International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 1 Jan - Feb 2025, pp: 1529-1533 www.ijprajournal.com ISSN: 2456-4494

Comparitive Study on Risk Factors of Psoriasis and Inflammatory bowel Disease during Pubescence

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Date of Submission: 15-02-2025 Date of Acceptance: 25-02-2025

ABSTRACT:
Psoriasis, categorised as a noncommunicable disease, is a chronic, painful, disfiguring disease for which there is no cure. It negatively impacts patients quality of life. It can occur at any age, and it is most common in the age group of 50–69 years. Inflammatory bowel disease is a chronic inflammatory disease of the gastrointestinal tract and is divided into Crohn disease and ulcerative colitis. It occurs in genetically susceptible individuals after an exaggerated immune response to a normal stimulus, such as food or intestinal flora. Psoriasis and the spectrum of inflammatory bowel disease are chronic, inflammatory organotrophic conditions. The pathogenesis of both diseases implicates innate and adaptive segments of the immune system. The immune response between psoriasis and IBD is similar and comprises phagocytic, dendritic, and nature-killer cells, along with a milieu of cytokines and antimicrobial peptides that stimulate T cells. Psoriasis and IBD are highly heterogeneous diseases, and there is a need for more precise and deeper phenotyping to identify specific subgroups and their genetic. Less than 1% of psoriasis patients developed CD (or UC) during follow-up. Clinically, psoriasis is characterised by either widespread or localised, sharply demarcated silvery, scaly, and erythematous plaques distributed symmetrically. Obesity is an independent risk factor for psoriasis. The main clinical features of CD include diarrhoea, abdominal pain, and weight loss, whereas UC is characterised by mucopurulent, bloody stools.

Key words: Noncommunicable disease, negatively impacting quality of life, is most common in the 50–69 age group; chronic inflammatory disease; Crohn's disease; and ulcerative colitis.

AIM: To analyse and compare various risk factors for psoriasis and inflammatory
bowel disease in pubescence**.**

Objectives: The objective of psoriasis and inflammatory bowel disease is to

- Reducing the symptoms
- Improving the quality of life of the patient
- Minimising the side effects
- Controlling inflammation.

I. INTRODUCTION:

Psoriasis with inflammatory bowel disease is a chronic, inflammatory organotrophic condition with a lifelong relapsing-remitting course. The former affects the skin of 2%–3% of the population, with hyperproliferation of keratinocytes, impaired epidermal barrier function at the site of skin lesions, and skin filtration by activated inflammatory cells. Ulcerative colitis and Crohn's disease are the two prevalent representatives of the IBD groups. Ulcerative colitis affects the rectum and may extend to the colon, with restriction of the inflammatory process to the mucosa and submucosa layers, while Crohn's disease can affect any site of the digestive tract. In many circumstances, determining the aetiology is critical to effective therapy. The aetiology of psoriasis is a great psychological, emotional, and social burden. Quality of life in general is often significantly impaired. Skin lesions are localised or generalised, mostly symmetrical, sharply demarcated, with red papules and plaques, and usually covered with white or silver scales. Lesions cause itching, stinging, and pain. Cardiovascular diseases, chronic kidney disease, Uveitis, Psychiatric disturbances, and Metabolic syndrome and its relevant components (Obesity, Hypertension, Dyslipidaemia, and type 1 and 2 Diabetes), being more prevalent in psoriatic patients which resulting in impaired quality of life. There is a significant impact on the health, well-being, and quality of life of those affected. and shortening of life expectancy. The epidemiology of this relationship remains poorly defined. If the pathogenesis underlying psoriasis has led to significant advancements and highly effective treatments, psoriasis is characterised by inflammatory hyperproliferation of keratinocytes, impaired barrier function of the skin, and

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infiltration of activated immune cells. Psoriasis can be associated with a variety of clinical presentations depending on the underlying causes of scaling of the skin, itching, erythema, fatigue, swelling, burning, and bleeding. Psoriasis involves the skin and nails and is associated with a number of comorbidities. Indeed, it is estimated that 20–30% of psoriasis patients will develop psoriatic arthritis (PsA), and several studies have reported an association between psoriasis, PsA, and inflammatory bowel disease (IBD). Similarly, psoriasis patients also have an increased risk of developing metabolic syndrome (Mets) and type 2 diabetes. Psoriasis has a multifactorial pathogenesis involving both genetic and environmental factors as well as innate and acquired immune responses.

