decade of life, is the beginning of disease involvement is often unusual or mild and an assured diagnosis may be difficult to establish. In children's lesions are often smaller, thinner & less scaly in adults. Therapeutic options have drawbacks or are not approve in childhoods treatment can be challenged. Psoriasis is life-long disease affects children & their parents need special cares & supervision. Erythrodermic psoriasis is rare

affecting to the people living with psoriasis. Psoriasis can cause shedding of skin layer in large sheets intense redness. The whole body gets often affects & can be life-threatening. So, in this Erythrodermic Psoriasis review article we will see new comorbid, treatment & self-helping techniques for patient to co-up with regular lifestyle.

KEYWORDS: Psoriasis, Genetic diseases, Erythrodermic, Treatment.

INTRODUCTION

Psoriasis is a red, itchy, and scaly skin disease, typically on the knees, the cobblestones, the trunk, and the skinner trend to undergo cycles, flames for a couple of weeks or months, then subsidies or remissions for some time. You use the treatments to manage your symptoms. The body produces too many T cells, a type of white blood cell that usually spreads away from bacteria and viruses in patients with psoriasis. No infectious psoriasis. Something usually causes psoriasis that results or worsens symptoms. The triggers differ between people. These T cells attack healthy cells of the skin during psoriasis. As a result, skin cell overproduction occurs in relation to other symptoms. Psoriasis symptoms mostly occur on the skin, but can also affect the nails, joints, and other parts of the body. The erythroderma psoriasis is very rare and severe. Erythrodermic psoriasis is a rare form of psoriasis, an aggressive form of inflammatory response. Symptoms include a rash peeling over the whole-body surface. The rash can burn or itch, and it spreads fast. Psoriasis erythroderma is one of the most severe psoriasis types. It can be life-threatening if complications develop. Erythrodermic psoriasis is also called by Zumbusch psoriasis, one of the most severe psoriasis. According to the World Psoriasis Day consortium, millions of people in the United States and worldwide – 2 to 3 percent of all people – have psoriasis. With a prevalence of 0.44-2.8% in India, men are generally afflicted two times more than women in their third or fourth decade.[48]

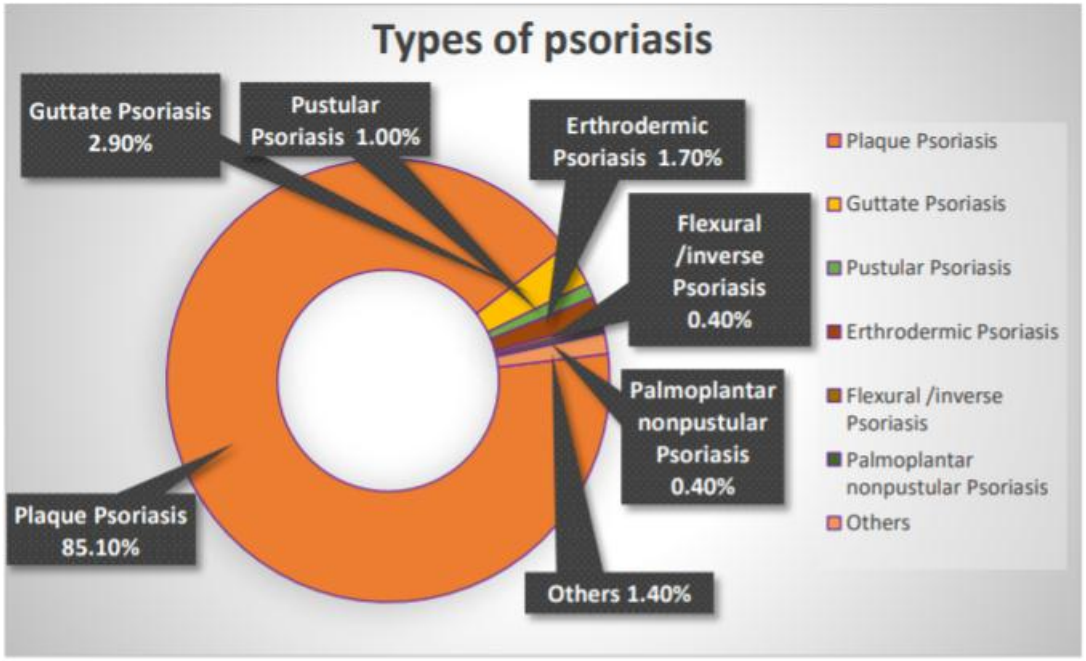

Figure 1: Types of Psoriasis.

History

- In the 1st century AD, the first explanation of psoriasis is given in the book of Cornelius Celus De re medica libri octo during the Roman Empire.
- The Greek word leprosy was lopos and lepo leprosy (to scale).
- Hippocrates (460–377 BC) was the first person to write accounts of skin conditions.
- Many historians have been credited to the Roman scholar Celsus the first clinical account of papulosquamous conditions (around 25 BC-45 AD).
- Galen (133–200 AD) used the first word psoriasis, but the description did not consider the psoriasis.
- In 1868, Von Hebra described erythrodermic psoriasis for the first time.

