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Case Report

Psoriasis and metabolic dysfunction-associated steatotic liver disease: an underestimated connection: a case report

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ABSTRACT

Psoriasis is a chronic inflammatory skin disease influenced by immunological, genetic, and environmental factors, and is closely linked to metabolic comorbidities such as metabolic dysfunction-associated steatotic liver disease (MASLD). MASLD is defined by hepatic fat accumulation in the presence of cardiometabolic risk factors such as obesity, type 2 diabetes, dyslipidemia, or hypertension. Both psoriasis and MASLD share inflammatory mechanisms, particularly involving the TNF-α/IL-23/IL-17 axis, which promotes systemic inflammation, insulin resistance, steatosic liver disease, and fibrosis. We present the case of a 44-year-old man with untreated type 2 diabetes, active tobacco use, and chronic alcohol consumption, who presented with progressive abdominal distension, shortness of breath, and worsening of preexisting psoriatic lesions. Laboratory findings showed altered liver function, ultrasound confirmed ascites and grade, hepatic steatosis with fibrosis. A skin biopsy confirmed the diagnosis of psoriasis vulgaris. Initial treatment included non-selective beta-blockers, emollients, and therapeutic paracentesis, followed by referral to a tertiary care center. The coexistence of psoriasis and MetALD (a mixed form of MASLD and alcohol-related liver disease) worsens liver prognosis and limits the use of hepatotoxic drugs such as methotrexate. Therefore, early liver ultrasound in psoriatic patients with metabolic risk factors is essential to support safe therapeutic decisions and prioritize biologic treatments when liver damage is present.

Keywords: Psoriasis, MASLD, Metabolic dysfunction-associated steatotic liver disease, Steatosis, Screening, Methotrexate, Biologic therapy

INTRODUCTION

Psoriasis is a chronic inflammatory skin condition mediated by immunological, genetic, and environmental triggers and has been associated with a higher risk of metabolic conditions.[1] It has been linked mainly to insulin resistance syndrome, elevated cardiovascular risk, metabolic dysfunction-associated steatotic liver disease (MASLD), psoriatic arthritis, among others.[2] Its pathogenesis involves the inflammatory axis TNF-α/IL-23/IL-17. In this process, dendritic cells produce IL-23, which activates a signaling cascade by binding to the Th17 receptor, inducing IL-17 secretion.[3] This cytokine acts on keratinocytes, prompting the release of proinflammatory mediators. The sustained interaction between these cytokines and keratinocytes promotes excessive, accelerated, and immature proliferation of keratinocytes.[3]

Conversely, MASLD applies to patients with steatosis plus one or more cardiometabolic risk criteria.[4] Psoriasis and MASLD are closely related due to a shared systemic

inflammatory state. In psoriasis, cytokines such as IL-6, TNF-α, and IL-1β not only perpetuate cutaneous inflammation but also induce insulin resistance and hepatic lipogenesis, promoting fat deposition in the liver.[5] Additionally, IL-23/Th17 axis activation contributes to immune imbalance by increasing IL-17 and IL-22 production, which are involved in progression to steatosic liver disease.[5] Oxidative stress from free fatty acids and reactive oxygen species further exacerbate hepatic dysfunction and systemic inflammation.[6] These immunometabolic mechanisms explain the high prevalence of MASLD in patients with psoriasis, especially in moderate to severe forms.[2] This case underscores the clinical importance of coexisting severe psoriasis and MASLD, a frequent but underdiagnosed comorbidity in chronic inflammatory diseases.

CASE REPORT

A 44-year-old male with a long history of untreated type 2 diabetes, smoking (pack-index 4.2), and chronic weekly alcohol consumption leading to inebriation, denies illicit drug use. He reports a four-year history of dermatitis characterized by thick, erythematous, plaque-like lesions resembling plaster, located on the abdomen, arms, and lower extremities, unresponsive to prior topical salicylic acid treatment.

He presented to the emergency department at Hospital General de Atizapán due to progressive abdominal distension, dyspnea on mild exertion, and exacerbation of cutaneous lesions over the past month. Upon admission, vital signs were: blood pressure 140/85 mmHg, heart rate 75 bpm, respiratory rate 19 breaths per minute, oxygen saturation 92%, and temperature 97.9°F.

Labs revealed glucose 150 mg/dl, total bilirubin 5.3 mg/dl, direct 2.1, indirect 3.2, albumin 2.1 g/dl, AST 206 U/l, ALT 123 U/l, alkaline phosphatase 238 U/l, and platelets 154×10^9/l, with no other notable abnormalities. Hepatic ultrasound showed normal size liver (150×82 mm), regular contours, grade III steatosis, diffuse echogenicity with fibrosis indicators, absent gallbladder, 6 mm common bile duct, approximately 8,500 cc of free abdominal fluid and splenomegaly.