Psoriasis and CD occur more often in the same person than it would be expected if the diseases were mutually exclusive. Data from five case-control studies reported a prevalence of psoriasis of 8.9% in patients with CD but only of 1.4% in the control group. However, although biologics inhibiting the IL-17 pathway have been shown to be efficacious for the treatment of psoriasis and psoriatic arthritis (PsA), concerns have been raised regarding their potential for worsening existing IBD or perhaps even inducing first-time CD or UC. The main clinical features of CD include diarrhoea, abdominal pain, and weight loss, whereas UC is characterised by mucopurulent, bloody stools. Currently, the most commonly accepted hypothesis regarding IBD aetiology is that environmental factors acting on genetically susceptible populations result in a leaky gut barrier, microbiota dysbiosis, and ultimately overactivation of immune responses. Psoriatic disease, consisting of both psoriasis and PsA, is considered a multifactorial disease that has been associated with multiple systemic disorders. However, it is essential to acknowledge that the prevalence of PS can vary significantly depending on the studied population. Reports have indicated variable rates ranging from 2% to 9%. This variability underscores the importance of considering demographic and clinical factors when evaluating the relationship between PS and IBD across different cohorts.

Numerous studies involving diverse populations and settings have associated psoriatic diseases with autoimmune, metabolic, gastrointestinal, and mental health disorders, which has caused a worldwide disease burden.

RELATIONSHIP BETWEEN PSORAISIS AND INFLAMMTORY BOWEL DISEASE;

The common genotype, clinical course, and immunologic features shared by psoriasis and IBD, including the chromosomal locus 6P21 and the 1L32R genes, have been identified. Psoriasis is a chronic, painful, disfiguring, and disabling disease for which there is no cure. There are several pathophysiological links between psoriasis and IBD. Elevated concentrations of cytokines in serum and tissues (i.e., tumour necrosis factor [TNF], interleukin [IL]-12, and IL-23) are present both in psoriasis and IBD, and agents that inhibit their action often improve both conditions. Approximately 15% of patients with IBS are diagnosed with cutaneous EIM of IBD. It is defined by the WHO. It negatively impacts the patient's quality of life. It can occur at any age and is most common in 50- to 69-yyear-olds. IBD, a chronic inflammatory disease of the gastro-intestinal tract, is divided into Crohn's disease and ulcerative colitis. It occurs in genetically susceptible individuals after an exaggerated immune response to a normal stimulus, such as food or intestinal flora.

EITOLOGY OF PSORIAISIS: Psoriasis is a multifactorial disease with extrinsic and intrinsic factors that play a major role in
Idiopathic cause
Genetic
Autoimmune
Infection
Injury to the skin
Chances in climate

PATHOPYSHOLOGY OF PSORIASIS:

Psoriasis is a disease primarily linked to immune-related genes, with psoriatic plaques originating from the interaction between the immune system and skin cells. The disease has a wide clinical spectrum, including chronic plaque-type psoriasis, which is the most common manifestation, and inverse psoriasis, which lacks typical predilection sites and scales. Scalp psoriasis is a rare form of psoriasis. Psoriasis is also associated with other diseases such as psoriatic arthritis, Crohn's disease, cancer, depression, non-alcoholic fatty liver disease, metabolic syndrome, and cardiovascular disorders. The association of some of these comorbid diseases with psoriasis may be due to common genetics.