Etiology

The cause of psoriasis is unclear but involves immune stimulation of epidermal keratinocytes; T cells seem to play a central role. Family history is common, and certain genes and human leukocyte antigens (Cw6, B13, and B17) are associated with psoriasis. Genome wide linkage analysis has identified numerous psoriasis susceptibility loci; the PSORS1 (psoriasis susceptibility gene 1) locus on chromosome 6p21 plays the greatest role in determining a patient's susceptibility of developing psoriasis. An environmental trigger is thought to evoke an inflammatory response and subsequent hyper proliferation of keratinocytes.[46]

Well-identified triggers include

- Injury (Koebner phenomenon)
- Sunburn
- HIV infection
- Beta-hemolytic streptococcal infection (leading to guttate psoriasis)
- Drugs (especially beta-blockers, chloroquine, lithium, angiotensin-converting enzyme inhibitors, indomethacin, terbinafine, and interferon- α)
- Emotional stress
- Alcohol consumption
- Tobacco smoking
- Obesity

Pathogenesis of Ep

Exact pathogenesis is not well known in erythrodermic psoriasis.[49] The report None the less, some findings indicate that this disease is primarily associated with a Th2 Immune phenotype with a Th17 contribution. This includes a complex interplay of inflammatory tracts Th1, Th2, & Th17.

The Th1-Th2 ratio was lower in EP compared to PV patients (T-helper 1 & T-helper 2) There's been vastly greater interleukin level (IL-4) & (IL-10) in EP patients. Ratio of the interferon (IFN) –IL-4 and T box in EP banding Protein 3 (GATA-3) in T cell/GATAs were both below 1.0 which refers to the reversal in compared to two other types.

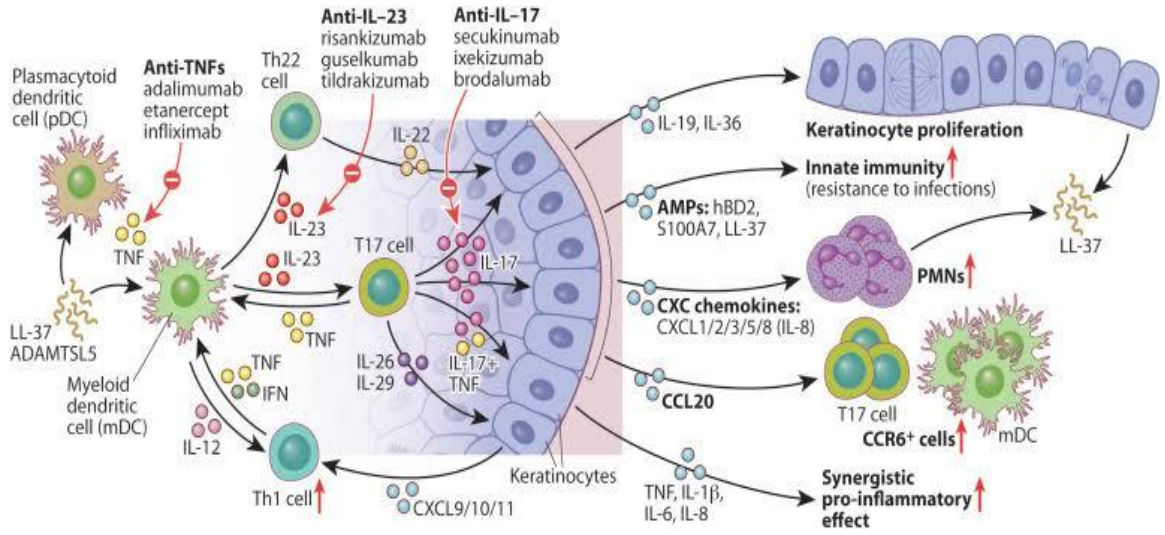

Figure 2: Pathogenies of Erythrodermic Psoriasis.

The Th1 cytokines INF-β & IL-2 as well as the Th2 cytokines IL-4 & IL-10 had higher serum levels in patients with EP.[50] EP marked by significant growth factor-bb growth of the protein platelet attributing plasma level of IL-4, IL-6 and monocyte chemo. EP patients had significantly increased IL-13 and macrophages inflammatory protein-1-beta compared to healthy control.[51]

Th17 cells have recently been investigated as part of the EP pathogenesis. Th17 cells secrete T-cells, dendritic cells & neutrophils IL-17, IL-22, & IFN-secrete α including the development of chemical kinesis.[57] After Th2 in EP lesion, Th17 was considered to be the most prominent T cell subset.[52]

The TNF α is another key player in EP pathogenesis, particularly in the light of the documented efficacy of anti-TNF α agent in disease therapy studies, the fast systematic discharge of TNF α from EP could lead to the onset and severity of the disease.[53] The epidermal turnover rate of TNF greatly increased, leading to a mitotic rate above average.[54] The volume of germ cells increases keratinocyte transfer times by decreasing epidermis allowing the loss of only surface cellular content.[55]

Cytokine tumor necrosis is a factor in the pathogenesis of many inflammatory disorders such as EP due to weak apoptosis inducer (TWEAK)[56] It is not well studied but can play a role in PV, PP and EP pathogenesis via synergy to IL-36 Gama but not IL-22. In the activation of proinflammatory cytokines & chemokines, the key function of TWEAK (a tumor necrosis factor-like poor apoptosis inducer).

FACTORS AFFECTING ERYTHRODERMIC PSORAISIS

- **Food and Erythrodermic psoriasis**

- **Red meat**

Red meat especially bacon, dairy, and eggs contain a polyunsaturated fatty acid called arachidonic acid which are known to create psoriatic lesions.