Non-selective beta-blocker therapy was initiated for suspected portal hypertension syndrome, along with topical emollients for skin care. Diagnostic and therapeutic paracentesis drained 5,000 cc ascitic fluid with albumin replacement. Fluid analysis showed < 50 leukocytes per field and sterile culture; serum-ascites albumin gradient indicated portal hypertension. Skin biopsy revealed abundant laminated hyperkeratosis, areas of parakeratosis with neutrophils forming small microabscesses, marked acanthosis with elongation and anastomosing rete ridges, focal hypogranulosis, neutrophil and lymphocyte exocytosis; moderate superficial and mid-dermal inflammatory infiltrate of lymphocytes, histiocytes, neutrophils, and congested dilated capillaries, consistent with psoriasis vulgaris. He was discharged after clinical improvement, with referral to gastroenterology and dermatology tertiary care for evaluation and optimal therapy planning, addressing both suspected chronic liver insufficiency with cirrhosis and severe psoriasis.

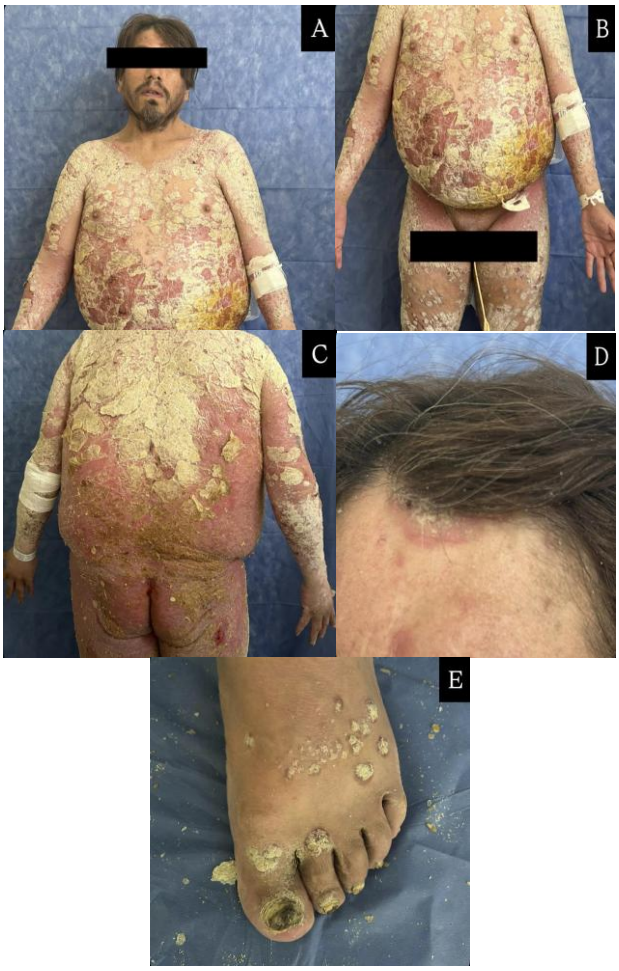

Figure 1: 44-year-old man with liver failure, MASLD, and severe plaque psoriasis (PASI 57%). (A) Generalized dermatosis of the upper trunk with thick, yellowish plaques and irregular borders of varying sizes. (B) Involvement of the abdomen, arms and legs, with thick, plaster-like plaques and irregular borders. (C) Extensive lesions on the back and arms with thick, yellowish plaques with irregular margins. (D) Thick, yellowish plaque measuring 3×1 cm on the anterior scalp margin, with perilesional erythema. (E) Multiple round plaques on the left foot with thick, yellow scales, poorly defined borders, and perilesional erythema.

DISCUSSION

The coexistence of metabolic dysfunction-associated steatotic liver disease (MASLD) and psoriasis represents a clinically significant intersection that is often underestimated and insufficiently explored in medical practice.[5] MASLD is characterized by hepatic steatosis in the presence of at least one cardiometabolic risk factor,

such as obesity, type 2 diabetes, dyslipidemia, or hypertension.[4] It was redefined in 2023 by an international multi-society consensus, replacing the term NAFLD to eliminate an exclusion-based diagnostic approach and reduce stigma.[7] The key cardiometabolic risk factors considered in the diagnosis of MASLD include elevated body mass index or waist circumference, impaired fasting glucose or glucose tolerance, elevated HbA1c, hypertension or use of antihypertensive medication, high triglyceride levels, and low HDL cholesterol levels, with diagnostic thresholds adjusted by sex.[7] The 2023 Delphi consensus also introduced the term steatotic liver disease (SLD) as an umbrella category that includes all forms of liver disease characterized by fat accumulation. Within SLD, three primary subtypes are recognized: MASLD (associated with metabolic dysfunction), ALD (alcohol-related liver disease), and MetALD (a mixed form involving both metabolic dysfunction and moderate alcohol consumption).[7] In mixed cases, it is essential to assess which factor predominates to guide diagnosis and clinical management, given the potential for overlapping presentations.[7]

