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Relationship Between Nonalcoholic Fatty Liver Disease and Psoriasis - A Comprehensive Review

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ABSTRACT

A number of comorbidities are linked to psoriasis. It is a systemic inflammatory disease. Research indicates that there is a higher prevalence of nonalcoholic fatty liver disease in psoriasis patients compared to controls. Individuals who have both psoriasis and nonalcoholic fatty liver disease are more likely to develop severe liver fibrosis and have more severe skin diseases overall. In addition to reviewing the diagnosis of nonalcoholic fatty liver disease, the authors will go over psoriasis treatments and lifestyle modifications that can either help or worsen the condition.

KEYWORDS: Psoriasis, Nonalcoholic fatty liver disease, Inflammation, Skin disease

INTRODUCTION

The term "fatty liver disease" describes a disorder where hepatocytes acquire fat. After ruling out other possible causes such as alcoholic fatty liver disease, hepatitis C, autoimmune hepatitis, primary biliary cirrhosis, and Wilson's disease; nonalcoholic fatty liver disease (NAFLD) is identified. NAFLD is a metabolic illness that falls within a range of diseases. It can range from nonalcoholic steatohepatitis (NASH), which is an inflammatory condition that causes scarring, fibrosis, and perhaps cirrhosis, to steatosis (isolated fatty liver) without particular liver damage [1]. Triglycerides build up in the hepatocytes as a result of aberrant hepatic metabolism, which is the cause of NAFLD. Male gender, age, obesity, insulin resistance, and metabolic syndrome are the most common risk factors for this disease [2]. Proinflammatory adipokines or cytokines produced from the skin can cause hepatic fat buildup and insulin resistance. Sometime few people experience more severe illness than others;but till now the reason is unknown. Lifestyle factors such as smoking, food, and sedentary behaviour might affect

severity. A role for genetic predisposition is also believed to exist. It has been demonstrated that risk is conferred by a mutation in the PNPLA3 (phospholipase domain containing protein 3) gene [3]. Excess proinflammatory cytokines/adipokines, mitochondrial dysfunction, and oxidative stress are additional variables that lead to more severe illness [1]. Patients with autoimmune disorders, including psoriasis, may undoubtedly be more vulnerable to more severe liver scarring due to inflammation. Thus cirrhosis and less commonly hepatocellular cancer can occur in certain NASH patients. By 2025, NAFLD is predicted to be the primary cause of liver transplants globally [4].

EPIDEMIOLOGY OF NONALCOHOLIC FATTY LIVER DISEASE (NAFLD)

Doctors, Nurses and other healthcare providers should be aware that the most common liver condition in the United States is fatty liver, affecting around 30% of the population; 3–10% of these patients have nonalcoholic steatohepatitis (NASH). The development of NAFLD occurs in almost two thirds of diabetic patients. Obese individuals have a greater than 33% chance of getting NASH, while lean individuals have a lower than five percent risk. In proportion to the degree of obesity, fatty liver becomes more common and more severe. NAFLD, unrelated to metabolic syndrome, is a risk factor for cardiovascular disease. More often than liver-related mortality, these patients die from major adverse cardiovascular events (MACE) [5]. It is estimated that around 10% of children between the age groups of 2 - 19 years in the United States suffer from non-alcoholic fatty liver disease (NAFLD). Children are not exempt from the present obesity pandemic. Similar to adults, the frequency rises to 40–70% with obesity, and there have been reports of paediatric cirrhosis cases [2].

DIAGNOSIS OF NONALCOHOLIC FATTY LIVER DISEASE (NAFLD)

Unless the disease is quite advanced, most patients don't feel any symptoms. Rarely, the liver is enlarged enough to feel sensitive to the touch. Cirrhosis of the liver symptoms might manifest when the disease is advanced. Although up to two-thirds of individuals may have normal liver function tests (LFTs), modestly raised LFTs are frequently the first step towards the diagnosis of NAFLD. Alanine aminotransferase (ALT) is usually greater than aspartate aminotransferase (AST), but rarely greater than three times upper limit normal. Gamma-glutamyltransferase (GGT) is often high and alkaline phosphatase may be slightly raised. Hyperbilirubinemia and low albumin level indicates the severe condition of this disease. The four variables that make up the Fatty Liver Index (FLI) are serum triglyceride levels, body mass index (BMI), waist circumference, and GGT. The FLI was created as a straightforward algorithm to predict fatty liver [1]. NAFLD is most frequently diagnosed with magnetic resonance imaging (MRI), computed tomography (CT) and ultrasound. Liver biopsy is the only method that can differentiate between fatty liver disease and the more severe form of NASH in a patient. Patients with diabetes, metabolic syndrome, and obesity (BMI>30) are high-risk for NASH and may be candidates for liver biopsy [1].