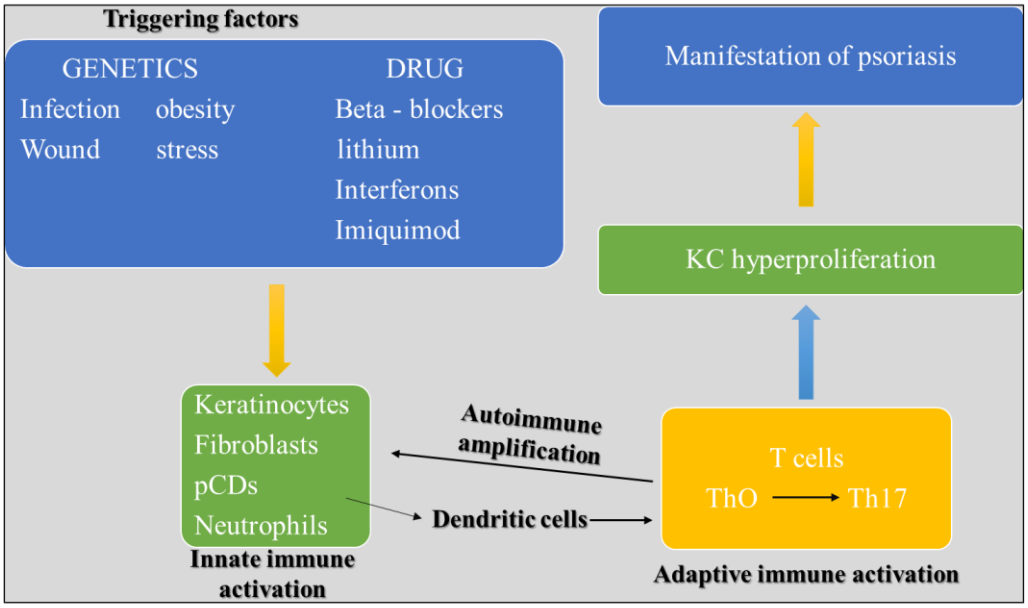

Figure 3: Triggering factor.

- **Gluten**

Celiac disease is a health condition in which characterized by an autoimmune response to the protein gluten. People with psoriasis should cut gluten containing foods such as wheat and wheat derivatives, barley, certain processed foods, certain sauces, and beverages.

- **Processed food**

Eating too many processed food high in salt, sugar, high calorie food can lead to obesity, metabolic syndrome, and certain conditions such as chronic inflammation in body, which linked to source to psoriasis flare.

- **Alcohol & EP**

Alcohol may cause psoriasis through various pathways, such as increased infection susceptibility, lymphocyte activation and keratinocyte proliferation and proinflammatory cytokine production.[40] Alcohol or alcohol abuse and psoriasis also are related. Depression, a common co-occurring condition of psoriasis, causes abuse of alcohol several times. Abuse of alcohol can cause psoriasis and other skin conditions, such as pink skin and acne. Certain ways the trusted source may have a detrimental effect on psoriasis Dangerous reactions with certain psoriasis medications such as methotrexate are more likely to cause liver injury and disease more severe psoriasis.[41]

❖ MANAGEMENT OF EP

- **General considerations**

Correction of any fluid, protein, and electrolyte abnormalities; a nutritional assessment; hypothermia prophylaxis; and treatment of any secondary infections must all be part of the initial management of EP. Sepsis caused by skin pathogens, most commonly Staphylococcus aureus, has been reported to be a particularly severe and potentially fatal complication.[43]

The medical board of the National Psoriasis Foundation in the United States approved consensus guidelines in 2010 regarding the appropriate management of EP after approach is the best measures were implemented. In acute and unstable cases, they advise cyclosporine or infliximab as first-line therapy. Acitretin and methotrexate, on the other hand, are preferred agents for more stable cases. Etanercept and combination therapy were also second-line options. However, additional information about the efficacy of newer agents has emerged since that publication. Regarding which, we would go over the most recent data on the treatment of EP.[44,45]

Guidelines on Erythrodermic Psoraisis

Guidelines on screening for comorbidities in pediatric patients with psoriasis have been issued by the Pediatric Dermatology Research Alliance and National Psoriasis Foundation.[42] Features include the following:

- Overweight or obesity - Start at age 2 years; use body mass index criteria
- Type 2 diabetes -Starting at age 10 years or puberty onset in overweight patients with two risk factors, screen every 3 years; screen obese patients every 3 years regardless of risk factors; use fasting serum glucose value for screening
- Dyslipidemia - Start at age 9-11 years and then again at age 17-21 years; use universal lipid screening; fasting lipid panel recommended
- Hypertension - Starting at age 3 years, screen yearly using age, sex, and height reference charts
- Nonalcoholic fatty liver disease (NAFLD) - Starting at age 9-11 years, use alanine aminotransferase in overweight or obese children with risk factors (e.g., insulin resistance, prediabetes or diabetes, central adiposity, dyslipidemia, sleep apnea, family history of NAFLD/nonalcoholic steatohepatitis (NASH); consider screening at a younger age if patients have greater risk factors (e.g., severe obesity, family history of NAFLD/NASH, hypopituitarism); with normal screening results, repeat alanine

aminotransferase screening every 2-3 years based on risk factors (or sooner if they increase in number or severity)

- Polycystic ovary syndrome - Consider screening in patients with symptoms (e.g., oligomenorrhea, hirsutism)
- Gastrointestinal disease - Considering evaluating patients with decreased growth rate, unexplained weight loss, or symptoms of inflammatory bowel disease
- Arthritis - Screen periodically with review of systems and physical examination
- Uveitis - Only warranted in psoriatic arthritis.
- Mood disorders and substance abuse - Regardless of age, annually for depression and anxiety; at age 11 years, annually for substance abuse
- Quality of life - Consider using formal instrument (e.g., Children's Dermatology Life Quality Index).