RISK FACTORS OF PSORIASIS: Genetics (family history of the disease)

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Immune system
Environmental triggers (infections, weather, skin injury, smoking, alcohol, medications)
Lifestyle-related infections (strep throat or skin infections)
Hormonal changes
Skin trauma

ETIOLOGY OF INFLAMMATORY BOWEL DISEASE:
Environmental factors (luminal bacteria, infections, NSAIDs, smoking)
Microbial agents
Smoking
Oral contraceptives
Diet
Lack of hygiene

PATHOPHYSOLOGYOF INFLAMMATORY BOWEL DISEASE:

The pathophysiology of inflammation is depicted in the graphic, along with the steps that lead from the original stimuli to the inflammation that results. Triggered by both environmental influences and genetic predispositions, it starts with triggering events. In genetically sensitive people, these triggers induce a dysregulated inflammatory and immunological response, which results in an excessively active or inappropriate immune response. The immune response is subsequently heightened by this imbalance, which results in the production of proinflammatory interleukins, TNF alpha, and other cytokines—inflammatory mediators that play a critical role in inducing inflammation.

These inflammatory mediators help to tear down mucous barriers as the immune response intensifies. The intestinal lumen is continuously exposed to bacterial or food antigens as a result of this breakdown, exacerbating the immunological response. This subsequently results in a chronic inflammatory state because the immune system is unable to effectively respond to microbial antigens. All of these elements combine to produce chronic inflammation, which damages tissue and exacerbates the signs and symptoms of inflammatory illnesses. The graphic clearly illustrates the important roles that immune system dysregulation, genetic predisposition, and ongoing antigen exposure play in the emergence of inflammation.

RISK FACTORS OF INFLAMMATORY BOWEL DISEASE:
Lifestyle factors (nonsteroidal anti-inflammatory drugs, stress)
Environmental factors (air pollution)
Genetic factors (family history of IBD, medications, hormonal replacement)

II. DISCUSSION:

We found a significantly increased risk of CD and UC among patients with psoriasis. No difference was seen in anatomical IBD location between psoriasis patients and the general population. A higher CD (but not UC) risk was seen with longer psoriasis duration. The positive association between psoriasis and the risk of CD and UC, although IBD was not limited, The observational studies support an underlying bidirectional relationship between psoriasis and inflammatory bowel disease (IBD), although the mechanisms remain unclear. Many factors, such as common genetic susceptibility loci, clinical course, and immunologic features, are shared by psoriatic disease, and IBD is considered a subsequent extra-articular manifestation of PsA, which should be specially monitored in PsA patients, according to the studies. However, although we defined cases of definite CD and UC as patient reports.

Shared Immunological Features: Both psoriasis and IBD involve chronic inflammation driven by dysregulated immune responses. Highlight the specific immune cells and cytokines implicated in both diseases, such as T cells, dendritic cells, and cytokines like TNF-alpha and IL-17.

Genetic and Environmental Factors: Discuss the genetic predispositions that contribute to both psoriasis and IBD. For instance, mention the identified genetic loci (like 6P21 and IL-23R) that are shared between the two diseases. Also, consider environmental triggers that may exacerbate symptoms in genetically susceptible individuals.

Clinical Presentation and Symptoms: Compare and contrast the clinical manifestations of psoriasis and the two main types of IBD (Crohn's disease and ulcerative colitis). Highlight how these diseases impact different organ systems (skin vs. gastrointestinal tract) and their characteristic symptoms (e.g., skin plaques and gut inflammation).

Impact on Quality of Life: Emphasise the significant burden on the quality of life experienced by patients with psoriasis and IBD. Discuss the

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physical, emotional, and social challenges these chronic conditions pose, including their effects on daily activities and psychological well-being.

Treatment Approaches: Briefly touch upon current treatment options for both diseases, focusing on similarities and differences. Include therapies targeting the immune system, such as biologics and immunosuppressants, and their effectiveness in managing symptoms and improving quality of life.

III. CONCLUSION:

In this review we finally conclude that therePsoriasis and inflammatory bowel disease (IBD), like Crohn's disease and ulcerative colitis, share genetic and immune system similarities, impacting patients' quality of life significantly. Psoriasis and IBD were traditionally seen as immune-mediated inflammatory diseases that followed a predetermined course, with limited prospects for effective, let alone curative, treatments.A relationship between psoriasis and IBD remains unclear, but the evidence supports a significant association based on shared genetic, immunological, and clinical features.risk factors related IBD and Psoriasis is genetics, immune system dysregulation, environmental triggers, and lifestyle factors.skin, common drugs, environment and pollution, lifestyle, psychological stress, hormonal and metabolic alterations this are the risk factors for psoriasis.

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