NONALCOHOLIC FATTY LIVER DISEASE (NAFLD) IN PSORIASIS

NAFLD is 1.5–3 times more common in people with psoriasis, according to several observational studies conducted in hospitals

[3]. NAFLD was identified in 59% of psoriatic individuals in Italian prospective research. It showed a strong correlation with obesity, psoriatic arthritis, and metabolic syndrome. According to noninvasive NAFLD fibrosis scores, psoriatic individuals had a higher likelihood of having more severe liver fibrosis than the non-psoriatic cohort [6]. In another Italian investigation, 130 psoriatic patients and matched controls were compared. In comparison to controls (28%), psoriasis patients (47%) had considerably higher NAFLD. Psoriasis patients with NAFLD (PV-NAFLD) were more likely to have metabolic syndrome, higher C-reactive protein, and greater psoriasis area and severity index (PASI) scores than psoriasis patients without NAFLD. Lower levels of adiponectin and higher levels of interleukin-6 (IL-6) were seen in a subset of the PV-NAFLD group [7]. In an Indian case-control study, 333 psoriasis patients and matched non-psoriasis patients were compared. Compared to 7.9% of controls, 17.4% of psoriasis patients had NAFLD. Compared to psoriasis patients without NAFLD, psoriatic patients with NAFLD had skin diseases that were more severe and had lasted longer. They also had a higher likelihood of diabetes and multiple sclerosis. Patients with psoriasis exhibited more severe liver damage than the general population [8]. Psoriasis and nonalcoholic fatty liver disease (NAFLD) both are linked to metabolic disorders such obesity and metabolic syndrome. Therefore, it has not been evident if psoriasis is linked to NAFLD on its own. The Rotterdam research, a prospective population-based cohort study involving Dutch patients 55 years of age and above, looked at this problem. There was an independent correlation between psoriasis and a 70% higher risk of NAFLD. Psoriasis, with an odds ratio of 1.7, and metabolic syndrome, with an odds ratio of 3.5, both was the two significant predictors of illness. Patients with psoriasis had a 60% increased risk of developing the more severe types of liver disease. This came after accounting for factors including age, gender, and alcohol use [3].

TREATMENT OPTIONS FOR NONALCOHOLIC FATTY LIVER DISEASE (NAFLD)

A study revealed that, only 10% weight loss might reduce the amount of liver fat. In addition to decreasing liver fat, vigorous exercise can help lower NASH inflammation. It is uncertain if diet or exercise will prevent cirrhosis in these people over the long run. According to studies, diets heavy in high-fructose corn syrup or saturated fats can both cause NAFLD. A diet that reduces these would thus probably be advantageous [9]. Research has demonstrated a correlation between the degree of insulin resistance and the development of liver fibrosis [10]. Individuals who consume over two cups of coffee daily exhibit reduced liver fibrosis or scarring. It is therefore advised to include coffee in the diet [11]. It is important to address vitamin D insufficiency as it can lead to elevated inflammation by impairing T suppressor cell activity [12]. Lastly, people with NAFLD who have psoriasis must limit their alcohol intake. Although the long-term safety and efficacy of vitamin E supplementation are uncertain, it can be utilised to decrease inflammation and fibrosis in NASH patients at levels of 800 IU/day [5]. Larger trials are being conducted; however smaller studies have demonstrated that omega-3 fatty acids decrease liver fat in NAFLD [13]. It has also been demonstrated that this supplement lowers cardiovascular mortality in people who are at high risk of MACE episodes.

BIOLOGICS AND MANAGEMENT OF NONALCOHOLIC FATTY LIVER DISEASE (NAFLD)

For a maximum of 24 weeks, 89 patients with psoriasis, metabolic syndrome, and non-alcoholic fatty liver disease (NAFLD) were treated in either group when etanercept with psoralen and ultraviolet A (PUVA) were compared. Aspartate transaminase and alanine transaminase (AST/ALT) ratio, C-reactive protein, fasting insulin levels, homeostasis model assessment index (HOMA), and insulin sensitivity check index (QUICK) were all significantly lower in the etanercept group alone. It was determined that etanercept may be a more effective treatment than PUVA in lowering the risk of developing hepatic fibrosis since the risk of fibrosis advancement is directly linked to insulin resistance [10]. 32 psoriasis patients receiving biologic treatment for moderate-to-severe liver damage were examined in a retrospective analysis. 3 of the 32 individuals had fatty liver disease. Adalimumab-related side effects and liver disease development were not seen during the course of a five-year follow-up [14].

Adalimumab was used to treat a 21-year-old woman with rheumatoid arthritis who had NASH but no appreciable fibrosis, according to a case study. After four months, the levels of gamma-glutamyl transferase (GGT) decreased and subsequently returned to normal. ALT, AST, and GGT levels remained normal 10 months [15].

PSORIASIS TREATMENT OPTIONS TO AVOID IN NONALCOHOLIC FATTY LIVER DISEASE (NAFLD)

A study showed that, patients with hepatic, renal, or haematologic problems should not use methotrexate [16]. Compared to controls and rheumatoid arthritis patients, psoriasis patients are more likely to suffer methotrexate-induced liver damage [17]. The kidney is the main organ of concern with cyclosporine although it can increase lipid levels and thus potentially worsen NAFLD [16]. Elevated LFTs have been linked to actitretin; yet liver disease development is extremely uncommon. It frequently causes hyperlipidemia, which should be avoided in NAFLD patients [16].