Severity of Disease

We highlight specific advances and discoveries of psoriasis research in this article. Systemic treatment of methotrexate resuscitation was also used in our early case handling.[27] Erythroderma can injure skin's thermal function, leading to hypothermia, high cardiac power causes malfunction and metabolic changes including hypoalbuminemia and anemia due to iron loss Vitamin B12 and folate.[28]

Disease severity can assist in the treatment of diseases. Various topical therapies for mild to severe diseases are safe and effective. More extreme conditions could require systemic therapy, such as phototherapy, acitretin, methotrexate, cyclosporine, or biological treatment.[29]

Erythrodermic psoriasis therapeutic therapy is needed in patients with erythrodermic psoriasis. Retinoids, MTX (methotrexate), Cs-A (cyclosporine-acitretin) and biologic agents are used in this medication. The treatment of erythrodermic psoriasis has demonstrated long-term effectiveness of acitretin and MTX (methotrexate). Their beginning is late, though. A progressive dose reduction could effectively avoid recurrence. For patients suffering from serious or unhealthy conditions, it is advised that Cs-A (cyclosporine-acitretin) or biological agents be used. Local or systemic GLS are not commonly indicated until severe, life-threatening signs of toxicity are present in patients.

In situations of severe illness or urgent patient condition, systemically using steroids should be used in order to suppress acute inflammation; the dosage will eventually be reduced by removal of the medicine if the disorder is con-trimmed. If the patient has fever, hypoproteinemia, fluid and electrolyte disequilibrium, secondary infections or liver failure, so the systemic situation should be controlled by offering diet assistance, water and electrolyte treatment imbalances, infection prevention and care and hepatic function safety. Moreover, the operation of essential organs and structures including the heart, kidneys and central nervous system should be protected.[30]

Topical therapy in the first case is typically prescribed for patients with minor to moderate diseases. If not enough, mild to serious disease & light therapy or standard therapy (methotrexate, cyclosporine, acitretin) are considered. If this is not a small molecule in medication (FAEs, apremilast) or biologics is seen.[31] FAEs (Fumaric acid ester) are useful in patients with plaque type psoriasis and mild PsA (Psoriasis Arthritis).

The effect of cigarettes and alcohol on psoriasis has been highly focused primarily.[32] Systemic corticosteroids should be avoided in general since they can exacerbate erythrodermic psoriasis (but may be the only option in pregnant or breastfeeding mothers) and phototherapy is contraindicated because these patients are often photosensitive.[33]

Differential diagnoses of EDP include extreme dermatitis, opioid eruptions, skin lymphoma and rare psoriasis as keratinizing disease is equivalent to yellow palm hyper characteristics. EDP differential diagnosis is similar in terms of the patient.[34] The lesion region is more than 90% BSA (Body Surface Area) and EDP (Erythrodermic Psoriasis) has a record of other types of psoriasis.[35]

Multitudes such as cardiovascular and metabolic drawbacks are related to malignant and physiological conditions. Anxiety, depression, low effective expressiveness, and poor conflict resolution abilities, sleep disturbance, sexual disorder & treatment are part of the physiological comorbidities.[36]

While its quality of life is seriously affected, it is not infectious.[37] Dermatology Life Quality Index (DLQI) is designed to measure the health-related quality of life of adult patients suffering from a skin disease.[38] Psoriasis Area & Severity Index (PSAI) is a tool to measure the severity & extent of psoriasis response rate.[39]

- **TREATMENT**

Existing therapy

Treatment goals for each patients are customizing on the basis of the concomitant comorbities, adverse effects, existing quality of life, self-care capability, drug history.[1] The initial management of erythrodermas the same regardless of etiology
This should include replacement of nutritional, fluid electrolyte losses.[2]

Local skin care measure should be employed, such as oatmeal baths as well as wet dressing to weeping or crusted sites followed by application of bland emollients & low-potency corticosteroids.[3] Known precipitants & irritants are to be avoided & underlying cause with its complication is to treated.[4]

Secondary infection is treated with antibiotics Edema is dependent area such as periorbital & pedal areas, may require diuretics. Use of cyclosporine or infliximab is first-line therapy in acute &unstable case. Second-line options include etanercept & combination therapy.[5]
Topical treatments include: -
Topical steroid cream &moisturizers
Wet dressing
Oatmeal baths

Topical Steroid

Steroids are often used as a temporizing or adjunctive measure while an alternative treatment is introduced.[6] For instance, clobetasol 0.05% for study & desonide 0.05% for free have been used successfully in combination with methotrexate in a severe patient requiring intensive care unit.[7]

Topical vitamins D analogues

Two groups effectively incorporated a vitamin d analog as one arm of a combination therapy to treat erythrodermic manifestation on the side of patient's body treated with the vitamin D analogues. The whole body was then treated with 100g of calcitriol in conjunction with low-dose ultraviolet is, leading to marked improvement in 4 weeks.[8]Calcitriol & calcipotriene have emerged as important alternatives to topical cortico- steroids for long term therapy of therapy.[9]

Corticosteroids

They are most frequency prescribed medications for treating mild to moderate psoriasis they slow cell turnover by suppressing the immune system which reduces inflammation &itching. Low potency corticosteroid ointments are usually recommended for sensitive areas such as diabetes, hypertension & HPA (hypothalamic-Pituitary-Axis) suppression.[10]

Anthralin (Oithranol)

It is derived from the araroba tree found in South America. it includes reactive oxy-gen species release which has inhibitory effects on hyper proliferating keratinocytes transformation of leucocytes. It is used in increase concentration for application to scalp It can be applied on in-patient basis also out-patient short contact therapies. Adverse effects are discoloration of hair & skin irritation.[11]

Coal tar

It is one of the oldest typical therapies both as monotherapy & in combination with other topical agents' systemic agents with other topical agents, systemic agents & phototherapy for treatment of Psoriasis The polycyclic aromatic hydrocarbons present in coal tar makes the skin more sensitive to ultraviolet light.[12] Coal tar has anti-inflammatory, anti-proliferative & anti-pruritic properties. its un-pleasant smell, staining properties & mutagenic potential has made it less compliant in order to increase compliance, some non-staining & shampoos are available either alone or in combination with other active agents.[13]