The case presented in this research underscores the relevance of this framework, as the patient had a history of alcohol consumption, initially suggesting a diagnosis of MetALD. However, the clinical profile reveals a predominance of metabolic dysfunction, thus aligning the case more closely with MASLD. It is important to emphasize that MASLD and ALD can coexist in a single patient, especially when both metabolic risk factors and alcohol intake are present.[7]

Both conditions, particularly MASLD, are associated with inflammatory comorbidities such as psoriasis. This association is due to shared inflammatory pathways involving disturbances in innate immunity, oxidative stress, and dysfunction of the gut-liver axis.[5,8] Psoriasis is a chronic inflammatory disease of multifactorial origin, characterized by abnormally accelerated epidermal turnover leading to the development of well-demarcated erythematous plaques covered with scale, most commonly located on the elbows, knees, and scalp.[9] From a pathophysiological perspective, psoriasis results from complex interactions among genetic, immunologic, and environmental factors that provoke dysregulated activation of the innate and adaptive immune systems.[10]

Dendritic cells, which play a central role as antigen-presenting cells in the immune response, are activated mainly by antimicrobial peptides (AMPs), which are released following tissue damage in the skin.[10] Among the AMPs most implicated in psoriasis are LL37, β-defensins, and S100 proteins. LL37, released by damaged keratinocytes, forms complexes with extracellular DNA that activate Toll-like receptor 9 (TLR9) in plasmacytoid dendritic cells, initiating the production of type I interferons and triggering a cascade of inflammation. This process leads to the maturation of myeloid dendritic cells and differentiation of Th1 and Th17 cells, which release inflammatory cytokines such as IFN-γ and IL-17, contributing to the formation of psoriatic plaques.[10] The persistent inflammatory response promotes excessive keratinocyte proliferation and impaired differentiation, giving rise to the characteristic clinical and histopathological features of the disease.[10] Histologically, psoriasis is marked by acanthosis, elongation of rete ridges, parakeratosis, thinning of the granular layer, inflammatory infiltrates, and, in some cases, the presence of Munro microabscesses and Kogoj pustules.[1] The treatment of moderate to severe plaque psoriasis has shifted toward a goal-oriented model, favoring systemic therapy when there is extensive skin involvement, involvement of sensitive anatomical areas, or inadequate response to topical treatments. Available therapeutic options include topical agents, phototherapy, conventional systemic agents such as methotrexate and cyclosporine, and biologic therapies that target specific inflammatory mediators, including TNF-alpha, IL-17, and IL-23.[1] The pathophysiological link between psoriasis and MASLD is grounded in the chronic systemic inflammatory state that both conditions share, primarily driven by immune system dysregulation.[5]

In psoriasis, Th17 pathway activation promotes the release of proinflammatory cytokines such as IL-17, IL-6, TNF-α, and IL-1β.[1] These cytokines not only maintain skin inflammation but also impair systemic metabolism by inducing insulin resistance and promoting hepatic de novo lipogenesis while simultaneously inhibiting fatty acid oxidation.[5] This combination fosters hepatic fat accumulation and stimulates immune cell infiltration in the liver, thereby intensifying hepatic inflammation and tissue damage.[5] These mechanisms explain how psoriasis may predispose individuals to the development of MASLD and highlight the systemic and metabolic aspects of psoriasis as an inflammatory disease with significant hepatic implications.[1,5]

CONCLUSION

The coexistence of psoriasis and metabolic dysfunction-associated steatotic liver disease (MASLD) is not uncommon, particularly in patients with cardiometabolic comorbidities such as obesity, hypertension, or chronic alcohol consumption. This clinical case illustrates how this association can have meaningful clinical implications when selecting systemic treatments. Methotrexate, although an effective agent for psoriasis, carries a risk of hepatotoxicity and requires a thorough assessment of liver status prior to its use.

Therefore, systematic screening for MASLD, using tools such as hepatic ultrasound, should be considered in patients with psoriasis who have metabolic risk factors. This approach enables early detection of subclinical liver involvement and supports safer, more individualized therapeutic decision-making. In patients with evidence of hepatic impairment, initiating treatment with biologic therapies may represent a more appropriate and safer

strategy, reduce risks while optimize the integrated management of both conditions.

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