DISCUSSION AND CONCLUSION

It is important to determine whether a psoriasis patient has an underlying fatty liver condition for two reasons. First, fatty liver may indicate underlying illness susceptibility, including metabolic syndrome, diabetes, obesity, and cardiovascular disease. Additional correlations include hyperthyroidism, polyps in the colon, high uric acid, low vitamin D, and polycystic ovaries. Occlusive sleep apnoea affects almost half of those with non-alcoholic fatty liver disease [18]. The second reason is that cirrhosis and hepatocellular cancer are conditions that NASH patients are more likely to acquire. Selecting the right psoriasis treatment choices to reduce liver toxicity requires careful consideration of this information. Patients may be benefitted by changing their lifestyles, such as quitting alcohol and cigarettes, doing exercise regularly, losing weight, and consuming less high fructose corn syrup and saturated fat. Oral vitamin D3, E, and omega-3 supplements have also been demonstrated to be beneficial in some patient populations. However, a few case reports suggest that TNF-alpha antagonists may help psoriatic people with NAFLD; although large controlled studies are needed.

Declaration by Authors

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- **Conflict of Interest:** The authors declare no conflict of interest.

REFERENCES

1. Dowman JK, Tomlinson JW, Newsome PN. Systematic review: the diagnosis and staging of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis. *Ailment Pharmacol Ther*. 2011;33(5):525–540.
2. Bellentani S, Scaglioni F, Marino M, Bedogni G. Epidemiology of non-alcoholic fatty liver disease. *Dig Dis.* 2010; 28:155–161.
3. Van der Voort EAM, Koehler EM, Dowlatshahi EA, et al. Psoriasis is independently associated with nonalcoholic fatty liver disease in patients 55 years old or older: results from a population based study. *J Amer Acad Dermatol.* 2014; 70:517–524.
4. Musso G, Gambino R, Cassader M, et al. Meta-analysis: natural history of non-alcoholic fatty liver disease (NAFLD) and diagnostic accuracy of non-invasive tests for liver disease severity. *Ann Med.* 2011; 43:617–649.
5. Smith BW, Adams LA. Non-alcoholic fatty liver disease. *Crit Rev Clin Lab Sci*. 2011;48(3):97–113.
6. Miele L, Vallone S, Cefalo C, et al. Prevalence, characteristics and severity of non-alcoholic fatty liver disease in patients with chronic plaque psoriasis. *J Hepatol.* 2009; 51:778–786.
7. Gisondi P, Targher G, Zoppini G, Girolomoni G. Non-alcoholic fatty liver disease in patients with chronic plaque psoriasis. *J Hepatol.* 2009; 51:758–764.
8. Madanagobalane S, Anandan S. The increased prevalence of non-alcoholic fatty liver disease in psoriatic patients: a study from South India. *Australas J Dermatol.* 2012;53(3):190–197.
9. Harrison SA, Day CP. Benefits of lifestyle modification in NAFLD. *Gut.* 2007; 56:1760–1769.
10. Campanati A, Ganzetti G, DiSario A, et al. The effect of etanercept on hepatic fibrosis risk in patients with nonalcoholic fatty liver disease, metabolic syndrome and psoriasis. *J Gastroenterol.* 2013;48(7):839–846.
11. Gutierrez-Grobe Y, Chavez-Tapia N, Sanchez-Valle V, et al. High coffee intake is associated with lower grade nonalcoholic fatty liver disease: the role of peripheral antioxidant activity. *Ann Hepatol.* 2012; 11:350–355.
12. Eliades M, Spyrou E, Agrawal N, et al. Meta-analysis: vitamin D and non-alcoholic fatty liver disease. *Aliment Pharmacol Ther.* 2013; 38:246–254.
13. Scorletti E, Byrne CD. Omega-3 fatty acids, hepatic lipid metabolism, and nonalcoholic fatty liver disease. *Annu Rev Nutr.* 2013; 33:231–248.
14. Vilarrasa E, Puig L, Alomar A. Biologic treatments for psoriasis in patients with hepatitis C virus infection and other liver diseases: experience in 29 patients. *JEADV.* 2010;24 P013.
15. Schramm C, Schneider A, Marx A, et al. Adalimumab could suppress the activity of non alcoholic steatohepatitis (NASH) *Z Gastroenterol.* 2008; 46:1369–1371.
16. Bhutani T, Hong J, Koo J. Newtown, PA: Handbooks in Healthcare; 2011. Contemporary Diagnosis and Management of Psoriasis, 5th ed. pp. 50–59.
17. Taylor WJ, Korendowych E, Nash P, et al. Drug use and toxicity in psoriatic disease: focus on methotrexate. *J Rheumatol.* 2008; 35:1454–1457.
18. Mirrakhimov AE, Polotsky VY. Obstructive sleep apnea and nonalcoholic fatty liver disease. *Front Neurol.* 2012; 3:149–190.

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