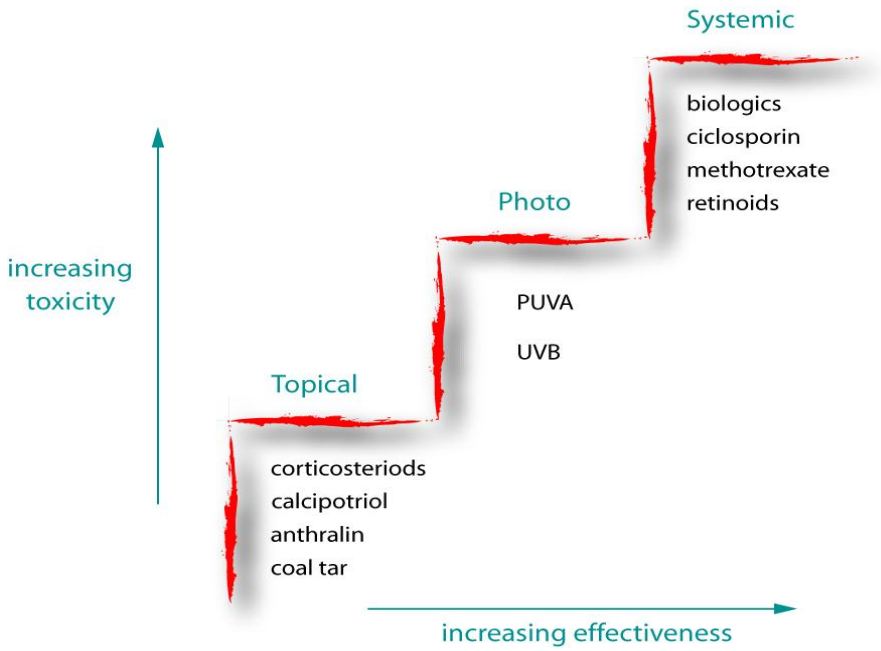

Figure 4: Various Treatment.

Phototherapy

Phototherapy is an effective, first-line of treatment for moderate -too severe plaque psoriasis that works by inhibiting keratinocyte, proliferation, promoting keratinocyte apoptosis, & dampening the inflammatory.[14]

Th1 & Th17 pathways

The use of phototherapy in acute fulminant ep is discouraged given the risk of koeberrzation.[15] Phototherapy can play a role in long-term management of EP once the disease course becomes more stable.[16] It is recommended for the patient who do not respond to topical therapies or for patient with plaques of psoriasis covering 20% or more of body surface. Ultraviolet B (UVB) is radiation, combined with coal tar (Goeckerman therapy) or anthralin (In gram region) has seen to be effervescing in patient's moderate-to-psoriasis. Ultraviolet a radiation (UVA) combined with systemic psoriasis PUVA therapy (psoralen and ultraviolet A) has seen to highly effective in clearing skin lesion, but both these therapies require maintained treatment & they increase the risk of skin cancer.[17]

Acitretin

Acitretin is a synthetic retinoid indicated for treatment for moderate to severe Psoriasis, it is role as an adjunctive therapy to after systemic gents has been well documented to enhance efficacy, lower dose & reduce occurrence of side effects.[18] Com-mon side effect includes mucocutaneous dryness, arthralgia, gastrointestinal upset & photo sensitivity. Acitretin is a potent teratogen that is best avoid in women of childbearing age & potential is recommended that women not get pregnant for 3yrs after discontinuing the medication.[19]

Retinoids

The second-generation retinoids have also used in combination with other systemic agents, such as cyclosporine & infliximab. Oral retinoids are mainly used as performance therapy in used in erythrodermic psoriasis but it seems to be less efficacies.

Methotrexate

Like acitretin, methotrexate also has a slower onset of alerion & is considered a high -priority agent for more stable cases of EP. Its s either taken orally or administered

As a once-weekly injection, commonly 5.75 mg or 15-20mg & can be supplemented with 1mg daily folic acid long term use of methotrexate may increase risk of hepato-toxicity, hepatic fibrosis, & bone marrow suppression.[20]

5-Cyclosporine

Cyclosporine is an immunosuppressive agent that stock IL-2 Tran-scription, there by impairing the growth & the activity of t-cells, given its rapid on-set of alerion, cyclosporine is considered a critical first-line drug for the control of unstable cause of EP more severe disease after requires a starting dose of 5mg /kg/d.

Case series & reports advocate the use of cyclosporine in treatment of EP at dose of 1.5-5mg/kg/d for 2 week to 4 months.[21]

Mycophenolate mofetil

It is another immune suppressant that selectively inhibits aeriated lymphocytes use of mycophenolate mofetil in two patient with severe EP has also been reported in literature, over a 6-week period both patient experienced 70% skin improvement with no side effects or disease relapse after drug cessation. It should not use in pregnancy.[22]

- **Target Based therapy**

Biologics

Biologics therapy encompasses an emerging category of drugs that target specific cytokines of immune system. Some biologic is adopted for treatment of EP, including

- **Types of Biologics**

- TNF- α inhibitors
- IL-12/IL-23 inhibitors
- IL-17 inhibitors.

TNF- α inhibitors

- **Etanercept**

It is a soluble in TNF- α receptor fusion protein that acts as a decoy for endogenous TNF- α. this interaction inhibits a biological inflammatory cascade of TNF- α. Etanercept is administered subcutaneous once to twice weekly.

- **Adalimumab**

It is a fully human monoclonal antibody against TNF- α. It is administered subcutaneously at an initial dose of 80mg at week 0, followed by 40mg every other week.[23]

- **Infliximab**

It is a chimeric anti-TNF-α monoclonal antibody that interacts with both soluble & membrane TNF- α. This interaction results in decrease epidermal T-cell infiltration. It has relatively quick onset & alerion that is comparable to that & cyclosporine. Therefore, expert consensus consider drug an additional first line option for unstable case of EP.

SUMMARY OF THERAPIES AVAILABLE TO TREAT PSORIASIS :			
EXISTING THERAPY	Corticosteroids Vitamin D analogue Anthraline Coal tar Retinoids Methotrexate Cyclosporine Phototherapy		
TARGET BASED THERAPY :			
BIOLOGICS	Anti TNF-A agents	Infliximab	Certolizumab pegol
		Etanercept	Adalimumab
		Golimumab	
	IL – 23 and IL – 12 inhibitors	Ustekinumab	Apilimod
		Guselkumab	
	IL -17 A receptor inhibitor	Brodalumab	
	IL -17 A inhibitors	Secukinumab	Ixekizumab
	Fusion protein inhibitor	Allefacept	
	Janus kinase inhibitor	Tofacittinib	
	Phosphodiesterase – 4 inhibitor	Apremilast	
	Anti-CD-6 Monoclonal antibody	Itolizumab	

Figure 5: Therapies.

- **IL-12/23 Inhibitors**

- **Ustekinumab**

It is a fully human monoclonal antibody that binds the P40subunit of both IL-12 & iL-23. These two cytokines are involved in pathogenesis of psoriasis by stimulating the Th1 & Th17 inflammatory pathways. It is approved for treatment of moderate-to-severe plaque psoriasis & psoriasis arthritics & is administered subcutaneously.[24]

- **IL-17 inhibitors**

- **Ixekizumab**

It is as humanized IgG monoclonal antibody that inhibitsIL-17A, an inflammatory cytokine of Th17 pathway that has been implicated in pathogenesis of psoriasis.[25]

Treatment in children

Consist of supportive management in the form of maintenance of fluid & electrolyte balance & ambient temperature, strict aseptic environment.

Therapeutic options in pediatrics psoriasis are plenty, but choice should be governed by type & extent of the disease, otherwise it can lead to significant problems Indications for using systemic therapy in childhood psoriasis are refractory disease, erythrodermic pustular, psoriatic arthropathy. Methotrexate is 1st line therapy or childhood psoriasis, EP respond better to systemic retinoids the response was good but, there were multiple episode of reoccurrence.[26]

How can Erythrodermic Psoriasis be prevented?

At present, because they are an inherited disease, there are no prevention methods for erythrodermic psoriasis. Proper self-care and frequent visits to medical will help alleviate severity and uncomfortable conditions.

Check for continuous and appropriate care of psoriasis vulgaris can help, in some cases, to prevent erythrodermic psoriasis. The disposal of psoriasis medication should be planned and accompanied by instructions. Avoiding reactions that worsen the disorder can aid in therapy Genetic testing of anticipated parents and prenatal (molecular pregnancy fetal testing) diagnosis (including family members) may help us understand the risks during pregnancy better. Genetic counselling can help identify a vulnerability prior to caring for the infant where a genetic aspect of the disease exists. Actual active work is being conducted to investigate the possibilities.[47]

CONCLUSION

According to our findings, it accounts for more than half of all serious psoriasis cases. primarily impacts adult males It normally aggravates a mild case of psoriasis. Erythrodermic psoriasis affects less than 3% of all psoriasis patients, distinguished by a scaly, erythematous rash, desquamation, and exfoliation affecting more than three-fourths of the skin surface region. Identifying and manage erythrodermic psoriasis is particularly relevant due to its scarcity and subsequent complications.[58] Improving the EDP (Erythrodermic Psoriasis) patient lifestyle substantially linked to life qualities can increase and decrease patient morbidity and mortality. Our data show that the treatment of the self-care of medicines and the check-up of the routine is a successful treatment of EDP (Erythrodermic Psoriasis)

patients however, in the current research, more medications with fewer side effects should be created.

REFERENCES

1. Sauder DN, Mamelak AF. Understanding the New Clinical Landscape for Psoriasis: A Comparative Review of Biologics. J Cutan Med Surg., 2004; 8: 205-12.
2. Rothe MJ, Baily TL, grant-kels JM. erythroderma. Dermatol clin., 2000; 18: 405-15.
3. Rothe MJ, Bernstein ML, grant-kels JM. Life-threatening erythroderma: diagnosing and treating the "red man". Clin Dermatol, 2005; 23: 206-17.
4. Karakayli G, Beckham G, Orengo I, Rosen T. Exfoliative dermatitis. Am Fam Physician 1999; 59:625-30 & Sehgal VN, Srivastava G, Sardana K. Erythroderma/exfoliative dermatitis: A synopsis. Int J Dermatol, 2004; 43: 39-47.
5. Rosenbach M, hsus, korman NJ, et al. treatment of erythrodermic psoriasis: from the medical board of national psoriasis foundation. J Am acad Dermatol, 2010; 62(4): 655-662.
6. Prystowsky JH, Cohen PR. pustular and erythrodermic psoriasis. Dermatol clin, 1995; 13(4): 757-770.
7. Vaidya TSv, Lewallen RS, fledman SR. erythrodermic psoriasis and severe hypotension requiring intensive care with hospitalization: poor treatment Outcome as a result of poor adherence. J dermatolog treat, 2015; 27(2): 134-135.
8. Van der Vleuten CJ, Gerritsen MJ, Steijlen PM, de Jong EM, van de kerkhof PC. A therapeutic approach to erythrodermic psoriasis: report of a case and a discussion of therapeutic options. Acta derm venereal, 1996; 76(1): 65-67.
9. Kragballe K. Calcipotriol: a new drug for topical psoriasis treatment. Pharmacol Toxicol, 1995; 77: 241-6.
10. Schlager JG, Rosumeck S, Werner RN, Jacobs A, Schmitt J, Schlager C et al. Topical treatments for scalp psoriasis. Cochrane Database of Systematic Reviews, 2016; 2: Art.No. CD009687.
11. Dogra S, Kaur I. Childhood psoriasis. Indian J Dermatol Venereol Leprol, 2010; 76: 357-65.
12. Thami GP, Sarkar R. Coal tar: Past, present, and future. Clin Exp Dermatol, 2002; 27: 99- 103.
13. NICE. The assessment and management of psoriasis. CG153. 2012. Available from https://www.nice.org.uk/guidance/cg153. October 2012.

14. Racz E, prens Ep. phototherapy and photochemotherapy for psoriasis. Dermatol clin., 2015; 33(1): 79-89.
15. Prystowsky JH, Cohen PR. pustular and erythrodermic psoriasis. Dermatol clin., 1995; 13(4): 757-770.
16. Rosenbach M, Hsu S, Korman NJ, et al. Treatment of erythrodermic psoriasis: from the medical board of national psoriasis foundation.J. Am acad dermatol, 2010; 62(4): 655-662.
17. Greaves MW, Weinstein GD. Treatment of psoriasis. N Engl J Med., 1995; 332: 581-8.
18. Lebwohl M, Drake L, Menter A, Koo J, Gottlieb AB, Zanolli M, et al. Consensus conference: acitretin in combination with UVB or PUVA in the treatment of psoriasis. J Am Acad Dermatol, 2001; 45(4): 544–53 and Koo J. Systemic sequential therapy of psoriasis: a new paradigm for improved therapeutic results. J Am Acad Dermatol, 1999; 41(3 Pt 2): S25–8.
19. Menter A, Korman NJ, Elmets CA, Feldman SR, Gelfand JM, Gordon KB, et al. Guidelines of care for the management of psoriasis and psoriatic arthritis: section 4. Guidelines of care for the management and treatment of psoriasis with traditional systemic agents. J Am Acad Dermatol, 2009; 61(3): 451–85.
20. Mahmood T, zaghi D, menter A. emerging oral drugs for psoriasis. Expert opin emerg drugs, 2015; 20(2): 209-220.
21. Management of erythrodermic psoriasis with low-dose cyclosporin. Studio Italiano Multicentrico Nella Psoriasis (SIMPSO) Dermatology, 1993; 187(1): 30–37.
22. Geilen CC, Tebbe B, Garcia Bartels C, Krengel S, Orfanos CE. Successful treatment of erythrodermic psoriasis with mycophenolate mofetil. Br J Dermatol, 1998; 138(6): 1101–1102.
23. Leonardi C, Papp K, Strober B, et al. The long-term safety of adalimumab treatment in moderate to severe psoriasis: a comprehensive analysis of all adalimumab exposure in all clinical trials. Am J Clin Dermatol, 2011; 12(5): 321–337.
24. Alunno A, Carubbi F, Cafaro G, et al. Targeting the IL-23/IL-17 axis for the treatment of psoriasis and psoriatic arthritis. Expert Opin BiolTher., 2015; 15(12): 1727–1737.
25. Ren V, Dao H. Potential role of ixekizumab in the treatment of moderate-to-severe plaque psoriasis. Clin Cosmet Investing Dermatol, 2013; 6: 75–80.
26. De Jager ME, de Jong EM, van de Kerkhof PC, Seyger MM. Efficacy and safety of treatments for childhood psoriasis: A systematic literature review. J Am Acad Dermatol., 2010; 62: 1013-30.

27. Recent Highlights in Psoriasis Research Samuel T. Hwang1, Tamar Nijsten2 and James T. Elder Page 550 Journal of Investigative Dermatology, 2017; 137 ª 2016 550 The Authors. Published by Elsevier, Inc. on behalf of the Society for Investigative Dermatology
28. Current trends in treatment and management of psoriasis : An update review, Charukesi Sowmya et al. Int. Res. J. Pharm., 2018; 9(3) 6 INTERNATIONAL RESEARCH JOURNAL OF PHARMACY www.irjponline.com ISSN 2230 – 8407.
29. Diagnosis and management of psoriasis Whan B. Kim MD Dana Jerome MD MEd FRCPC Jensen Yeung MD FRCPCCanadian Family Physician • Le Médecin de famille canadien, APRIL • AVRIL, 2017; 63: 278.
30. Guidelines for the Diagnosis and Treatment of Psoriasis in China: 2019 Concise Edition# Committee of Psoriasis, Dermatology Branch, Chinese Medical Association∗ Zhang et al., Int J Dermatol Venereol, 2020; 3: 22. www.ijdv-dermatol.com
31. Claire REID and Christopher E. M. GRIFFITHS Dermatology Centre, Salford Royal Hospital, University of Manchester, NIHR Manchester Biomedical Research Centre, Manchester, UK This is an open access article under the CC BY-NC license. www.medicaljournals.se/acta Journal Compilation © 2020 Acta Dermato-Venereologica, 72. Acta Derm Venereol 2020; 100: adv00032
32. What Characterizes the Severity of Psoriasis? Results from an Epidemiological Study of over 3,300 Patients in the Iberian Region Dermatology 138, 2008; 216: 137–151 Garcia-Diez et al. 147.
33. Diagnosis and Management of Cutaneous Psoriasis: A Review copyright © 2019 Wolters Kluwer Health, Inc. All rights reserved. WWW.WOUNDCAREJOURNAL.COM ADVANCES IN SKIN & WOUND CARE, February, 2019; 2(2): 66.
34. Diagnosis and Management of Cutaneous Psoriasis: A Review copyright © 2019 Wolters Kluwer Health, Inc. All rights reserved. WWW.WOUNDCAREJOURNAL.COM ADVANCES IN SKIN & WOUND CARE &, February, 2019; 32(2): 66.
35. Guidelines for the Diagnosis and Treatment of Psoriasis in China: 2019 Concise Edition# Committee of Psoriasis, Dermatology Branch, Chinese Medical Association∗ Zhang et al., Int J Dermatol Venereol, 2020; 3: 1 www.ijdv-dermatol.com 16.
36. Guidelines for the Diagnosis and Treatment of Psoriasis in China: 2019 Concise Edition# Committee of Psoriasis, Dermatology Branch, Chinese Medical Association∗ Zhang et al., Int J Dermatol Venereol, 2020; 3: 17. www.ijdv-dermatol.com

37. Guidelines for the Diagnosis and Treatment of Psoriasis in China: 2019 Concise Edition# Committee of Psoriasis, Dermatology Branch, Chinese Medical Association Zhang et al., Int J Dermatol Venereol, 2020; 3: 24. www.ijdv-dermatol.com.
38. Diagnosis and Management of Cutaneous Psoriasis: A Review copyright © 2019 Wolters Kluwer Health, Inc. All rights reserved. WWW.WOUNDCAREJOURNAL.COM ADVANCES IN SKIN & WOUND CARE &, Februay, 2019; 32(2): 60.
39. Diagnosis and Management of Cutaneous Psoriasis: A Review copyright © 2019 Wolters Kluwer Health, Inc. All rights reserved. WWW.WOUNDCAREJOURNAL.COM ADVANCES IN SKIN & WOUND CARE & VOL. 32 NO. 2 Page60 FEBRUARY 2019.
40. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6709030/
41. https://www.healthline.com/health/psoriasis/psoriasis-and-alcohol
42. Hackethal V. Guidelines on Psoriasis Comorbidity Screening in Kids Issued. Medscape News & Perspective. Available at http://www.medscape.com/viewarticle/880462?nlid=115307_1584&src=WNL_mdplsfeat_170530_mscpedit_derm&uac=106950CX&spon=33&impID=1357759&faf=1#vp_1. May 23, 2017; Accessed: May 31, 2017.
43. Boyd AS, Menter A. Erythrodermic psoriasis. Precipitating factors, course, and prognosis in 50 patients. J Am Acad Dermatol, 1989; 21(5,1): 985–991. [PubMed] [Google Scholar].
44. Hawilo A, Zaraa I, Benmously R, et al. Erythrodermie psoriasique: profil epidemio-clinique et therapeutique a propos de 60 cas. [Erythrodermic psoriasis: epidemiological clinical and therapeutic features about 60 cases] Tunis Med., 2011; 89(11): 841–847. French. [PubMed] [Google Scholar]
45. Levin E, Sako E, Famenini S, Wu J. Erythrodermic and Pustular Psoriasis. In: Koo J, Levin E, Leon A, Wu J, Gottlieb A, editors. Moderate to Severe Psoriasis. 4th ed. Boca Raton: CRC Press, 2014; 277–288. [Google Scholar].
46. https://www.msdmanuals.com/en-in/professional/dermatologic-disorders/psoriasis-and-scaling-diseases/psoriasis#v14431327.
47. Krish Tangella MD, MBA, FCAP, https://www.dovemed.com/diseases-conditions/erythrodermic-psoriasis/
48. Medically reviewed by Debra Sullivan, Ph.D. MSN, R.N., CNE, COI-Written byJenna Fletcher on February 19, 2019.

49. Stinco G, Errichetti E. Erythrodermic psoriasis: current and future role of biologicals. BioDrugs, 2015; 29(2): 91–101.
50. Zhang P, Chen H, Duan Y, et al. Analysis of Th1/Th2 response pattern for erythrodermic psoriasis. J Huazhong Univ Sci Technolog Med Sci., 2014; 34(4): 596–601.
51. Deeva I, Mariani S, De Luca C, et al. Wide-spectrum profile of inflammatory mediators in the plasma and scales of patients with psoriatic disease. Cytokine, 2010; 49(2): 163–170.
52. Moy AP, Murali M, Kroshinsky D, Duncan LM, Nazarian RM. Immunologic overlap of helper T-cell subtypes 17 and 22 in erythrodermic psoriasis and atopic dermatitis. JAMA Dermatol, 2015; 151(7): 753–760. [PubMed] [Google Scholar]
53. Lee W-K, Kim G-W, Cho H-H, et al. Erythrodermic psoriasis treated with golimumab: a case report. Ann Dermatol, 2015; 27(4): 446–449.
54. Sehgal VN, Srivastava G, Sardana K. Erythroderma/exfoliative dermatitis: A synopsis. Int J Dermatol, 2004; 43: 39–47.
55. Rubins AY, Hartmane IV, Lielbriedis YM, Schwartz RA. Therapeutic options for erythroderma. Cutis., 1992; 49: 424–6.
56. Q. Liu, S. Xiao, Y. Xia TWEAK/Fn14 activation participates in skin inflammationMediators Inflamm., 2017; 2017: 6746870
57. Nograles KE, Zaba LC, Guttman-Yassky E, et al. Th17 cytokines interleukin (IL)-17 and IL-22 modulate distinct inflammatory and keratinocyte-response pathways. Br J Dermatol., 2008; 159(5): 1092–1102.
58. B. Giannotti, P. Carli, and C. Veller-Fornasa, "Management of erythrodermic psoriasis with low-dose cyclosporin," Dermatology, 1993; 187(1): 30–37. View at: Publisher Site | Google Scholarf